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〔Original Article〕

# Reduced schematic inference in patients with social anxiety disorder

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**Abstract**

**【Purpose】Previous studies on social cognitive function in Social Anxiety Disorder（SAD）yielded inconsistent results. Therefore, this study hypothesized that patients with SAD exhibit social cognitive function impairments, which may contribute to the disorder's core symptoms. This study aimed to evaluate various aspects of social cognitive function in patients with SAD.**

**【Patients and methods】This study included 27 patients with SAD（mean age = 26.19 ± 7.97 years）and 27 healthy controls（HC）（mean age = 27.89 ± 11.03 years）matched for age, sex, and intelligence quotient. Social cognitive function was assessed using the Social Cognition Screening Questionnaire（SCSQ）（Japanese version）, which evaluates non-social cognitive abilities（working memory and schematic inference）and social cognitive domains（Theory of Mind（ToM）, metacognition, and hostile attributional bias）.**

**【Results】The total SCSQ（U = 219, $p$ = 0.012, r = -0.34）and schematic inference scores（U = 200.5, $p$ = 0.003, r = -0.40）were significantly lower in the SAD group than in the HC group. Even after adjusting for Autism-Spectrum Quotient and Beck Depression Inventory Second Edition scores, the schematic inference scores remained significantly lower in the SAD group（$F(1, 54) = 7.80$, $p$ = 0.007, $\eta^2p = 0.14$）, while no significant differences were observed in the total scores（$F(1, 54) = 0.95$, $p$ = 0.333, $\eta^2p = 0.02$）. Social cognitive abilities were preserved in patients with SAD.**

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**【Conclusions】The findings suggest that patients with SAD may experience specific difficulties in interpreting ambiguous or uncertain information, potentially leading to misunderstandings and heightened anxiety during social interactions. This selective impairment in schematic inference, while preserving other social cognitive abilities, emphasizes a unique cognitive profile in patients. Clinicians should consider evaluating and adjusting for autistic traits and depressive symptoms when assessing the cognitive profile of patients with SAD, as these factors may contribute to their social difficulties and inform treatment strategies.**

***Key words*: social anxiety disorder, social functioning, social cognition screening questionnaire, non-social cognitive function, interpretation bias**

## Ⅰ. Introduction

Social Anxiety Disorder（SAD）is marked by a profound fear of social interactions where the individual may be observed or judged by others, often resulting in significant discomfort and a persistent fear of negative evaluation[1]. This anxiety is frequently disproportionate to the actual threat posed by the social situation, and can severely impair daily social activities and relationships[2]. Additionally, SAD is one of the most prevalent anxiety disorders, with an estimated 12-month prevalence rate of approximately 7% in the United States[3].

Given that SAD is marked by a strong fear of negative evaluation in social situations, exploring the role of social cognition in SAD may offer valuable insights. Social cognition encompasses the mental processes involved in social interactions, such as perceiving, interpreting, and responding to the intentions, dispositions, and behaviors of others[4]. Impairments in social cognition are a hallmark of several psychiatric disorders and are included as key diagnostic criteria for certain conditions[5]. Disorders characterized by social functional impairments include schizophrenia and autism spectrum disorder（ASD）[6,7]. Recent studies suggested that SAD may also involve deficits in social cognition, particularly in areas such as Theory of Mind（ToM）, emotion recognition, and social cue interpretation[8-13], which may contribute to its core symptoms. Understanding these deficits is essential, given that they may explain the functional impairments in SAD, such as decreased life satisfaction and reduced quality of life. Clarifying these mechanisms may assist in the development of more targeted and effective treatment strategies[14]. However, previous studies on social cognition in SAD yielded inconsistent results, with some studies reporting significant differences between SAD and healthy control groups[8-13]and others finding no significant differences[15-18]. These inconsistencies may be owing to variations in the types of social cognitive tasks used, or methodological differences across studies[19]. Consequently, further research is needed to clarify the nature and extent of social cognitive deficits in SAD.

Roberts et al., initially focusing on patients with schizophrenia, designed the Social Cognition Screening Questionnaire（SCSQ）to simultaneously assess multiple domains of both social and non-social cognition[20,21]. The SCSQ comprises subscales measuring non-social cognitive abilities, such as verbal memory and schematic inference, alongside social cognitive domains, such as ToM, metacognition, and hostile attributional bias. Administering numerous tests to evaluate social cognition can be taxing for patients. Therefore, the SCSQ offers a practical and efficient approach to evaluating multiple aspects of social cognition in SAD, benefiting both clinicians and patients by providing a comprehensive yet manageable assessment. A Japanese version of the SCSQ, which has demonstrated strong reliability and validity[22], has also been used to assess social cognition in disorders other than schizophrenia[23,24]. Regarding clinical

practice, utilizing the SCSQ allows for a more tailored treatment approach based on a holistic evaluation of the patient's specific needs.

This study's primary aim was to evaluate both social and non-social cognitive functions in patients with SAD to clarify their cognitive profile. This study hypothesized that, similar to schizophrenia and ASD, patients with SAD may experience social cognitive function impairments, which may contribute to their core symptoms. To examine this, the SCSQ, a tool designed to assess multiple domains, including social cognition (e.g., ToM, metacognition, and hostile attributional bias) and non-social cognition (e.g., verbal memory and schematic inference), was employed.

## Ⅱ. Material and methods

### Participants

This study included 27 patients with SAD for whom the SCSQ data were available and 27 healthy controls (HC) matched for age, sex, and intelligence quotient (IQ). The SAD group was diagnosed by a psychiatrist using the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5)[1]. The inclusion criteria required SAD symptoms to be the primary cause of impairment in daily life, with any comorbid conditions being secondary. To ensure homogeneity in intellectual ability, participants with an IQ between 80 and 120 were selected. The exclusion criteria for the SAD group included: (1) a history of organic brain disorders, (2) imminent risk of suicide, and (3) severe psychiatric disorders requiring hospitalization. The SAD and HC groups were recruited through the participant recruitment site operated by the Department of Cognitive Behavioral Physiology, Graduate School of Medicine, Chiba University and the Research Center for Child Mental Development, Chiba University. Additionally, patients in the SAD group were invited to participate prior to their treatment at the Cognitive Behavioral Therapy Center, Chiba University Hospital. Participants with a history of psychiatric disorders were excluded from the HC group. In the SAD group, eleven patients had a diagnosis of SAD only, while others had comorbidities, including major depressive disorder (six patients), generalized anxiety disorder (four patients), persistent depressive disorder (dysthymia) (two patients), agoraphobia (six patients), panic disorder (two patients), eating disorder (one patient), and bipolar disorder (one patient). Four patients in the SAD group had two or more comorbidities. Regarding the SAD group, seven patients were not on medication, while the remaining 20 were receiving pharmacological treatment. The specific comorbidities and medications are presented in Supplementary Table S1.

Approval for this study was obtained from the Ethics Committee of the Chiba University Graduate School of Medicine, and all participants provided written informed consent.

### Measures

Participants were assessed by experienced clinical researchers using the following measures:

**Wechsler Adult Intelligence Scale - Third Edition (WAIS-III)**[25]

The Wechsler Adult Intelligence Scale-Third Edition (WAIS-III) is widely used to measure adult intellectual functioning. The Similarities and Matrix Reasoning subtests of the Japanese version of the WAIS-III were used to estimate participant IQs[26].

**Liebowitz Social Anxiety Scale (LSAS)**[27]

This study used the Japanese version of the Liebowitz Social Anxiety Scale (LSAS)[28]. The LSAS, which is widely used to assess social anxiety symptoms, comprises 24 items, each rated on a 4-point scale, with higher scores indicating more severe symptoms.

**Autism-Spectrum Quotient (AQ)**[29]

A self-report questionnaire was used to assess autism spectrum traits in adults. This study used the Japanese version of the Autism-Spectrum Quotient (AQ)[30], which comprises 50 items, each rated on a 4-point scale, with higher scores indicating stronger autistic traits.

**Beck Depression Inventory Second Edition (BDI-II)**[31]

The Beck Depression Inventory Second Edition (BDI-II) assesses depressive symptom severity. This

study used the Japanese version of the BDI-II, a 21-item self-report questionnaire[32], with higher scores indicating more severe depressive symptoms.

**SCSQ**

The Japanese version of the SCSQ (Version 3.2) was used to assess social cognition[22]. The SCSQ comprises subscales that assess both non-social cognitive domains, such as verbal memory and schematic inference, and social cognitive domains, including ToM, metacognition, and hostile attributional bias. The participants listened to ten brief stories and answered "yes" or "no" to questions pertaining to each story. Questions were presented in random order, and participants rated their confidence in their final answer. The verbal memory, schematic inference, and ToM scores were determined by the total number of correct responses, with possible scores ranging between 0 and 10; higher scores indicated better performance. Hostile attributional bias was assessed by counting the number of times participants incorrectly inferred negative emotions or thoughts in the characters in each scenario (range: 0–5), with higher scores reflecting stronger bias. Metacognition was evaluated based on participants' confidence in their correct and incorrect answers to the final question of each scenario, yielding scores between 0 and 10; higher scores indicated better metacognitive ability. The total score was calculated by summing all subscale scores, excluding the hostile attributional bias subscale.

**Statistical analysis**

Data analysis was performed using IBM SPSS Statistics version 29.0 for Windows (IBM Corp., Armonk, New York). Missing values were imputed using regression methods, with adjustments for residual estimates. Demographic and clinical variables between the two groups were compared using the $\chi^2$ test or the Mann-Whitney U test. Comparisons of SCSQ scores between the SAD and HC groups were conducted using the Mann-Whitney U test, with effect sizes calculated as r ($Z/\sqrt{N}$). Effect sizes were classified as small (< 0.1), medium (< 0.3), or large (≥ 0.5). Additionally, the relationships between SCSQ subscales were assessed using Spearman's correlation coefficients to examine the associations between variables. To further examine the influence of other clinical and demographic variables on SCSQ scores, a multivariate analysis of covariance (MANCOVA) was conducted. Variables indicating significant differences in the initial comparisons were included as covariates in the MANCOVA, which was conducted with 2000 bootstrap samples. Effect sizes were calculated as partial $\eta^2$ (SS effect / [SS effect + SS error]), with thresholds set for small (< 0.01), medium (< 0.06), and large (≥ 0.14) effects.

## Ⅲ. Results

Table 1 presents the demographic and clinical characteristics of the SAD and HC groups. No significant differences were observed between the groups concerning age, sex, or IQ. However, the SAD group achieved significantly higher scores on the LSAS (U = 582, $p < 0.001$, r = 0.51), AQ (U = 652, $p < 0.001$, r = 0.68), and BDI-II (U = 668, $p < 0.001$, r = 0.72).

Table 2 presents the SAD and HC groups' SCSQ scores. The total SCSQ (U = 219, $p = 0.012$, r = -0.34) and schematic inference (U = 200.5, $p = 0.003$, r = -0.40) scores were significantly lower in the SAD group. No significant differences were observed between the groups in the subscales for ToM, metacognition, and hostile attributional bias. Even after adjusting for AQ and BDI-II scores, the schematic inference scores remained significantly lower in the SAD group ($F(1, 54) = 7.80$, $p = 0.007$, $\eta^2p = 0.14$), while no significant differences were found in the total scores ($F(1, 54) = 0.95$, $p = 0.333$, $\eta^2p = 0.02$).

The results of the correlation analysis are shown in Table 3 and Table 4. In both the SAD and HC groups, schematic inference was not significantly correlated with any other subscales of the SCSQ. In the SAD group, ToM was negatively correlated with hostile attributional bias (r = -0.76, p < 0.001) and positively correlated with metacognition (r = 0.35, p = 0.072). ToM also showed a significant negative correlation with the AQ (r = -0.57, p = 0.002).

**Table 1 Demographic data and clinical measures**

| | SAD (n = 27) | HC (n = 27) | Statistics | *p*-value |
|---|---|---|---|---|
| Age, years | 26.19 ± 7.97 | 27.89 ± 11.03 | U = 370 | $p = 0.924$ |
| Male no. (%) | 15 (56) | 14 (52) | $\chi^2 = 0.07$ | $p = 0.785$ |
| IQ | 104.02 ± 8.99 | 105.77 ± 6.93 | U = 323.5 | $p = 0.478$ |
| Comorbidities, no. (%) | | | | |
| Major depressive disorder | 6 (22) | - | - | - |
| Others | 11 (41) | - | - | - |
| Medication, no. (%) | 20 (74) | - | - | - |
| LSAS | 76.33 ± 26.40 | 44.15 ± 25.74 | U = 582*** | $p < 0.001$ |
| AQ | 27.93 ± 5.72 | 19.15 ± 4.56 | U = 652*** | $p < 0.001$ |
| BDI-II | 21.37 ± 11.69 | 5.04 ± 4.75 | U = 668*** | $p < 0.001$ |

Notes: ***$p < 0.001$

Abbreviations: SAD, social anxiety disorder; HC, healthy controls; LSAS, Liebowitz Social Anxiety Scale; AQ, Autism-Spectrum Quotient; BDI-II, Beck Depression Inventory-II.

**Table 2 Social Cognition Screening Questionnaire scores for the SAD and HC groups**

| | SAD (n=27) | HC (n=27) | Statistics | *p*-value | Effect size |
|---|---|---|---|---|---|
| | Mean (SD) | Mean (SD) | | | |
| Verbal memory | 8.19 ± 1.04 | 8.44 ± 0.97 | U = 313.5 | $p = 0.350$ | -0.13 |
| Schematic inference | 8.30 ± 0.87 | 9.07 ± 0.96 | U = 200.5** | $p = 0.003$ | -0.40 |
| Theory of mind | 8.04 ± 1.13 | 8.26 ± 1.13 | U = 321.0 | $p = 0.436$ | -0.11 |
| Hostility bias | 1.22 ± 1.09 | 1.00 ± 0.88 | U = 398.5 | $p = 0.537$ | 0.08 |
| Metacognition | 9.51 ± 0.60 | 9.62 ± 0.49 | U = 330.5 | $p = 0.536$ | -0.08 |
| Total | 34.02 ± 2.24 | 35.40 ± 2.11 | U = 219.0* | $p = 0.012$ | -0.34 |

Notes: *$p < 0.05$, **$p < 0.01$, Statistics were based on the Mann-Whitney U-test

Abbreviations: SAD, social anxiety disorder; HC, healthy controls.

**Table 3 Correlation coefficients among Social Cognition Screening Questionnaire subscales and psychological measures in the SAD group**

| | Verbal memory | Schematic inference | Theory of mind | Hostility bias | Metacognition | Total score | LSAS | AQ | BDI-II |
|---|---|---|---|---|---|---|---|---|---|
| Verbal memory | 1.00 | | | | | | | | |
| Schematic inference | 0.07 | 1.00 | | | | | | | |
| Theory of mind | 0.28 | 0.02 | 1.00 | | | | | | |
| Hostility bias | 0.09 | 0.11 | -0.76** | 1.00 | | | | | |
| Metacognition | 0.11 | 0.20 | 0.35 | -0.01 | 1.00 | | | | |
| Total score | 0.62** | 0.34 | 0.73** | -0.31 | 0.55** | 1.00 | | | |
| LSAS | -0.03 | 0.10 | -0.39* | 0.44 | -0.08 | -0.22 | 1.00 | | |
| AQ | 0.04 | 0.19 | -0.57** | 0.47 | -0.25 | -0.25 | 0.49** | 1.00 | |
| BDI-II | 0.10 | 0.02 | -0.02 | 0.09 | -0.09 | -0.06 | 0.56** | 0.09 | 1.00 |

Notes: *P < 0.05, **P < 0.01, Spearman's correlation coefficients were calculated to examine the relationships between variables.

Abbreviations: SAD, social anxiety disorder; LSAS, Liebowitz Social Anxiety Scale; AQ, Autism-Spectrum Quotient; BDI-II, Beck Depression Inventory Second Edition scores.

## Ⅳ. Discussion

The findings indicated that the SAD group had significantly lower schematic inference scores than the HC group, suggesting that they experienced specific difficulties in certain aspects of cognitive function. Schematic inference refers to the ability to infer what is happening in a particular situation based on uncertain or ambiguous contextual information[22]. The significant difference in this domain between the SAD and HC groups emphasizes a selective impairment in schematic inference, which contrasts with the preservation of other

**Table 4 Correlation coefficients among Social Cognition Screening Questionnaire subscales and psychological measures in the HC group**

| | Verbal memory | Schematic inference | Theory of mind | Hostility bias | Metacognition | Total score | LSAS | AQ | BDI-II |
|---|---|---|---|---|---|---|---|---|---|
| Verbal memory | 1.00 | | | | | | | | |
| Schematic inference | 0.03 | 1.00 | | | | | | | |
| Theory of mind | 0.08 | 0.02 | 1.00 | | | | | | |
| Hostility bias | -0.24 | -0.10 | -0.83** | 1.00 | | | | | |
| Metacognition | -0.06 | -0.06 | 0.46* | -0.14 | 1.00 | | | | |
| Total score | 0.50** | 0.45* | 0.71** | -0.66** | 0.38* | 1.00 | | | |
| LSAS | -0.01 | 0.07 | -0.21 | 0.08 | -0.07 | -0.03 | 1.00 | | |
| AQ | -0.32 | 0.15 | -0.09 | 0.10 | 0.04 | -0.10 | 0.50** | 1.00 | |
| BDI-II | -0.16 | 0.18 | -0.03 | 0.06 | 0.22 | 0.11 | 0.66** | 0.59** | 1.00 |

Notes: *P < 0.05, **P < 0.01, Spearman's correlation coefficients were calculated to examine the relationships between variables.

Abbreviations: HC, healthy controls; LSAS, Liebowitz Social Anxiety Scale; AQ, Autism-Spectrum Quotient; BDI-II, Beck Depression Inventory Second Edition scores.

social cognitive abilities, such as ToM, metacognition, and hostile attributional bias. The significant difference in this domain between the SAD and HC groups suggests that SAD patients may struggle to accurately understand and interpret others' intentions and behaviors in social situations.

Typically, individuals draw on past experiences to make inferences when facing new situations. However, the significantly lower schematic inference scores in patients with SAD suggest that they may experience difficulties in this area. Cognitive models of SAD suggest that anxiety is perpetuated by interpreting ambiguous information as threatening, based on pre-existing negative beliefs[33,34]. While social information is often ambiguous, adults without SAD tend to interpret this information positively. However, patients with SAD lack this beneficial positive bias[35], which may play a fundamental role in maintaining their SAD[36]. Reduced schematic inference in patients with SAD suggests increased negative interpretation bias toward ambiguous situations, leading to misinterpretation of others' intentions and behaviors. This may further exacerbate anxiety and impair social functioning in these patients, resulting in a cycle of worsening symptoms.

The correlation analysis further highlights the unique cognitive profile of patients with SAD. Schematic inference showed no significant correlations with other SCSQ subscales, reinforcing its independence as a distinct cognitive domain. In contrast, ToM demonstrated meaningful correlations with other variables, consistent with previous findings[37-40]. ToM was negatively correlated with hostile attributional bias, suggesting that individuals with higher ToM abilities may be less prone to interpret ambiguous situations as hostile. Additionally, ToM showed a significant negative correlation with AQ, supporting previous evidence of the influence of autistic traits on social cognition[41-43]. These results suggest that while ToM and metacognition may interact to support social cognitive functioning, schematic inference operates independently, reflecting a distinct aspect of cognitive processing in patients with SAD.

Interestingly, while schematic inference was impaired in the SAD group, their preserved metacognition indicates an awareness of their difficulties in interpreting social contexts. This preserved metacognitive ability, while advantageous in some contexts, may paradoxically increase anxiety by heightening patients' awareness of their cognitive limitations. Extant studies indicated that heightened self-awareness of one's cognitive deficits, particularly in ambiguous situations, can exacerbate anxiety symptoms[44]. This awareness may lead to increased rumination or worry about social interactions, a core feature of SAD[45]. For example, recognizing that they struggle to infer others' intentions might cause heightened anxiety concerning potential negative evaluations[46]. Therefore, while metacognition is

preserved, the awareness of these cognitive deficits may perpetuate a cycle of anxiety and impaired social functioning.

While no significant differences were observed between the groups regarding ToM, metacognition, and hostile attributional bias, the Mann-Whitney U test indicated that the total score of the SCSQ was significantly lower in the SAD group. However, this effect did not persist when conducting the MANCOVA with AQ and BDI-II scores as covariates. Adjusting for autistic traits and depressive symptoms removed the effects of these factors, suggesting that some differences in the total SCSQ scores were attributable to depressive symptoms or autistic traits. These findings emphasize the importance of considering co-occurring traits, such as autistic traits and depressive symptoms, when evaluating the social cognitive profile of patients with SAD. They also provide important insights into understanding the social cognitive abilities of patients with SAD. Moreover, the findings align with several previous studies. Pepper et al. found that young adults with ASD and Early Psychosis (EP) exhibited more extensive social cognitive impairments than those with SAD[47]. This suggests that SAD might involve selective impairments in certain cognitive functions. Moreover, Pepper et al. emphasized depressive symptoms' important role in social functional impairment, noting that depressive symptoms were significant predictors of social cognitive impairment, even in the absence of objective social cognitive impairments[47].

Additionally, Alvi et al. emphasized the importance of appropriately adjusting for covariates when assessing SAD's direct impact on social cognitive function[48], which aligns with this study's results. Several previous studies reporting reduced social cognitive function in patients with SAD did not evaluate or consider autistic traits or depressive symptoms as covariates, leading to potentially inaccurate conclusions[8-10,12,13]. This study's findings reinforce the necessity of such adjustments to accurately interpret social cognitive abilities among patients with SAD. Further, SAD and major depressive disorder (MDD) have high comorbidity and share commonalities, such as high levels of negative emotions. A systematic review of the relationship between social anxiety and social cognition found a negative association between social anxiety and higher levels of empathy[47]. Conversely, depression has been associated with both lower and higher levels of cognitive empathy[48], highlighting the complex relationship between these disorders. Thus, depressive symptoms may negatively affect social cognitive function and if the reduction in social cognitive function in patients with SAD is primarily attributed to depressive symptoms, failure to consider this as a covariate may lead to erroneous conclusions. Next, the prevalence of SAD among individuals with ASD reportedly exceeds 50%[49-52]. ASD is defined by enduring challenges in social communication and interaction, coupled with restricted and repetitive behaviors, interests, or activities[1]. Individuals with autistic traits may face unique challenges in social interactions and communication, which can affect the assessment of social cognitive function. Therefore, autistic traits should also be considered as a factor influencing social cognitive function. Thus, the results of this study, which incorporated these factors as covariates, may more accurately reflect the impact of SAD on social cognitive function than previous studies.

The results of this study not only shed light on the nature of social cognitive function in patients with SAD but also inform the development of treatment strategies. Given that cognitive behavioral therapy already incorporates techniques to address negative interpretation biases, it may be beneficial to emphasize these techniques more explicitly for patients with SAD who face difficulties in schematic inference. Additionally, a comprehensive evaluation that considers the effects of depressive symptoms and autistic traits may help tailor interventions more precisely to individual patient profiles, potentially leading to better outcomes in social-cognitive functioning.

This study has several limitations. First, the relatively small sample size may constrain the extent to which the findings can be generalized. Second, approximately 74% of the participants with SAD were undergoing pharmacotherapy, which may have affected their cognitive function and introduced bias. Given that the

participants were undergoing pharmacotherapy, it is essential to consider the potential effects of medication on social cognitive performance. While there is currently no established pharmacological treatment for improving social cognition, this limitation emphasizes the need for future studies to examine the specific effects of pharmacotherapy on schematic inference and related domains. Third, ceiling effects were observed in some SCSQ subscales, where most healthy participants achieved perfect scores, potentially limiting the sensitivity to detect subtle differences in social cognitive function. Fourth, the cross-sectional nature of the study prevents the establishment of causality, emphasizing the need for longitudinal studies to better understand the interplay between SAD, depressive symptoms, and autistic traits.

## Ⅴ. Conclusion

This study revealed selective impairment in schematic inference among patients with SAD. The difficulties in interpreting ambiguous or uncertain information may extend beyond non-social contexts to impact social interactions, leading to misunderstandings and increased anxiety in social situations. Unlike other social cognitive functions, such as ToM, impaired schematic inference represents a unique challenge for patients with SAD in processing uncertain cues. Additionally, this study's findings emphasize the influence of autistic traits and depressive symptoms on cognitive functioning in SAD. It specifically highlights the importance of considering these co-occurring traits as covariates when evaluating the cognitive profile of patients with SAD, given that they may contribute to social functional impairments. Future interventions should consider addressing schematic inference difficulties while accounting for autistic traits and depressive symptoms to enhance treatment effectiveness and improve social cognitive outcomes.

## Contributors

Conceptualization: YI, SH, and YH; Data curation: YI and JH; Formal analysis: YI; Funding acquisition: ES and YH; Investigation: YI, RK, TT, TS, and IO; Methodology: YI, YN, and YH; Project administration: YI and YH; Supervision: ES and YH; Visualization: YI and YH; Writing - original draft: YI; Writing - review & editing: YI, SH, and YH.

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## Conflict of interest

ES is a member of the Editorial Board of the Chiba Medical Journal.

## Ethical approval

This study was approved by the Research Ethics Committee of the Graduate School of Medicine, Chiba University（Approval No. M10545）. Written informed consent was obtained from all participants. The study was conducted in accordance with the Declaration of Helsinki. The trial was registered as UMIN000024087.

## Data availability

This study's data is available from the corresponding author upon reasonable request.

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