Schizophrenia

https://doi.org/10.1038/s41537-025-00723-w

Article in Press

Social cognitive deficits and their relationship with clinical symptoms in schizophrenia

Received: 16 June 2025

Accepted: 18 December 2025

Cite this article as: He, D., Zhang, D., Ding, Y. *et al.* Social cognitive deficits and their relationship with clinical symptoms in schizophrenia. *Schizophr* (2026). https://doi.org/10.1038/s41537-025-00723-w

Danni He, Douyu Zhang, Yushen Ding, Xiaoyao Wang, Yujie Xing, Tian Li, Zhiqi Liu, Chenyu Zhou, Zhen Mao, Yimeng Wang, Lei Zhao, Feng Li, Fuchun Zhou, Fang Dong, Chuanyue Wang & Qijing Bo

Social Cognitive Deficits and Their Relationship with Clinical Symptoms in Schizophrenia

Running title: Social cognition deficits and symptoms in schizophrenia

Danni He [a#], Douyu Zhang [a,b#], Yushen Ding [a], Xiaoyao Wang [a], Yujie Xing [a], Tian Li [a], Zhiqi Liu [a], Chenyu Zhou [a], Zhen Mao [a], Yimeng Wang [a], Lei Zhao [a], Feng Li [a], Fuchun Zhou [a], Fang Dong [a], Chuanyue Wang [a], Qijing Bo [a*]

Author affiliations:

[a] The National Clinical Research Center for Mental Disorders & Beijing Key Laboratory of Mental Disorders & Beijing Institute for Brain Disorders Center of Schizophrenia, Beijing Anding Hospital, Capital Medical University, Beijing 100088, China

[b] Medical Psychological Center, The Second Xiangya Hospital, Central South University, Changsha, Hunan, 410011, China.

[#] Dnni He and Douyu Zhang contributed equally to this work and share first authorship.

*Corresponding author: Email: bqj718@163.com; Qijing Bo, Beijing Anding Hospital, Capital Medical University, No. 5 Ankang Hutong, Xicheng District, Beijing 100088, China.

Abstract

While individuals with schizophrenia (SZ) exhibit deficits in social cognition, the specific profile of these deficits across multiple domains and their relationship with clinical symptoms warrants further characterization. This study aimed to systematically assess key social cognitive domains—theory of mind (ToM), emotion recognition, attributional style, and social perception — and examine their associations with psychopathology in SZ. Sixty-eight individuals with SZ and 68 matched healthy controls (HCs) completed a comprehensive battery of social cognitive measures, including the false-belief task (assessing first- and second-order ToM), the faux pas task, the emotional recognition task, the attributional style questionnaire, and the social perception scale. Clinical symptoms were assessed using the Positive and Negative Syndrome Scale (PANSS). Compared to HCs, individuals with SZ showed significant deficits across all social cognitive measures. Specifically, the SZ group exhibited deficits in emotion recognition for all negative emotions (fear, anger, sadness, disgust) but not for happiness, and in attributional style for positive but not negative events. Correlation analyses identified a statistically significant inverse relationship between attributional stability for negative events (i.e., the tendency to attribute the causes of negative events to factors that are persistent over time) and PANSS general psychopathology scores ($r = -0.25$, $P < 0.043$). Furthermore, no other social cognitive domains (ToM, emotion recognition, social perception) showed significant correlations with any PANSS

symptom dimensions. Network analysis further characterized second-order ToM as the core deficit, exhibiting the highest strength and centrality within the social cognitive network, with mediation effects most pronounced for sadness and happiness recognition. These findings highlight second-order ToM as a core deficit in individuals with schizophrenia and suggest that a stable attributional style may be associated with a lower overall burden of general psychopathology. These social cognitive domains may represent promising targets for future cognitive remediation interventions for people living with schizophrenia.

Keywords: Schizophrenia, Social Cognition, PANSS, Network Analysis

According to the Global Burden of Disease Study 2021, schizophrenia ranked as the 18th leading cause of years lived with disability (YLDs) [1]. Schizophrenia (SZ) is a serious mental health condition characterized by "positive symptoms," "negative symptoms," and "cognitive deficits" [2]. These Cognitive deficits represent not only a core characteristic of SZ but can also be a strong predictor of illness progression, clinical outcomes, and overall prognosis [3]. Cognitive deficits in SZ encompass both neurocognitive and social cognitive functions.

Social cognition refers to the mental operations underlying social interactions, which involve perceiving, interpreting, and responding to the intentions, traits, and behaviors of others[4]. Core domains of social cognition include theory of mind (ToM), emotion recognition, attributional style, social perception, and so on[5]. A substantial body of empirical evidence has established that social cognition serves as a critical predictor of functional outcomes in individuals with schizophrenia and accounts for more variance in functional differences compared with other factors [3]. For instance, social cognition not only demonstrates a greater capacity than neurocognition in explaining functional disparities but is also theorized to mediate the impact of neurocognitive deficits on real-life functional outcomes [6,7]. Previous research has indicated that rehabilitation interventions targeting social cognition are more effective in promoting improvements in individuals' social skills, quality of life, and interpersonal functioning[8-11]. Based on this pivotal role of social cognition in predicting and mediating functional outcomes, this domain has emerged as a

significant area of research in schizophrenia, with a marked increase in related studies over the past two decades[12,13].

Furthermore, clinical symptoms are closely associated with functional outcomes in individuals with schizophrenia. Research has demonstrated that the severity of clinical symptoms in schizophrenia—including positive, negative, and disorganization dimensions—exhibits a consistent and robust direct association with a range of adverse functional outcomes[14]. The disability associated with negative symptoms is particularly prominent, as these symptoms are considered among the strongest predictors of reduced community functioning and quality of life, typically demonstrating a more stable relationship with functional impairment than positive symptoms[15].

Social cognitive deficits and clinical symptoms in SZ are both strongly associated with individuals' functional abilities and jointly influence social functioning. Consequently, understanding their interplay is crucial, as it may illuminate the psychological mechanisms that underlie functional recovery. Empirical evidence indeed suggests a link between clinical symptoms and social cognition. For instance, Frith's influential model posits that Theory of Mind (ToM) deficits are central to the disorder's psychopathology, specifically explaining difficulties in volition, self-monitoring, and inferring others' intentions. An impaired ability to attribute the origin of one's own thoughts, for example, may give rise to positive symptoms like delusions of control [16]. Previous research has primarily

focused on examining the relationship between ToM and clinical symptoms in schizophrenia. Although associations have been reported, the evidence remains inconsistent [17]. Among symptom dimensions, ToM tends to be more strongly associated with negative symptoms than with positive or disorganized symptoms. However, this pattern requires qualification when examining specific symptoms, as our analysis at the individual symptom level indicated that deficits in formal thought processes — specifically, difficulty in abstract thinking and conceptual disorganization, which are key components of the disorganization dimension — demonstrated the strongest correlations with ToM. In contrast, the correlations between ToM and core positive symptoms were relatively modest [18]. Moreover, the distinction between affective and cognitive ToM provides further nuance; extant research has validated that their correlations with clinical symptoms are distinct, with affective ToM showing a significant association with negative symptoms, while cognitive ToM is linked to positive symptoms[19].

Despite these findings, the relationship between clinical symptoms and social cognitive deficits in SZ remains insufficiently understood, as no study has conclusively determined the specificity or strength of associations between distinct social cognition domains and clinical symptoms. Understanding the interaction between cognitive deficits and clinical symptoms is critical for identifying precise therapeutic targets and developing personalized treatment strategies for SZ — strategies that directly address the mechanisms underlying functional impairment.

Rather than applying a one-size-fits-all cognitive remediation, clinicians may prioritize emotion-recognition training for an individual whose negative symptoms contribute to social withdrawal, or focus on perspective-taking exercises for someone whose paranoia and misinterpretation of others' intentions pose barriers to occupational functioning.

In summary, by clarifying how specific symptoms and cognitive deficits interact, this line of research holds the key to developing personalized, function-driven rehabilitation strategies for individuals with SZ. However, the structure of social cognition itself also remains unclear. Despite growing research interest, the structural components of social cognition remain inadequately characterized, and the architecture of relationships among these domains within the individual is not yet clearly defined[20]. It remains to be elucidated which specific aspects of social cognition are most impaired in individuals with schizophrenia. This study employed network analysis to systematically explore the core manifestations within the social cognitive domain and their specific relationships with the three major clinical symptom dimensions in SZ. The specific aims were: (1) to verify the presence of deficits across multiple core domains of social cognition in SZ. (2) to investigate the potential association patterns between various social cognitive components and clinical dimensions; and (3) to visualize the structure of the social cognitive network to identify the core nodes with the highest connectivity strength and centrality, which may represent critical intervention targets. Based on prior research, we hypothesized

that individuals with SZ would demonstrate broad deficits across all social cognitive domains, and that each social cognitive domain would be correlated with clinical symptoms. Additionally, this study will exploratorily examine other social cognitive domains that may play crucial, yet under-investigated, central roles within the overall network.

METHODS

Subjects

The sample initially consisted of 79 individuals with SZ, recruited from Beijing Anding Hospital, Capital Medical University (Beijing, China). All individuals met the DSM-5 criteria for SZ, were aged between 16 and 50 years, had at least 6 years of education, demonstrated normal intelligence, and had normal or corrected-to-normal vision. Exclusion criteria included: a history of alcohol or substance abuse, pregnancy or breastfeeding, severe or unstable physical illness, significant agitation, impulsivity, suicidality, self-harming behavior, or uncooperativeness, a history of electroconvulsive therapy or repetitive transcranial magnetic stimulation within the past six months, and color blindness or other ophthalmic disorders. Of these, 4 did not meet the intelligence criteria, and 7 were unable to complete the tasks.

The healthy controls (HCs)group initially consisted of 74 individuals. None met the DSM-5 diagnostic criteria for any mental disorders. Inclusion criteria were: age 16- 50 years, at least 6 years of education, normal intelligence (IQ > 70) as assessed

by the short form of the Wechsler Adult Intelligence Scale-Revised (Chinese version), and normal or corrected-to-normal vision. Exclusion criteria included: a current or past diagnosis of SZ spectrum or bipolar disorders, a family history of neurological disorders, a history of serious neurological or other severe physical illnesses, or any current or past mental disorder secondary to physical illness or substance use. Six HCs were excluded due to demographic (age/education) mismatch with the patient group, resulting in a final HC sample of 68.

These exclusion criteria were adopted to prevent comorbid substance abuse and acute states of agitation or suicidality from confounding neurocognitive and social cognitive task performance[21-23]. Furthermore, to minimize the potential confounding effects of age- and education-related differences in social exposure on assessment results[24], the SZ and HC groups were matched on age and years of education. The limitations associated with these methodological choices are discussed in a later section.

The final analysis included a total of 136 participants: 68 in the SZ group and 68 in the HC group. All participants, or their legal guardians where applicable, provided voluntary informed consent. Upon completion of the study, each participant received pro-rated monetary compensation as a transportation subsidy, based on their level of participation. This study was approved by the Medical Ethics Committee of Beijing Anding Hospital (Approval Number: [2020] KY No. 104).

Clinical assessment

The Positive and Negative Syndrome Scale (PANSS) was used to evaluate the clinical symptoms of SZ. Originally developed by Kay, Opler, and Fiszbein in 1987, this scale assesses the severity of SZ symptoms. It consists of three subscales: the Positive Scale, the Negative Scale, and the General Psychopathology Scale, with each item rated on a 7-point Likert scale. The Chinese version, presented by He Yanling and colleagues in 1998, was used in the present study. This version has established Chinese norms in the schizophrenia population and demonstrates sound psychometric properties. Factor analysis supported a five-factor model (negative, cognitive, positive, excitement, and anxiety/depression), with correlations between each factor and the total score ranging from 0.27 to 0.76, and high correlations ($r > 0.83$) with corresponding factors of the original English version, affirming strong construct validity. All PANSS assessments were conducted according to standardized procedures by trained clinicians through semi-structured interviews.

Measurements of social cognition

ToM was assessed using two tasks: the false-belief task [25] and the faux pas task [26]. The false-belief task measures four dimensions of ToM: first-order emotional ToM, first-order cognitive ToM, second-order emotional ToM, and second-order cognitive ToM[27,28]. For this study, scenarios were adapted from existing protocols validated for adults with schizophrenia [29]. The number of items per dimension was reduced to four, resulting in a total of 16 items. Each question had a single correct answer, scored 1 point for correct and 0 for incorrect, yielding a

total score range of 0-16. Previous research indicates that participants' semantic comprehension ability does not significantly predict performance on the false-belief task, and there is no significant correlation between task performance and working memory [30,31].

The Faux Pas Task, originally developed by Valerie E. Stone[32] and later adapted by Baron-Cohen, assesses ToM in healthy adults and has since been applied in adult patients with schizophrenia in China[29]. Standard versions contain 10 or 20 items. For this study, we selected and culturally adapted 6 items from the 20-item English version to ensure cultural relevance for Chinese participants. Each item presents a brief social scenario involving 2–3 characters, where a social faux pas occurs due to a speaker's lack of knowledge or a listener's misunderstanding. Following each scenario, participants answered 7 questions. Each correct answer received 1 point, yielding a maximum of 7 points per item and a total possible score ranging from 0 to 42. During testing, the examiner read each scenario aloud, and participants recorded their responses in a booklet.

Emotional recognition was assessed using a facial emotion recognition task. Ten facial images (five men and five women) were randomly selected from the Chinese Affective Picture System (CAPS) [33], , a well-validated system providing normative ratings for valence, arousal, and dominance tailored to the Chinese population. The images depicted the five basic emotions: happiness, sadness, disgust, fear, and anger. For each image, participants were required to identify the emotion

by selecting from five options. Scoring was 1 point per correct response, for a total score range of 0–10.

The Attributional Style Questionnaire (ASQ) [34] was used to assess participants' attributional styles and to identify potential attributional biases. The Chinese version employed in this study was adapted by Guo et al. (2003) from the original English scale developed by Peterson et al. (1982). This version has demonstrated reliability and validity in Chinese healthy, depressive, and schizophrenia populations. The original ASQ presents 12 hypothetical scenarios(6 positive, 6 negative), For each scenario, participants imagine the event happening to them, identify the most likely cause, and then rate that cause on three dimensions (internality, stability, globality) using 5-point Likert scales. In this study, to manage time constraints, we used a shortened version comprising 8 scenarios (4 positive, 4 negative). Scores for the three attributional dimensions were summed to yield a total score.

Social perception was assessed using the Relationships Across Domains (RAD) questionnaire [35]. The original version, developed by Sergi et al. (2009), comprises 25 relationship models (each with 3 follow-up scenarios) where participants predict the likelihood of subsequent events. The RAD has been used in studies of schizophrenia[36,37] , though less commonly than other batteries. In this questionnaire, participants are presented with relationship models involving two individuals and are asked to predict the likelihood of three subsequent events. The original version comprises 25 relationship models and 75 follow-up scenarios. In this study, 10

relationship models were selected and culturally adapted to shorten the length and improve relevance to the Chinese context. During the administration, the examiner read each item aloud, and participants recorded their responses in a booklet after each question. One point was awarded for each correct response, resulting in a total score ranging from 0 to 30.

Statistical analysis

All analyses were conducted in R (R Core Team, 2014). Descriptive statistics summarized the demographics (e.g., gender, age, ethnicity, and educational level) and, for the patient group, clinical (e.g., medication status) characteristics of both samples.

Group differences were tested as follows. After confirming that the continuous variables (age, education) violated normality assumptions in both groups via Shapiro-Wilk tests ($p < 0.05$), we employed the non-parametric Mann-Whitney U test for comparisons, reporting the U statistic, standardized Z score, and exact p-value. For the categorical variable (gender), differences were assessed using Pearson's χ^2 test, reporting the χ^2 and p-values. The significance level was set at $p < 0.05$ for all tests.

Three methods were employed to: (1) determine whether social cognitive deficits were present in the SZ group compared with HC group, (2) examine the relationship between these deficits and clinical symptoms, and (3) identify core features of social cognitive dysfunction in SZ. First, the nonparametric Mann-

Whitney U test was used for all between-group comparisons, with multiple comparisons using the Bonferroni method to control the Type I error rate. To visually illustrate group differences, radar plots (Radar Chart) were employed to compare standardized scores across multiple social cognitive domains (e.g., emotion recognition, ToM, social perception, and attributional style) between SZ and HC groups. Radar plots were generated using the ggplot2 (Wickham, 2016) and ggradar (v0.2; Bion, 2021) packages in R. For this, raw scores were normalized using Min-Max scaling to range from 0 to 1. This normalization was based on the minimum and maximum values from both SZ and HC groups, ensuring direct comparison on the same scale. Next, Kendall correlation analysis was conducted to clarify the relationship between social cognitive deficits and clinical symptoms in SZ.

Additionally, network analysis was used to identify core deficits in social cognition. A correlation network model[38] using the qgraph package in R Statistics for network construction, analysis, and visualization. In this model, each of the 17 social cognitive variables was represented as a node, and the associations between pairs of variables were represented as edges. The network included variables from four key domains: five Theory of Mind, six Attributional Style, five Emotional Recognition, and one Social Perception. Centrality measures (strength, betweenness, and closeness centrality) were calculated to identify the most influential nodes within the network[38]. Strength centrality (Str) represents the total sum of the absolute edge weights connected to a node, indicating its overall connectivity and

direct influence within the network. Betweenness centrality (Bet) quantifies how often a node acts as a bridge along the shortest paths between other nodes, indicating its role in promoting communication and maintaining network connectivity. Nodes with higher betweenness centrality exert greater influence over the flow of information in the network. Closeness centrality (Clo) reflects how close a node is to all other nodes, which is calculated as the inverse of the total shortest path distances from that node to others. Nodes with higher closeness centrality have shorter average distances to all other nodes, allowing for more efficient information dissemination. The stability of centrality indices was assessed using a case-dropping bootstrap (2,500 iterations), and only indices with a correlation stability coefficient (CS) ≥ 0.25 were considered interpretable. Consistent with prior research, the top four centrality indices were identified as core features [39].

RESULTS

Demographic and basic clinical data

The sample included 136 participants, comprising 68 in the SZ group (35 men and 33 women) and 68 in the HC group (equal numbers of men and women). The SZ group had an average illness duration of 72 months. All but one patient were receiving stable pharmacological treatment at the time of assessment (Table 1). The most frequently prescribed antipsychotics were second-generation agents, including olanzapine, aripiprazole, risperidone, and paliperidone. Additionally, 47% of SZ

were also taking antidepressants, and 4% were prescribed anxiolytics.

Group Differences in Social Cognition Scores

The normality of all continuous social cognitive variables was assessed using the Kolmogorov-Smirnov test. Results indicated a predominantly non-normal data structure, with most variables significantly deviating from normality in at least one group (all $p < 0.05$). Specifically, only three measures— Faux Pas task in the patient group, Theory of Mind composite score in the patient group, and negative-globality attribution in the healthy controls—approached normality (all $p > 0.05$, $D < 0.1$). Given the overall pattern of non-normality across the variables, non-parametric statistical tests (Mann-Whitney U) were selected for all between-group comparisons.

The non-parametric tests (Mann-Whitney U) revealed significant differences between the SZ group and the HC group in terms of several ToM tasks, including first- and second-order cognitive ToM, first- and second-order affective ToM, the false belief tasks, and the faus pas tasks. The radar plot visualization further demonstrated that the HC group scored significantly higher than the SZ group in three key domains: emotion recognition, ToM, and social perception, which aligns with the non-parametric test results (Supplementary Figure S1). Regarding attributional style, individuals with SZ showed significant differences compared with HCs in positive internality, stability, and globality. Significant group differences were also found in the recognition of specific emotions (fear, anger, sadness, and disgust), as well as in social perception abilities (Table 2).

Correlation Between Social Cognition and Clinical Symptoms

Kendall correlation analysis was performed between the social cognition scores and clinical symptom scores in the SZ group. The results showed a significantly negative correlation between the stability attribution for negative events and the general psychopathology subscale of PANSS (τ = -0.25, $P < 0.05$) (Figure 1).

Network Analysis

Network analysis of social cognition in SZ identified core deficits in second-order cognitive ToM (Bet = 10, Clo = 0.017, Str = 4.87, Exp = 4.56), fear emotion recognition (Bet = 11, Clo = 0.015, Str = 4.31, Exp = 3.30), sadness emotion recognition (Bet = 3, Clo = 0.015, Str = 3.95, Exp = 3.84), and happiness emotion recognition (Bet = 11, Clo = 0.016, Str = 3.81, Exp = 1.84). Among them, second-order cognitive ToM exhibited the highest strength and closeness (Figure 2). The CS coefficients for the centrality measures were 0.041 for betweenness, 0.122 for closeness, and 0.204 for strength. Since the CS values for betweenness and closeness are below the recommended threshold of 0.25, their interpretation is not advised. While the CS value for strength is slightly below this threshold, it shows a relatively higher degree of stability under subsampling, allowing for cautious interpretation.

DISCUSSION

This study investigated the social cognitive performance of individuals with SZ and its relationship with clinical symptoms. The findings indicated significant

deficits in social cognitive skills, consistent with previous studies [5,40].

In the analysis of the correlation between various social cognitive factors and clinical symptoms in SZ, a significantly negative correlation was found between the stability of attribution for negative events in social cognitive functioning and patients' general pathology, contrasting with previous research findings. Previous research demonstrated that individuals with SZ are more likely to attribute negative events to stable and enduring causes, and a greater symptom severity is associated with an increased tendency to make such attributions[41]. First, this may relate to the specific symptom profile, as different symptom patterns may be linked to distinct attributional styles. For instance, previous research has suggested that schizophrenia is associated with attributional biases, which are particularly linked to psychotic symptoms, especially delusions. Specifically, individuals with persecutory delusions have been found to be more likely to exhibit self-serving attributional biases. [42]. Moreover, some studies have suggested that the relationship between attributional biases and delusions is more complex. Even among patients with delusional symptoms, variations in delusion types can lead to different attributional styles, highlighting the importance of considering individual symptom patterns in research [43]. Additionally, while maladaptive, attributing negative events to stable causes may provide individuals with a sense of predictability about the world. This, to some extent, reduces their endless exploration and rumination about the causes of events, thereby objectively buffering the transformation of daily stress into clinical distress.

This mechanism is similar to cognitive reframing in resilience theory, which involves altering the cognitive appraisal of stressful events to mitigate their psychological impact[44]. Finally, prior studies have predominantly concentrated on the internal-external dimension of attributional style, with less emphasis on the stability dimension. Therefore, further research is needed to explore the role of attributional biases in the social cognitive deficits of SZ.

In contrast to attributional biases, extensive research has examined the relationship between ToM and clinical symptoms in SZ. Studies have shown that ToM is associated with clinical symptoms, with patients exhibiting more severe negative symptoms tending to underestimate others' mental states [45-49], while those with more severe positive symptoms may overinterpret others' mental states [50]. A meta-analysis also found that, in addition to negative symptoms, disorganization is significantly associated with ToM [18]. However, the current research findings are heterogeneous, with some studies showing no significant relationship between ToM and clinical symptoms of SZ [51,52]. The lack of significant correlations between Theory of Mind, emotion recognition, social perception, and clinical symptoms in this study may be due to several factors. Firstly, assessment tools have not been standardized. Previous studies used various measurement instruments, many of which lack thorough validation and standardization. Additionally, most of these instruments were developed abroad, and cultural differences may affect their reliability and validity in domestic settings [53]. Second, the conceptualization of

clinical symptoms is heterogeneous. Both this study and most prior research used the original three PANSS subscales, while few have adopted the five-factor model [17]. This limitation may hinder the establishment of a more precise association between social cognition and clinical symptoms. Finally, the restricted range of PANSS scores in our sample may have reduced statistical power. The relatively concentrated distributions of positive symptoms, general psychopathology, and total scores limit variability, and restricted range is known to attenuate correlation coefficients.

This study used network analysis to explore the core symptoms of social cognitive deficits in individuals with SZ. Network analysis addresses a key limitation of traditional psychopathology, which often overlooks the potential direct interactions among symptoms. By considering symptoms as interconnected network nodes, this approach offers a novel perspective for examining the complex relationships among symptoms through the analysis of these nodes and their connections [54].

The network analysis identified second-order ToM as a node with high strength and closeness centrality, although its relative centrality should be interpreted cautiously due to stability coefficients (CS) falling below the recommended threshold. Despite this, the prominence of second-order ToM aligns with the view that its deficits are a core feature of social cognitive dysfunction in this population. Compared with other psychiatric disorders, the effect size and severity of ToM

deficits in SZ are greater [55,56], and these deficits are detectable across all stages of the disorder [57,58]. A meta-analysis has shown that the SZ group exhibits overall deficits in ToM compared to non-clinical control groups [59,60]. Some scholars have suggested using ToM as a framework to explain certain SZ symptoms, hypothesizing that psychotic symptoms may be the external manifestation of underlying ToM deficits [61]. However, there is still controversy regarding the specific components of ToM deficits in SZ; while it is generally accepted that first-order ToM is relatively preserved compared to second-order ToM, other studies suggest that the structure of ToM may be nonhierarchical [62,63].

This study also has certain limitations. Firstly, it is not a large-sample study, and the relatively small number of participants limits statistical power and the generalizability of the results. Future research should therefore include larger and more diverse samples to draw more robust conclusions. Secondly, limitations related to the assessment of social cognition, including those originally noted in the manuscript, warrant further emphasis. Assessment tools in this field remain underdeveloped, particularly for social perception. In domestic research contexts, standardized instruments are lacking, and researchers often rely on abridged translations of commonly used international measures. In this study, because the primary aim was preliminary screening, abbreviated and modified versions of social-cognition tasks were employed, which naturally introduced certain constraints. Although these tasks were culturally adapted for the Chinese context,

comprehensive psychometric validation—such as internal consistency, test–retest reliability, and construct validity—has not yet been completed. These factors may have resulted in incomplete or less precise evaluations of specific subdomains of social cognition, contributing to inconsistencies both within our findings and relative to previous literature. Moreover, laboratory-based tasks may lack ecological validity, as they typically rely on static or highly structured stimuli that may not reflect the complexity of real-world social interactions. Future research should therefore incorporate multi-method assessments, such as naturalistic observations, virtual reality paradigms, or dynamic social simulations, following preliminary screening to enhance ecological validity and deepen the understanding of social-cognitive profiles. Thirdly, the study did not systematically control for the potential confounding effects of antipsychotic medications, including types, dosages, or treatment durations, which may influence both social cognitive performance and clinical symptoms. Future research should include detailed pharmacological records or examine individuals at illness onset who are medication-naïve to better clarify illness-related mechanisms. Fourthly, sample selection procedures may have introduced certain selection biases. To ensure assessment stability and participant safety, individuals with acute agitation, suicidal risk, or comorbid substance use disorders were excluded. Additionally, matching the schizophrenia group and healthy controls by years of education, although important for isolating the influence of educational level, may have constrained the range of clinical heterogeneity and

symptom severity within the sample. These procedures may thus limit the generalizability of the findings and attenuate the observed associations between social cognition and clinical symptoms. Future work should aim to validate these findings in more diverse clinical samples to examine their robustness and boundary conditions. Furthermore, although social cognitive abilities may vary across different illness stages [64], this study compared only individuals with schizophrenia and healthy controls without examining stage-related differences. Future studies should explore variability across illness stages and incorporate measures of social functioning to determine how the observed patterns relate to everyday social behavior.

CONCLUSION

In summary, individuals with schizophrenia exhibited widespread deficits in social cognition, including deficits in theory of mind, emotion recognition, attributional style, and social perception. Attributional biases in social cognition were also significantly correlated with clinical symptoms. Moreover, the structure and core components of social cognitive deficits in SZ have been further clarified. Specifically, ToM deficits are central to social cognitive dysfunction, with emotion recognition serving as a bridge among other social cognitive impairments.

Given that the lack of standardized and culturally adapted assessment tools contributes to the heterogeneity of findings, future research should focus on

improving the ecological and cross-cultural validity of these measurement instruments. Further, the relationships between specific social cognitive deficits and clinical symptoms require deeper investigation, which may be achieved by using different clinical symptom rating scales and analyzing the associations between the PANSS five-factor model and social cognition.

DATA AVAILABILITY

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

REFERENCES

1. Ferrari, A. J. *et al.* Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. *The Lancet* 403, 2133-2161, doi:10.1016/s0140-6736(24)00757-8 (2024).
2. Kahn, R. S. *et al.* Schizophrenia. *Nature Reviews Disease Primers* 1, 15067, doi:10.1038/nrdp.2015.67 (2015).
3. Corbera, S. *et al.* Predictors of social functioning and quality of life in schizophrenia and autism spectrum disorder. *Psychiatry Research* 303, Article 114087, doi:10.1016/j.psychres.2021.114087 (2021).
4. Green, M. F. *et al.* Social cognition in schizophrenia. *Schizophrenia Bulletin* 34, 670–672, doi:10.1093/schbul/sbn045 (2008).
5. Green, M. F. *et al.* Social cognition in schizophrenia: an NIMH workshop on definitions, assessment, and research opportunities. *Schizophrenia Bulletin* 34, 1211–1220, doi:10.1093/schbul/sbm145 (2008).
6. Addington, J., Saeedi, H. & Addington, D. Influence of social perception and social knowledge on cognitive and social functioning in early psychosis. *The British Journal of Psychiatry* 189, 373-378, doi:10.1192/bjp.bp.105.021022 (2006).
7. Sergi, M. J., Rassovsky, Y., Nuechterlein, K. H. & Green, M. F. Social perception as a mediator of the influence of early visual processing on functional status in schizophrenia. *American Journal of Psychiatry* 163, 448–454, doi:10.1176/appi.ajp.163.3.448 (2006).
8. Giordano, G. M. *et al.* Negative symptoms and social cognition as mediators of the relationship between neurocognition and functional outcome in schizophrenia. *Frontiers in psychiatry* 15, 1333711, doi:10.3389/fpsyt.2024.1333711 (2024).
9. Mazza, M. *et al.* Could schizophrenic subjects improve their social cognition abilities only with observation and imitation of social situations? *Neuropsychological rehabilitation* 20, 675-703, doi:10.1080/09602011.2010.486284 (2010).
10. Roberts, D. L. & Penn, D. L. Social cognition and interaction training (SCIT) for outpatients with schizophrenia: a preliminary study. *Psychiatry research* 166, 141-147, doi:10.1016/j.psychres.2008.02.007 (2009).
11. Kamp, D. *et al.* Efficacy of integrated social cognitive remediation vs neurocognitive remediation in schizophrenia: Results from the multicenter randomized controlled ISST (Integrated Social Cognition And Social Skills Therapy) study. *Schizophrenia research* 277, 44-56, doi:10.1016/j.schres.2025.02.015 (2025).
12. Pinkham, A. E. *et al.* The social cognition psychometric evaluation study: Results of the expert survey and RAND panel. *Schizophrenia Bulletin* 40, 813–823, doi:10.1093/schbul/sbt081 (2014).
13. Kubota, R. *et al.* Study protocol: The evaluation study for social cognition measures in Japan (ESCoM). *Journal of Personalized Medicine* 11, 625, doi:10.3390/jpm11070667 (2021).
14. Galderisi, S. *et al.* The influence of illness-related variables, personal resources and context-related factors on real-life functioning of people with schizophrenia. *World psychiatry : official journal of the World Psychiatric Association (WPA)* 13, 275-287, doi:10.1002/wps.20167 (2014).
15. Galderisi, S., Mucci, A., Buchanan, R. W. & Arango, C. Negative symptoms of schizophrenia: new

developments and unanswered research questions. *The lancet. Psychiatry* 5, 664-677, doi:10.1016/s2215-0366(18)30050-6 (2018).

16. Harrington, L., Siegert, R. J. & McClure, J. Theory of mind in schizophrenia: a critical review. *Cognitive neuropsychiatry* 10, 249-286, doi:10.1080/13546800444000056 (2005).
17. Cayouette, A., Thibaudeau, É., Cellard, C., Roy, M. A. & Achim, A. M. Associations between theory of mind and clinical symptoms in recent onset schizophrenia spectrum disorders. *Frontiers in Psychiatry* 14, Article 1044682, doi:10.3389/fpsyt.2023.1044682 (2023).
18. Thibaudeau, E., Rae, J., Raucher-Chéné, D., Bougeard, A. & Lepage, M. Disentangling the relationships between the clinical symptoms of schizophrenia spectrum disorders and theory of mind: A meta-analysis. *Schizophrenia Bulletin* 49, 255–274, doi:10.1093/schbul/sbac150 (2023).
19. Wu, Y., Song, S. & Shen, Y. Characteristics of theory of mind impairment and its relationship with clinical symptoms and neurocognition in patients with schizophrenia. *BMC Psychiatry* 23, doi:10.1186/s12888-023-05224-7 (2023).
20. Barbato, M. *et al.* Theory of mind, emotion recognition and social perception in individuals at clinical high risk for psychosis: Findings from the NAPLS-2 cohort. *Schizophrenia Research: Cognition* 2, 133-139, doi:10.1016/j.scog.2015.04.004 (2015).
21. Richard-Devantoy, S., Berlim, M. T. & Jollant, F. Suicidal behaviour and memory: A systematic review and meta-analysis. *The world journal of biological psychiatry : the official journal of the World Federation of Societies of Biological Psychiatry* 16, 544-566, doi:10.3109/15622975.2014.925584 (2015).
22. Hoptman, M. J. Impulsivity and aggression in schizophrenia: a neural circuitry perspective with implications for treatment. *CNS spectrums* 20, 280-286, doi:10.1017/s1092852915000206 (2015).
23. Volkow, N. D. *et al.* Effects of Cannabis Use on Human Behavior, Including Cognition, Motivation, and Psychosis: A Review. *JAMA psychiatry* 73, 292-297, doi:10.1001/jamapsychiatry.2015.3278 (2016).
24. Spinelli, S. L., Rodríguez-Testal, J. F., Cabana, Á., Romano, S. & Gómez-Sena, L. Relationship between cognitive reserve (education), social cognition and negative symptoms. *Schizophrenia research. Cognition* 42, 100379, doi:10.1016/j.scog.2025.100379 (2025).
25. Li, X. D., Xu, J., Liu, P. & Zhou, S. Z. Knowledge bias in children's false belief tasks. *Acta Psychologica Sinica* 28, 53–58 (2008).
26. Wang, Y. G., Wang, K., Zhu, C. Y. & et al. A study of theory of mind deficit in first-episode schizophrenic patient. *Chinese Journal of Neurology and Psychiatry* 32, 481–486 (2006).
27. Perner, J. & Wimmer, H. "John thinks that Mary thinks that···": Attribution of second-order beliefs by 5- to 10-year-old children. *Journal of Experimental Child Psychology* 39, 437-471, doi:10.1016/0022-0965(85)90051-7 (1985).
28. Wimmer, H. & Perner, J. Beliefs about beliefs: representation and constraining function of wrong beliefs in young children's understanding of deception. *Cognition* 13, 103-128, doi:10.1016/0010-0277(83)90004-5 (1983).
29. Zheng, M., Pu, C., Yu, X. & Shi, C. Impaired theory of mind in individuals at ultra-high risk for schizophrenia. *Chinese Journal of Psychiatry* 49, 6 (2016).
30. Mo, S. & Su, Y. False belief understanding in 3- to 4-year-old children: The roles of complement syntax, semantic understanding, and working memory. *Psychological Development and Education* 25, 15–19, 25 (2009).

31. Slade, L. & Ruffman, T. How language does (and does not) relate to theory of mind: A longitudinal study of syntax, semantics, working memory and false belief. *British Journal of Developmental Psychology* 23, 117–141, doi:10.1348/026151004X21332 (2005).
32. Baron-Cohen, S., O'Riordan, M., Stone, V., Jones, R. & Plaisted, K. Recognition of faux pas by normally developing children and children with Asperger syndrome or high-functioning autism. *Journal of autism and developmental disorders* 29, 407-418, doi:10.1023/a:1023035012436 (1999).
33. Lu, B., Hui, M., Yuxia, H. & Yuejia, L. Compilation of China Emotional Picture System—Trial among 46 China college students. *Chinese Journal of Mental Health* 19, 4 (2005).
34. Guo, W., Yao, S., Yi, J., Peng, C. & Yang, B. Preliminary development of attribution style questionnaire. *Chinese Journal of Clinical Psychology* 2, 92–95 (2003).
35. Sergi, M. J. *et al.* Development of a measure of relationship perception in schizophrenia. *Psychiatry Research* 166, 54–62, doi:10.1016/j.psychres.2008.03.010 (2009).
36. Pinkham, A. E., Penn, D. L., Green, M. F. & Harvey, P. D. Social Cognition Psychometric Evaluation: Results of the Initial Psychometric Study. *Schizophrenia bulletin* 42, 494-504, doi:10.1093/schbul/sbv056 (2016).
37. Vaskinn, A., Ueland, T., Melle, I. & Sundet, K. Sex differences in social cognition among individuals with schizophrenia and in healthy control participants: a secondary analysis of published data. *Archives of women's mental health* 27, 661-667, doi:10.1007/s00737-024-01422-8 (2024).
38. Epskamp, S., Borsboom, D. & Fried, E. I. Estimating psychological networks and their accuracy: A tutorial paper. *Behavior research methods* 50, 195-212, doi:10.3758/s13428-017-0862-1 (2018).
39. Mullarkey, M. C., Marchetti, I. & Beevers, C. G. Using network analysis to identify central symptoms of adolescent depression. *Journal of Clinical Child & Adolescent Psychology* 48, 656–668, doi:10.1080/15374416.2018.1437735 (2019).
40. Weinreb, S., Li, F. & Kurtz, M. M. A meta-analysis of social cognitive deficits in schizophrenia: Does world region matter? *Schizophrenia Research* 243, 206–213, doi:10.1016/j.schres.2022.04.002 (2022).
41. Lysaker, P. H., Lancaster, R. S., Nees, M. A. & Davis, L. W. Attributional style and symptoms as predictors of social function in schizophrenia. *Journal of Rehabilitation Research and Development* 41, 225–232, doi:10.1682/jrrd.2004.02.0225 (2004).
42. Bentall, R. P., Corcoran, R., Howard, R., Blackwood, N. & Kinderman, P. Persecutory delusions: A review and theoretical integration. *Clinical Psychology Review* 21, 1143-1192, doi:10.1016/s0272-7358(01)00106-4 (2001).
43. Jolley, S. *et al.* Attributional style in psychosis—The role of affect and belief type. *Behaviour Research and Therapy* 44, 1597–1607, doi:10.1016/j.brat.2005.12.002 (2006).
44. Southwick, S. M. & Charney, D. S. The science of resilience: implications for the prevention and treatment of depression. *Science (New York, N.Y.)* 338, 79-82, doi:10.1126/science.1222942 (2012).
45. Oliver, L. D. *et al.* Lower- and higher-level social cognitive factors across individuals with schizophrenia spectrum disorders and healthy controls: Relationship with neurocognition and functional outcome. *Schizophrenia Bulletin* 45, 629–638, doi:10.1093/schbul/sby114 (2019).
46. Green, M. F. From social cognition to negative symptoms in schizophrenia: How do we get there from here? *Schizophrenia Bulletin* 46, 225-226, doi:10.1093/schbul/sbz113 (2020).
47. Griffiths, S. L., Birchwood, M., Khan, A. & Wood, S. J. Predictors of social and role outcomes in first

episode psychosis: A prospective 12-month study of social cognition, neurocognition and symptoms. *Early Intervention in Psychiatry* 15, 993–1001, doi:10.1111/eip.13056 (2021).

48. Weijers, J. *et al.* Mentalizing impairment as a mediator between reported childhood abuse and outcome in nonaffective psychotic disorder. *Psychiatry Research* 259, 463–469, doi:10.1016/j.psychres.2017.11.010 (2018).
49. Raucher-Chéné, D., Thibaudeau, E., Sauvé, G., Lavigne, K. M. & Lepage, M. Understanding others as a mediator between verbal memory and negative symptoms in schizophrenia-spectrum disorder. *Journal of Psychiatric Research* 143, 429–435, doi:10.1016/j.jpsychires.2021.10.007 (2021).
50. Peyroux, E. *et al.* From "under" to "over" social cognition in schizophrenia: Is there distinct profiles of impairments according to negative and positive symptoms? *Schizophrenia Research: Cognition* 15, 21–29, doi:10.1016/j.scog.2018.10.001 (2018).
51. Brüne, M., Abdel-Hamid, M., Lehmkämper, C. & Sonntag, C. Mental state attribution, neurocognitive functioning, and psychopathology: What predicts poor social competence in schizophrenia best? *Schizophrenia Research* 92, 151–159, doi:10.1016/j.schres.2007.01.020 (2007).
52. Ziv, I., Leiser, D. & Levine, J. Social cognition in schizophrenia: Cognitive and affective factors. *Cognitive Neuropsychiatry* 16, 71–91, doi:10.1080/13546805.2010.492693 (2011).
53. Vita, A. *et al.* European Psychiatric Association guidance on assessment of cognitive impairment in schizophrenia. *European Psychiatry* 65, e58, doi:10.1192/j.eurpsy.2022.2316 (2022).
54. Cai, Y. Q., Dong, S. Y., Yuan, S. & Hu, C. P. Network analysis models of variables and their applications. *Advances in Psychological Science* 28, 18 (2020).
55. Van Rheenen, T. E., Ganella, E. P., Bauer, I. E. & Bartholomeusz, C. F. in *Social cognition in psychosis: Interpersonal and clinical foundations* (ed D. L. & Penn Roberts, D. L.) 1-36 (2019).
56. de Sales, S. C. *et al.* Social cognition and psychosocial functioning in schizophrenia and bipolar disorder: Theory of mind as a key to understand schizophrenia dysfunction. *European Neuropsychopharmacology* 77, 12-20, doi:10.1016/j.euroneuro.2023.02.001 (2023).
57. Canty, A. L., Neumann, D. L. & Shum, D. H. K. Using virtual reality to assess theory of mind subprocesses and error types in early and chronic schizophrenia. *Schizophrenia Research: Cognition* 10, 15-19, doi:10.1016/j.scog.2017.06.001 (2017).
58. Canty, A. L., Cao, Y., Neumann, D. & Shum, D. H. K. The functional significance of cognitive empathy and theory of mind in early and chronic schizophrenia. *Psychiatry Res.* 299, Article 113852, doi:https://doi.org/10.1016/j.psychres.2021.113852 (2021).
59. Savla, G. N., Vella, L., Armstrong, C. C., Penn, D. L. & Twamley, E. W. Deficits in domains of social cognition in schizophrenia: A meta-analysis of the empirical evidence. *Schizophrenia Bulletin* 39, 979–992, doi:10.1093/schbul/sbs080 (2013).
60. Ventura, J., Ered, A., Gretchen-Doorly, D. & Subotnik, K. L. Theory of mind in the early course of schizophrenia: Stability, symptom and neurocognitive correlates, and relationship with functioning. *Psychological Medicine* 45, 2031–2043, doi:10.1017/S0033291714003171 (2015).
61. Frith, C. Neuropsychology of schizophrenia: What are the implications of intellectual and experiential abnormalities for the neurobiology of schizophrenia? *British Medical Bulletin* 52, 618–626, doi:10.1093/oxfordjournals.bmb.a011571 (1996).
62. Mehta, U. M. *et al.* Cognitive deconstruction of parenting in schizophrenia: The role of theory of mind.

Australian & New Zealand Journal of Psychiatry 48, 249–258, doi:10.1177/0004867413500350 (2014).

63. Ho, K. K. *et al.* Theory of mind impairments in patients with first-episode schizophrenia and their unaffected siblings. *Schizophrenia Research* 166, 1–8, doi:10.1016/j.schres.2015.05.033 (2015).
64. Diez-Alegría, C., Vázquez, C., Nieto-Moreno, M., Valiente, C. & Fuentenebro, F. Personalizing and externalizing biases in deluded and depressed patients: Are attributional biases a stable and specific characteristic of delusions? *British Journal of Clinical Psychology* 45, 531–544, doi:10.1348/014466505X86681 (2006).

CONFLICT OF INTERESTS.

The authors declare no competing interests.

FUNDING

This study was supported by the Project supported by the Beijing Hospitals Authority Clinical Medicine Development with special funding support, code: ZLRK202335.

AUTHOR'S CONTRIBUTION STATEMENT

Danni He, Douyu Zhang and Qijing Bo wrote the paper. Tian Li, Zhen Mao, Yimeng Wang, Fuchun Zhou , Fang Dong and Chuanyue Wang designed the study. Yujie Xing, Zhiqi Liu, Chenyu Zhou, Lei Zhao and Feng Li collected the data. Yushen Ding and Xiaoyao Wang performed all analyses. All authors reviewed the paper.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge all contributors to this cross-sectional study. We extend our deepest appreciation to the individuals diagnosed with schizophrenia and their families for their essential participation. Special thanks are accorded to the clinical teams, particularly the psychiatrists and nurses who conducted standardized diagnostic evaluations and psychometric assessments.

Figure legends

Figure 1. Correlations of negative situational stability attribution styles and general psychopathology scores. The analysis identified a significantly negative correlation between the attributional stability for negative events and the general psychopathology scores (r = -0.25, P< 0.05)

Figure 2. Network analysis of social cognition

a. Network structure of impaired social cognitive domains.

b. Centrality metrics of social cognitive domains.

ASQ1: internal attribution of negative situation; ASQ2: stable attribution of negative situation; ASQ3: global attribution of negative situations; ASQ4: internal attribution of positive situation; ASQ5: stable attribution of positive situation; ASQ6: global attribution of positive situation; ER1: happy; ER2: scale; ER3: angry; ER4: sadness; ER5: disgust; FBT1: first-order cognitive theory of mind; FBT2: second-order cognitive theory of mind; FBT3: first-order emotional psychology; FBT4: second-order emotional psychology; FPT: The Faux Pas Task; SP: social perception.

Table 1. Demographic and clinical characteristics of SZ and HC groups.

	SZ（n = 68）	HC（n = 68）	U/χ^2	Z	p
Gender			0.12		0.73
male	35（51%）	35（51%）			
female	33（49%）	33（49%）			
Age (years)	28.82 ± 8.53	28.29 ± 8.53	2210.5	-0.442	0.66
Years of education	13.613± 2.39	14.29 ± 2.23	1981.5	-0.658	0.14
Duration of illness (months)	72.07 ± 63.32				
PANSS					
positive symptoms	21.61 ± 5.27				
negative symptoms	17.69 ± 6.94				
General Psychopathology	36.91 ± 8.02				
PANSS total score	76.15 ± 16.32				
Medication status					
Antipsychotics	64（98%）				
Anxiolytics	3（4%）				
Antidepressants	32（47%）				
Unmedicated	1（1%）				

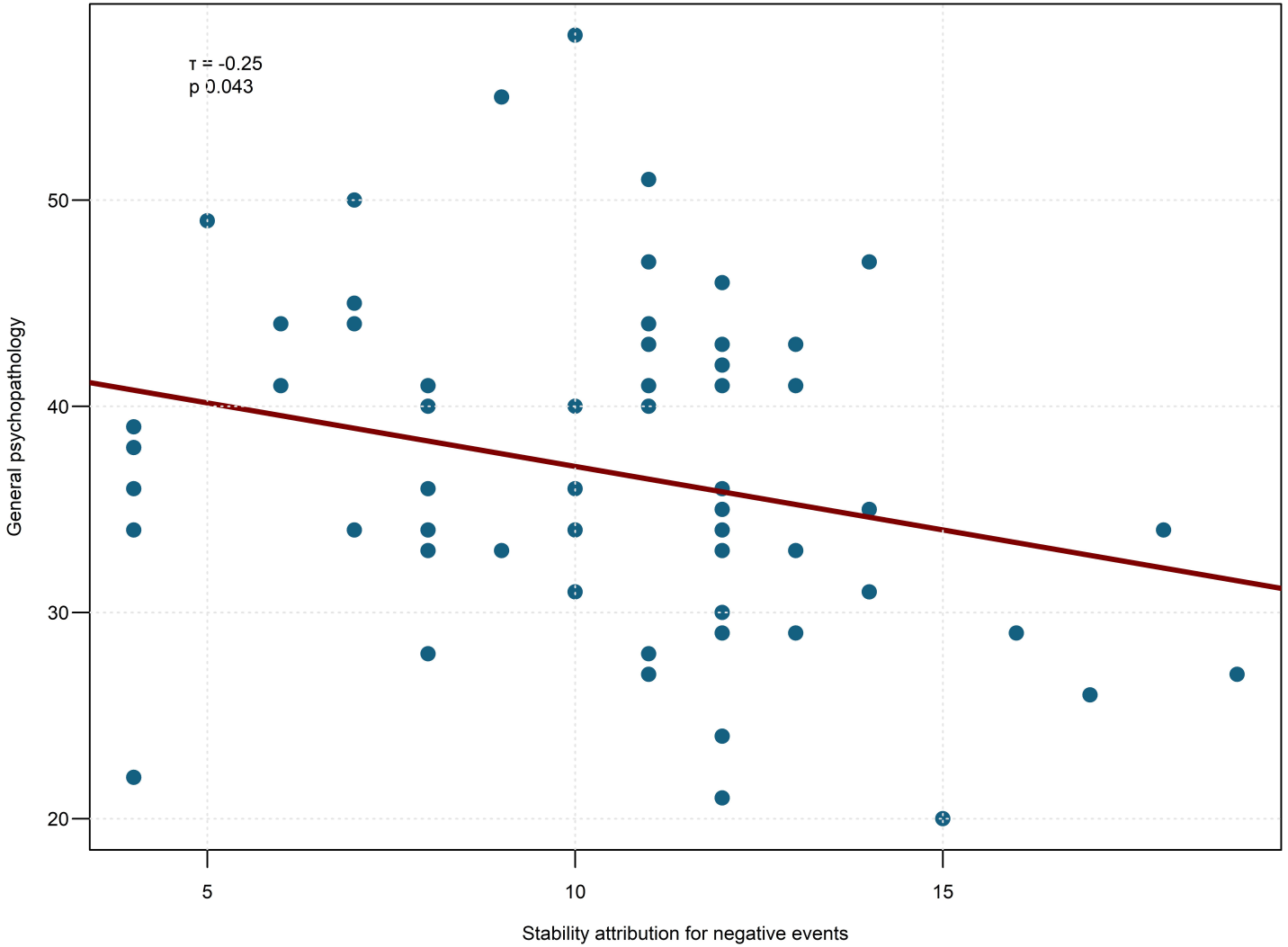
Note. SZ, group with schizophrenia; HC, healthy control group

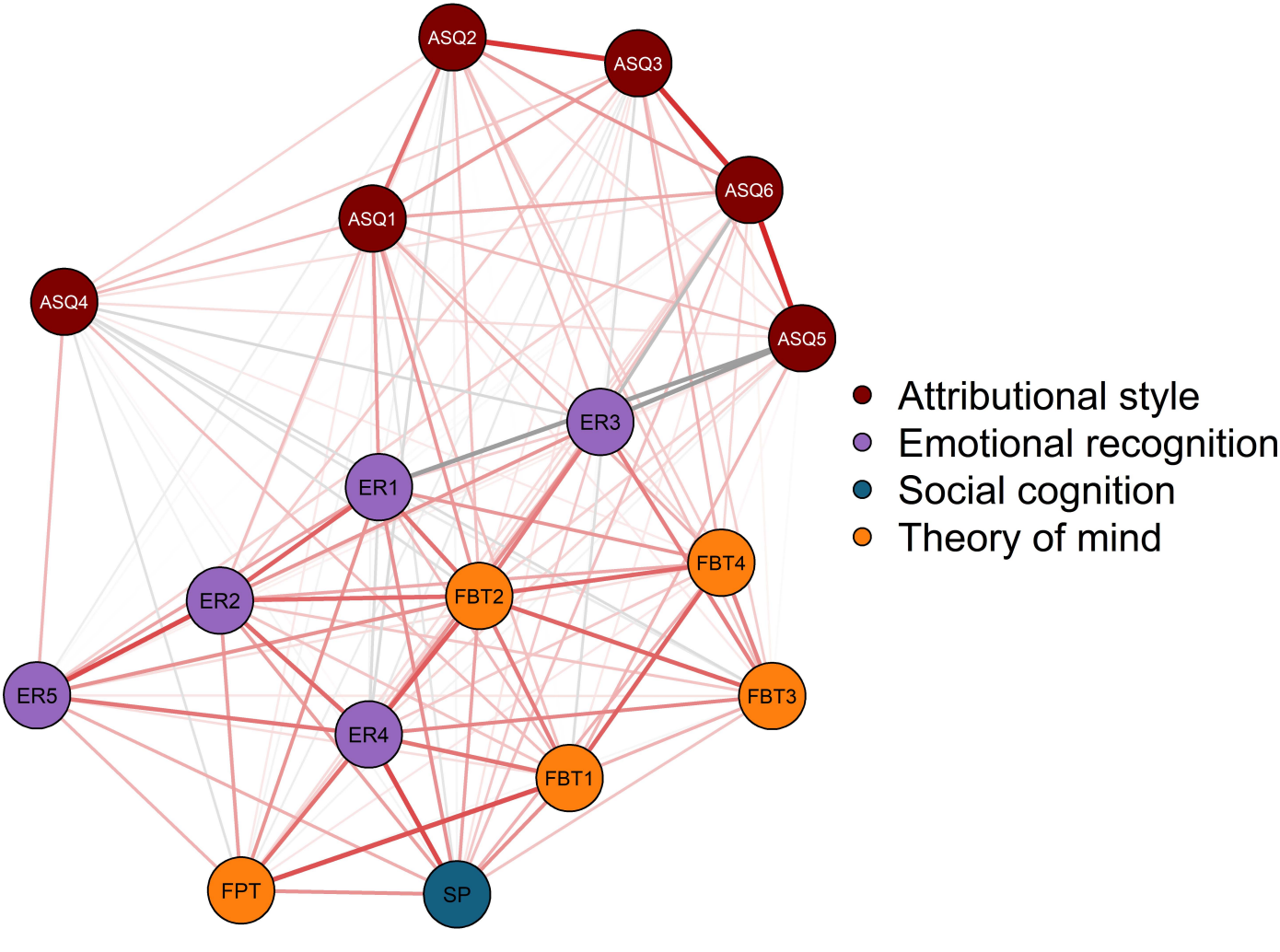
Table 2. Comparison of social cognitive scores between two groups

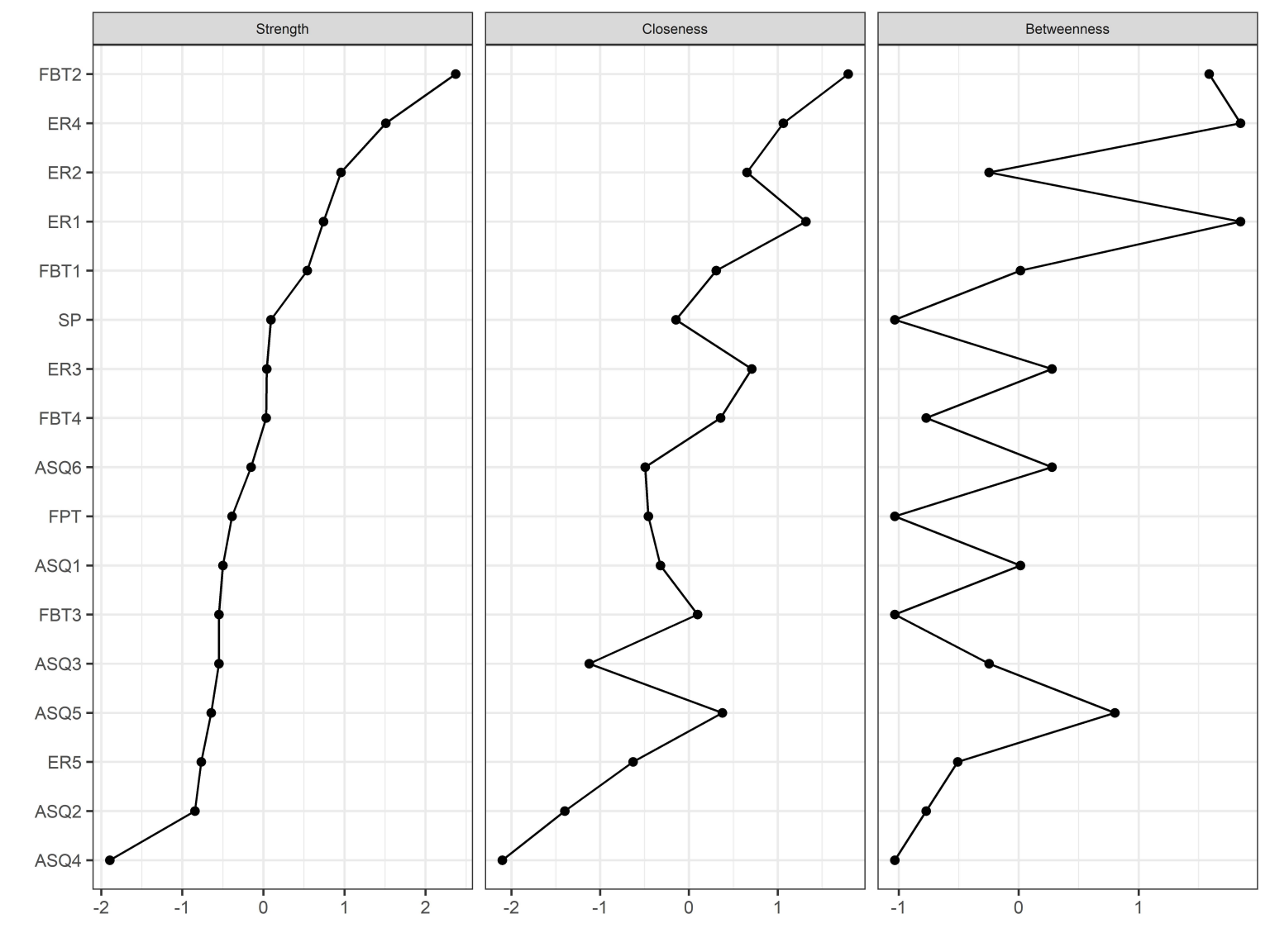
	SZ M (Q1˜Q3) (n=68)	HC M (Q1˜Q3) (n=68)	Z	p
Theory of mind:	38.72 (31˜46)	50.26 (48˜54.75)	-7.33	<0.001

The false-belief task	9.9 7 (8˜12)	12.75 (11.25˜14)	-5.54	<0.001
1-cog-TOM	3.21 (3˜4)	3.68 (3˜4)	-3.41	0.001
2-cog-TOM	2.60 (2˜3)	3.29 (3˜4)	-3.78	<0.001
1-emo-TOM	2.26 (2˜3)	3.88 (2˜4)	-3.37	0.001
2-emo-TOM	1.91 (1˜3)	2.91 (2˜4)	-5.05	<0.001
The Faux Pas Task	28.73 (21˜36)	37.51 (35.25˜41)	-6.66	<0.001
Attributional style:	62.06 (55.25˜71.75)	63.84 (56˜72)	-0.24	0.812
Neg-in	10.45 (8˜12)	10.12 (9˜12)	-0.75	0.451
Neg-sta	10.45 (8˜12)	9.72 (7˜12)	-1.28	0.200
Neg-glo	10.2 (7˜12)	9.5 (6˜12)	-1.22	0.223
Pos-in	9.83 (7.75˜12)	8.16 (5˜10)	-3.16	0.002
Pos-sta	11.94 (10˜14)	14.32 (11˜17)	-3.58	<0.001
Pos-glo	11.55 (8˜14)	12.97 (11˜16)	-2.25	0.025
Emotional recognition:	7.40 (7˜9)	8.74(8˜10)	-4.73	<0.001
happy	1.91 (2˜2)	1.97 (2˜2)	-1.45	0.146
scare	1.57 (1˜2)	1.85 (2˜2)	-3.31	0.001
angry	1.34 (1˜2)	1.78 (2˜2)	-4.31	<0.001
sadness	1.43 (1˜2)	1.76 (2˜2)	-2.63	0.009
disgust	1.15 (1˜2)	1.43 (1˜2)	-2.28	0.022
Social perception	17.23 (14˜19)	21.12 (19˜23.75)	-5.41	<0.001

Note: 1-cog-TOM: first-order cognitive theory of mind; 2-cog-TOM: second-order cognitive theory of mind; 1-emo-TOM: first-order emotional psychology; 2-emo-TOM: second-order emotional psychology; Neg in: internal attribution of negative situation; Neg sta: stable attribution of negative situation; Neg glo: global attribution of negative situations; Pos in: internal attribution of positive situation; Pos sta: stable attribution of positive situation; Pos glo: global attribution of positive situation.


τ = -0.25
p 0.043
General psychopathology
50
40
30
20
5
10
15
Stability attribution for negative events


ASQ2
ASQ3
ASQ6
ASQ1
ASQ4
ASQ5
ER3
ER1
FBT4
ER2
FBT2
ER5
FBT3
ER4
FBT1
FPT
SP
Attributional style
Emotional recognition
Social cognition
Theory of mind


Strength
Closeness
Betweenness
FBT2
ER4
ER2
ER1
FBT1
SP
ER3
FBT4
ASQ6
FPT
ASQ1
FBT3
ASQ3
ASQ5
ER5
ASQ2
ASQ4
-2
-1
0
1
2
-2
-1
0
1
-1
0
1