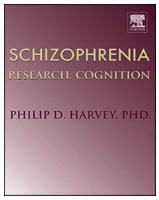
Schizophrenia Research: Cognition 41 (2025) 100361

Contents lists available at ScienceDirect

Schizophrenia Research: Cognition

journal homepage: www.elsevier.com/locate/scog



Research Paper

Domain-specific associations between social cognition and aggression in schizophrenia spectrum disorders

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ARTICLE INFO

Keywords:
Psychosis
Schizophrenia
Aggression
Social cognition
Emotion processing
Theory of Mind

ABSTRACT

Aggression in schizophrenia spectrum disorders (SSD) is rare but elevated relative to the general population. Existing studies have not identified reliable personal predictors of aggression in SSD. In line with social information processing models suggesting that difficulties interpreting social cues and others' intentions may lead to aggression, we evaluated whether social cognitive domains or global social cognition could be modifiable risk factors in SSD.

We examined aggression and social cognition in 59 participants with SSD and 43 healthy volunteers (HV). Self-reported aggression was measured via the Reactive-Proactive Aggression Questionnaire (RPAQ). Social cognition was assessed using five tasks measuring emotion processing, theory of mind, and social perception. Group differences were analyzed using Mann-Whitney-Wilcoxon tests. Multiple regressions examined effects of social cognition on aggression, controlling for demographic and clinical covariates. Supplemental mediation analyses tested whether impairments in emotion processing, theory of mind, or overall social cognition explained the relationship between SSD diagnosis and increased aggression.

Reported aggression was higher in the SSD group, and social cognitive abilities were impaired across domains ($p < .001$). Better emotion processing ($\beta = -0.35$, $p = .03$) and theory of mind ($\beta = -0.32$, $p = .03$) predicted lower aggression in SSD, even when accounting for demographic and neurocognitive variables. Exploratory models adjusting for overall psychiatric symptom severity showed that theory of mind remained significant, while emotion processing attenuated. However, social cognition did not mediate the relationship between diagnosis and aggression. Future studies should examine other social processing factors, such as attributional bias.

1. Introduction

Aggression refers to behavior directed toward another individual with the intent to harm them against their will. Aggression in schizophrenia is rare but has considerable consequences for both the individual and the community (Wehring and Carpenter, 2011); physical and psychological victimization usually affects caregivers (Estroff et al., 1998), and the mental and criminal justice systems hold a large burden to contain risk and offer adequate treatment (Hodgins et al., 2007; Malone et al., 2012). With approximately 5 % of severe violence and 10 % of homicides attributed to people with schizophrenia spectrum disorders (SSD) (Fazel and Grann, 2006; Taylor and Gunn, 1999), gaining a deeper understanding of social cognitive contributors to aggression risk within community samples of individuals with SSD holds potential clinical implications, ultimately aiming to enhance interventions and support for this population.

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https://doi.org/10.1016/j.scog.2025.100361
Received 22 November 2024; Received in revised form 5 April 2025; Accepted 6 April 2025

The increased propensity for aggression in individuals with SSD has historically been attributed to factors such as psychotic symptomatology, substance use, and executive functioning impairments (Elbogen and Johnson, 2009; Lysaker et al., 2004; Rasmussen et al., 1995) However, despite the well-established correlation between psychosis and aggression (Douglas et al., 2009), no modifiable mediators of aggression risk have yet been identified. A systematic review by Witt and colleagues (Witt et al., 2013) identified several risk factors for extreme violence in psychosis, including hostility and impaired impulse control, suggesting the potential role of social cognitive processes.

Research on the development of aggressive behavior has highlighted the critical role of social information processing, particularly how individuals perceive, encode, and interpret social cues such as others' intentions or emotional states (Crick and Dodge, 1994). These processes are fundamental to appropriate social interactions, as deficits in accurately processing social information may lead individuals to misinterpret social cues and respond with inappropriate defensive behaviors, including aggression (Dodge et al., 2015; Blackwood et al., 2001). Understanding these mechanisms may be particularly relevant for individuals with SSD, who commonly demonstrate impairments in social cognitive processes.

Impairment in social cognition has come to be recognized as a hallmark of SSD, referring to processes involved in understanding, predicting, and applying information in social and interpersonal contexts (Frith, 2007). This deficit in social cognition is a key determinant of social and functional outcomes in SSD and is commonly understood as being represented by multiple domains, including emotion processing, theory of mind, and social perception (Green et al., 2008; Pinkham et al., 2018) Emotion processing refers to one's ability to perceive and utilize emotions, including low-level identification abilities, such as facial recognition, as well as higher-level emotional understanding and management (Pinkham et al., 2004). Theory of Mind (ToM), also known as mentalizing, involves the capacity to attribute mental states to oneself and others. ToM includes the awareness that individuals possess mental states distinct from one's own and the ability to infer what one person believes another is thinking (Salva et al., 2013). Social perception is the process of interpreting social roles, rules, and context by extracting meaningful cues from social stimuli to understand others' intentions, emotions, and attributes for effective interpersonal interactions (Green et al., 2008).

Overall, the literature on this topic presents conflicting results about the relationship between aggression and social cognition, likely due to variations in sample characteristics and social cognitive measures. In studies including both healthy volunteers and participants with SSD, overall social cognitive impairment has been identified in participants with a history of violence, with larger and more widespread impairment seen in those with SSD (Vaskinn et al., 2023). Several reports connect social cognitive performance in SSD to aggression, but most assessments of social cognition in this context have been relatively narrow. Critically, the operationalization of social cognition in SSD has been highly varied in measure and quality (Browne et al., 2016). For instance, previous studies have shown that patients with schizophrenia with a history of violence perform worse than healthy volunteers on ToM and emotion recognition tasks but outperform non-violent patients with schizophrenia (Barlati et al., 2020). O'Reilly and colleagues (O'Reilly et al., 2015) found that social cognition has a direct effect on violence in SSD independent of neurocognition, violence proneness, and symptom severity, as assessed by a single task focused on emotion management. A meta-analysis of 11 studies showed that using social cognitive training was effective in reducing violence and aggression in people with severe mental illness, but it was unclear whether this was a broad effect of social cognition in general or if specific domains were implicated (Jones and Harvey, 2020).

In terms of ToM specifically, findings have been more inconsistent. Johansson et al. (Johansson et al., 2020) found that individuals with SSD who had committed psychotically driven acts of aggression (PDA) exhibited lower ToM scores than non-aggressive and control groups, even after controlling for general intellectual ability and antipsychotic medication dosages. These results suggest that ToM may be specifically implicated in psychotically driven aggression. However, other studies have found no differences in ToM task performance between violent and non-violent individuals with SSD (Majorek et al., 2009; Mitchell et al., 2014), while some have reported that violent patients with SSD outperformed their non-violent counterparts on ToM tasks (Abu-Akel and Abushua'leh, 2004; Bo et al., 2011). As noted by Barlati et al. (Barlati et al., 2020), inconsistencies in ToM performance may reflect broader variations in social cognitive functioning and the complexity of its relationship with aggression.

This study seeks to contribute to the ongoing discourse by using a more comprehensive set of tasks to examine the primary social cognitive domains of emotion processing, ToM, and social perception. We aimed to understand whether the relationships between social cognition and self-reported aggression is specific to a domain under the umbrella of social cognition, or more generalized to overall social cognitive ability. By focusing our investigation on outpatients, we address a gap in the current literature that predominantly studies inpatient or forensic populations. Our primary objective is to investigate whether impairments in social cognitive domains are linked to increased self-reported aggression among individuals with SSD, exploring their potential as modifiable risk factors. Based on prior works, we expected to find increased self-reported aggression and impaired social cognition in SSD relative to healthy volunteers (HV). We also expected that among SDD, those with greater impairments in social cognition may report greater aggression. Given that impairment in social cognition may lead to hostile reactions in social situations (Dodge et al., 2015), we hypothesized that social cognitive impairment in one or more relevant domains may mediate the relationship between diagnosis and self-reported aggression.

2. Methods

2.1. Study setting and participants

The data presented here were collected at the Zucker Hillside Hospital (ZHH) as a single-site extension of the Social Processes Initiative in the Neurobiology of the Schizophrenia(s) (SPINS) study. The sample ($N = 102$) is a subset of the larger multi-site study previously described elsewhere (Tang et al., 2022; Oliver et al., 2021). In brief, participants included individuals with SSD and HV participants. Those in the SSD ($n = 59$) group were stable outpatients and met the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (SCID-IV) (First and Gibbon, 2004) for schizophrenia, schizoaffective disorder, schizophreniform disorder, or unspecified psychotic disorder, with no change in antipsychotic medication or decrement in functioning during the month before study enrollment. Participants with a history of significant head trauma, recent substance use disorder, intellectual disability, or other conditions that may have affected their ability to participate were excluded. Participants with eligible diagnoses were recruited from outpatients who were previous participants in other studies and from active outpatients who were recommended as being appropriate for the study inclusion criteria by their clinical treatment team. The HV group ($n = 43$) had no first-degree family history of psychotic disorders and no lifetime diagnosis of an Axis I psychiatric disorder, except for those with past adjustment, phobic, or major depressive disorder who were, at the time of consent, in full remission for at least two years and not actively on psychotropic medications. All procedures were approved by the Institutional Review Board at the Feinstein Institutes for Medical Research. Before the initiation of study procedures, a quiz was administered to confirm decisional capacity. All participants provided written informed consent.

2.2. Measures and assessments

Psychopathology and social cognition were evaluated based on the SPINS protocol, as previously reported (Tang et al., 2022; Oliver et al., 2021). Psychopathology was assessed using the Brief Psychiatric Rating Scale (BPRS) and Scale for the Assessment of Negative Symptoms (SANS), following the SPINS protocol. Neurocognition was assessed using the MATRICS Consensus Cognitive Battery (MCCB), from which a neuropsychological composite score was derived. (Tang et al., 2022; Oliver et al., 2021). Illness history variables, including age of onset, duration, and number of hospitalizations, were also recorded.

2.3. Assessment of social cognition

Social cognition was assessed with five tasks, as described by Tang and colleagues (Tang et al., 2022). The Penn Emotion Recognition 40 (ER-40) captures the speed and accuracy of emotion identification from forty diverse photographs (Moore et al., 2013). An efficiency score for this task was calculated by adding and re-normalizing z-scores for accuracy and speed. The ER-40 shows good test-retest reliability (Pearson's $r = 0.710$ for SSD, $r = 0.679$ for controls) (Pinkham et al., 2018). The Empathic Accuracy (EA) task presents nine videos of actors describing both positive and negatively valenced autobiographical events and asks participant viewers to continuously rate how positive or negative the actor is feeling. Participant responses are correlated with true emotion ratings from the subjects in the videos, with the task demonstrating strong test-retest reliability and minimal practice effects (Kern et al., 2013). The Reading the Mind in the Eyes Task (RMET) presents the participant with a cropped photo of the eye region and asks for identification of the corresponding mental state (Baron-Cohen et al., 2001). The RMET demonstrates good test-retest reliability (Pearson's $r = 0.806$ for SSD, $r = 0.716$ for HV) and acceptable internal consistency (Cronbach's $\alpha = 0.750$ for SSD, $\alpha = 0.640$ for HV) (Pinkham et al., 2018). Relationships Across Domains (RAD) presents a series of vignettes and asks the participant to characterize the dyadic relationship described (Sergi et al., 2009). The TASIT demonstrates moderate test-retest reliability (Pearson's $r = 0.636$ for SSD, $r = 0.534$ for HV) and good internal consistency (Cronbach's $\alpha = 0.807$ for SSD, $\alpha = 0.825$ for HV) (Pinkham et al., 2018). The Awareness of Social Inference Test-Revised (TASIT) includes three parts, each based on brief videos showing an interaction between actors (McDonald et al., 2003). Part 1 asks the participant to identify the displayed emotions. Part 2 and Part 3 test social inference from situations, differentiating sincerity from sarcasm and sarcasm from lies, respectively. As each TASIT part tests a different social cognitive function, they were considered separately in the analyses.

We categorized social cognition into three domains, as per the model proposed by Pinkham, Harvey, & Penn (Pinkham et al., 2018). The emotion processing domain comprised ER40, EA, and TASIT Part 1. The theory of mind domain included RMET and TASIT Parts 2 and 3. The social perception domain was based on scores in the RAD. Domain scores were derived by summing and re-standardizing the *z*-scores. An overall social cognition score included all tasks, with equal weights assigned to each task.

2.4. Assessment of aggression

Supplemental aggression surveys were added at the ZHH site to evaluate the relationship between self-reported aggression and social cognition. Self-reported aggression was assessed through the Reactive-Proactive Aggression Questionnaire (RPAQ). The RPAQ is a 23-item self-report measure of verbal and physical aggression with two subscales for the measurement of proactive and reactive aggression. Scores across subscales were summed for a total aggression score. The RPAQ has demonstrated good criterion, convergent, discriminant, and construct validity with demonstrated alphas above 0.80 for all sub- and total scales (Raine et al., 2006). The Proactive Aggression scale consists of 12 items (e.g., "had fights with others to show who was on top"), and the Reactive Aggression scale includes 11 items (e.g., "got angry when others threatened them"). Each item is rated as 0 (never), 1 (sometimes), or 2 (often). Scores on the Reactive Aggression scale were $M = 7.02$ ($SD = 5.23$) for the SSD group and $M = 3.56$ ($SD = 2.94$) for HV. On the Proactive Aggression scale, scores were $M = 2.02$ ($SD = 3.00$) for the SSD group and $M = 0.21$ (SD = 0.79) for healthy controls. Because proactive aggression was exceedingly rare in both groups, self-reported aggression was represented solely as the RPAQ total score.

2.5. Statistical analysis

All analyses were conducted using R version 3.5.2 (R Core Team, 2023). Mann-Whitney-Wilcoxon tests were conducted to assess differences in RPAQ and social cognition scores between the two groups, including emotion processing (ER40, EA, TASIT Part 1), theory of mind (RMET, TASIT Parts 2 and 3), and social perception (RAD). This non-parametric test was chosen due to the non-normal distribution of the data.

Within each diagnostic group, the association between social cognitive ability and self-reported aggression was assessed through linear regression, allowing us to control for potential confounding variables and isolate the unique contribution of each social cognition domain to self-reported aggression. We first evaluated simple linear regressions predicting the total RPAQ score with each of the social cognition domain scores, as well as the overall social cognition score. For significant findings, we then evaluated the effect of potentially confounding demographic variables one at a time including age, sex, education, ethnicity, and race as well as clinical variables, including BPRS Total, SANS Total, and the MATRICS MCCB Composite Score.

To further examine the role of symptom dimensions in predicting aggression, we conducted exploratory post-hoc analyses using the Brief Psychiatric Rating Scale (BPRS) three-factor structure, which includes Positive Symptoms, Activation, and Hostility factors. These analyses were not pre-registered and were intended to better understand potential symptom-level predictors of aggression, above and beyond social cognitive factors.

Supplemental mediation analyses were performed using the lavaan package in R (Rosseel, 2012), including multiple regressions with diagnostic group as a predictor of total RPAQ scores and each of emotion processing, ToM, social perception, and overall social cognition as mediators. Mediation models were tested using full information maximum likelihood estimation, and indirect effects were determined using nonparametric bootstrapping with 5000 resamples, where the effect is considered significant ($p < .05$) if the bias-corrected and accelerated, 95 % confidence interval (CI) does not include 0 (Preacher and Hayes, 2008).

3. Results

Participant demographic information is presented in Table 1. The sample consisted of 59 participants in the SSD group and 43 in the HV group. Demographic analysis revealed no significant differences between groups in age, sex, or racial composition. However, the SSD group had a significantly higher proportion of Hispanic participants compared to the HV group ($p = .007$) and education levels were significantly higher in the HV group compared to the SSD group ($p < .001$).

Table 1 also presents clinical characteristics of the SSD group, including symptom severity, neurocognition score, and illness course. On average, participants exhibited moderate psychiatric symptoms, as indicated by BPRS and SANS scores. Mean neurocognitive performance was below the normative average, consistent with documented impairments in SSD (Rasmussen et al., 1995). Our sample demonstrated substantial variability in illness course, with a mean onset in early adulthood, an average illness duration exceeding a decade, and a large

Table 1
Demographic characteristics of participants with Schizophrenia Spectrum Disorders (SSD) and Healthy Volunteers (HV).

	SSD	HV	*p-value*
$N = 102$	$n = 59$	$n = 43$	
Age (*SD*)	33.5 (11.9)	34.8 (10.1)	0.54
Sex (%)			0.35
Female	26 (44.1 %)	23 (53.5 %)	
Male	33 (55.9 %)	20 (46.5 %)	
Race (%)			
Asian	12 (20.3 %)	8 (18.6 %)	
Black/African American	21 (35.6 %)	17 (39.53 %)	
Other	2 (3.4 %)	1 (2.33 %)	
White/Caucasian	23 (39.0 %)	17 (39.53 %)	
Not Reported	1 (1.7 %)	0	
Ethnicity			0.007
Hispanic	12 (20.3 %)	1 (2.3 %)	
Not Hispanic	47 (79.7 %)	45 (97.7 %)	
Education (*SD*)	14.32 (2.4)	16.14 (2.2)	<0.001
Diagnosis			
Schizoaffective	5 (8.5 %)		
Schizophrenia	46 (78 %)		
Schizophreniform	4 (6.8 %)		
Unspecified PD	4 (6.7 %)		
Clinical Information (SSD)	Mean (SD)	Range	
BPRS Total	29.36 (7.1)	19–54	
SANS Global Total	6.86 (3.26)	0–14	
MATRICS Composite	34.7 (15.17)	−8–69	
Age of Onset (years)	21.68 (5.57)	12–40	
Illness Duration (years)	11.78 (12.1)	0–37	
Number of Hospitalizations	2.61 (2.29)	0–10	

Note: PD = Psychotic Disorder. Education is reported in years. Group differences were tested using Mann-Whitney-Wilcoxon tests for continuous variables and chi-square tests for categorical variables. In the clinical sample, symptom severity was assessed using the Brief Psychiatric Rating Scale (BPRS) and the Scale for the Assessment of Negative Symptoms (SANS). Neurocognition was measured by the MATRICS MCCB.

range in number of hospitalizations.

Domain scores on self-reported aggression and social cognitive measures are presented in Table 2. We confirmed expected findings that the SSD group showed significantly higher total aggression scores ($p < .001$). Social cognition assessments also showed significantly poorer performance in the SSD group across all domains ($p < .001$). Fig. 1 presents density plots comparing aggression and social cognition domains between groups.

Within each group, the association between social cognitive ability and total self-reported aggression was assessed through linear regressions. We found that better performance in both emotion processing ($\beta = -0.35$, $p = .03$) and theory of mind ($\beta = -0.32$, $p = .03$) predicted decreased aggression in SSD. The detailed results from the regression models, including standardized coefficients, standard errors, *t*-values, and *p*-values for each predictor, are presented in Table 3. Fig. 2 shows the relationship between ToM and emotion processing with self-reported aggression, with linear regression lines fitted for each diagnostic group.

Table 2
Group differences in self-reported aggression and social cognitive performance between individuals with Schizophrenia Spectrum Disorders (SSD) and Healthy Volunteers (HV).

	SSD		HV			
	Mean	*SD*	Mean	*SD*	*p*	*d*
RPAQ: Total Aggression	9.09	8.91	3.86	3.86	<0.001	0.75
Social Cognition: Overall Performance	−0.51	1.02	0.23	0.68	<0.001	−0.85
Social Cognition: Theory of Mind	−0.51	1.04	0.26	0.72	<0.001	−0.86
Social Cognition: Emotion Processing	−0.44	1.03	0.14	0.71	<0.001	−0.65
Social Cognition: Social Perception	50.26	11.49	57.83	7.90	<0.001	−0.76

Note: RPAQ = Reactive-Proactive Aggression Questionnaire. Social cognition domain scores are presented as z-scores, except for Social Perception which uses raw scores. Higher scores indicate better performance for social cognition measures. *p*-values are based on Mann-Whitney-Wilcoxon tests. Effect sizes are reported as Cohen's *d,* all show medium-to-large effects.

To account for potential confounding factors, we ran separate models within the schizophrenia group including individual demographic covariates (age, sex, education, ethnicity, and race), as well as separate models for each clinical variable (BPRS, SANS, and MATRICS). None of the demographic covariates were significantly associated with self-reported aggression. Consistent with prior evidence that social cognition independently predicts violence even when controlling for neurocognition (O'Reilly et al., 2015), neurocognition, as measured by the MATRICS composite, did not significantly predict self-reported aggression in any model, and its inclusion did not reduce the strength of associations between social cognition and aggression. ToM remained a significant predictor of aggression in all covariate models, except when adjusting for total BPRS symptom severity, where ToM was marginally significant ($p = .058$). Emotion processing remained significant in all models except total BPRS ($p = .132$), where it was no longer significant. Social perception was not significantly associated with aggression in any model. Results of covariate models for overall social cognition, theory of mind, and emotion processing are presented in Table 4.

To further examine symptom-level contributions to self-reported aggression, we conducted exploratory post-hoc regression models using the BPRS three-factor structure. In the model including ToM and the Hostility factor ($R^2 = 0.16$), ToM significantly predicted self-reported aggression (b = −0.98, 95 % CI [−1.93, −0.02], $\beta = -0.28$, SE = 0.48, $t = -2.05$, $p = .046$), while Hostility showed a marginal effect (b = 0.75, 95 % CI [−0.10, 1.60], $\beta = 0.24$, SE = 0.42, $t = 1.77$, $p = .083$). In contrast, models including either the Positive Symptoms ($p = .069$) or Activation ($p = .093$) factors did not significantly predict self-reported aggression when controlling for ToM. ToM remained a significant predictor in these models.

In emotion processing models, the BPRS Hostility subscale significantly predicted self-reported aggression (b = 1.54, 95 % CI [0.41, 2.66], $\beta = 0.42$, SE = 0.55, $t = 2.78$, $p = .009$, $R^2 = 0.28$), whereas emotion processing was no longer significant (b = −1.09, 95 % CI [−2.57, 0.40], $\beta = -0.22$, SE = 0.73, $t = -1.49$, $p = .146$). Positive Symptoms ($p = .088$) and Activation ($p = .26$) were also not significant predictors in models including emotion processing.

Building on these findings, we conducted structural equation models to test whether emotion processing or ToM mediated the relationship between SSD diagnosis and aggression. Neither variable significantly mediated this relationship (see Supplementary Fig. 3), although both were significantly impaired in the SSD group relative to healthy volunteers.

4. Discussion

In this study, we investigated the relationship between social cognition and self-reported aggression in individuals with SSD compared to HV. We hypothesized that: 1) there would be meaningful group differences between SSD and HV in both social cognition and self-reported aggression; 2) greater impairment in social cognition would be associated with higher self-reported aggression among individuals with SSD; and 3) impairments in social cognition would drive aggression, mediating the relationship between diagnosis and self-reported aggression.

These hypotheses were partially supported by the findings of this study. As expected, individuals with SSD exhibited significantly higher levels of self-reported aggression across all measured domains—reactive, proactive, and total aggression—compared to HVs. This is consistent with previous research suggesting an increased risk for aggression in SSD. The study also identified clear and expected deficits

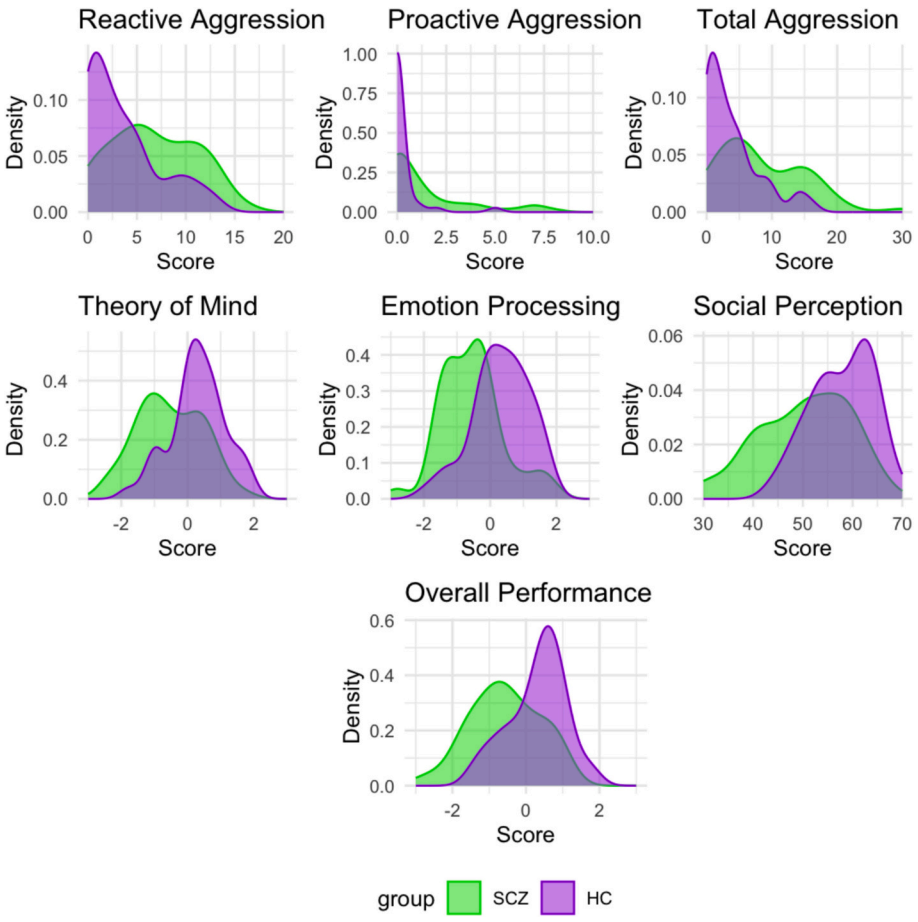

Fig. 1. Density plots aggression and social cognitive performance between groups
Note: Density plots showing the distribution of scores for total aggression (RPAQ) and social cognitive domains (emotion processing, theory of mind, and social perception) in individuals with schizophrenia spectrum disorders (SSD; green) and healthy volunteers (HV; purple). Higher scores indicate better performance for social cognitive measures and greater aggression for RPAQ.

Table 3
Regression table predicting total self-reported aggression.

Group	Predictor	*b*	95 % CI	β	SE	*t*	*p*	R^2
SSD	Overall Social Cognition	−1.07	[−2.17, 0.03]	−0.31	0.56	−1.93	0.062†	0.10
	Theory of Mind	−1.09	[−2.04, −0.13]	−0.32	0.48	−2.25	0.029*	0.10
	Emotion Processing	−1.70	[−3.21, −0.19]	−0.35	0.76	−2.24	0.031*	0.12
	Social Perception	−0.17	[−0.42, 0.07]	−0.20	0.12	−1.43	0.160	0.04
HV	Overall Social Cognition	0.34	[−0.78, 1.47]	0.10	0.57	0.61	0.548	0.01
	Theory of Mind	0.44	[−0.66, 1.54]	0.13	0.55	0.80	0.430	0.02
	Emotion Processing	0.25	[−0.83, 1.32]	0.08	0.54	0.46	0.650	0.01
	Social Perception	0.06	[−0.15, 0.28]	0.09	0.11	0.58	0.576	0.01

Note: Standardized regression coefficients (β) are shown for separate models predicting total RPAQ scores from each social cognitive domain. Models were run separately for SSD and HV groups. RPAQ = Reactive-Proactive Aggression Questionnaire; SSD = Schizophrenia Spectrum Disorders; HV = Healthy Volunteers. SE = Standard Error.
† $p < .10$.
* $p < .05$.

in individuals with SSD across multiple domains of social cognition, including emotion processing, theory of mind (ToM), and social perception. This is consistent with existing literature that identifies social cognitive impairments as a hallmark of SSD (Hajdúk et al., 2018).

We also confirmed our hypothesis that greater impairments in social cognition were related to a higher degree of self-reported aggression among people with SSD. This effect extended across social cognitive domains. Greater impairment in emotion processing and ToM were significantly associated with increased self-reported aggression in the SSD group, suggesting that difficulties in recognizing emotions and inferring mental states were related to self-reported aggressive behavior in individuals with SSD. These findings are consistent with The Integrated Emotional System (IES) model of psychopathy defined by Blair (Blair, 2005) which suggests that facial expressions of sadness and fear are distress cues that inhibit aggressive behavior. In SSD, significant deficits in emotion processing are well established, especially for negative emotions such as fear and anger (Kohler et al., 2010; Turetsky et al., 2007). These deficits are further pronounced in people with SSD who have had a history of criminal behavior (Weiss et al., 2006) and in people with SSD and comorbid psychopathy (Fullam and Dolan, 2006).

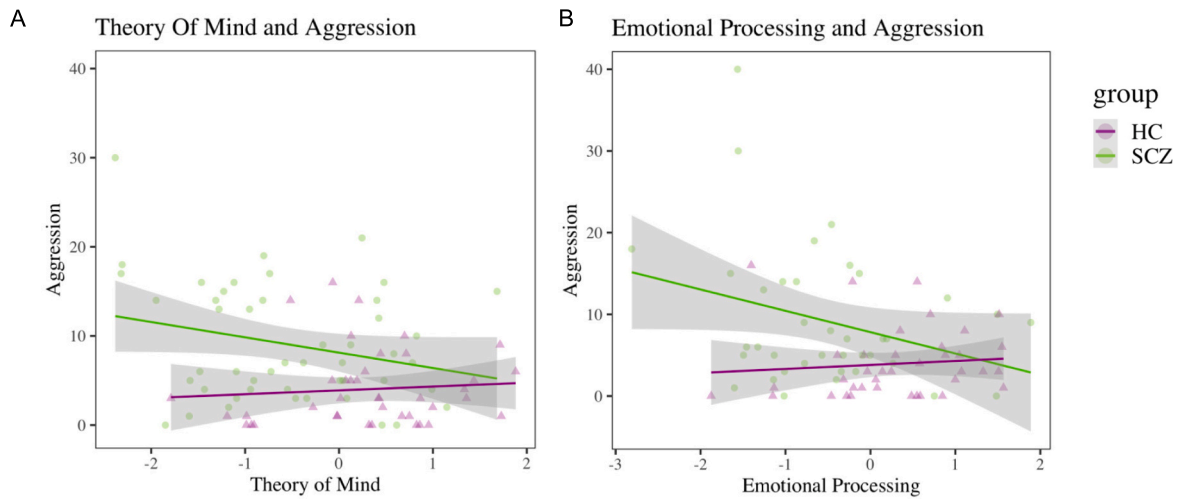

Fig. 2. Linear regression lines illustrating the relationship between ToM and Emotion Processing with Aggression for each diagnostic group.
Note: Scatterplots showing the relationship between total aggression (RPAQ scores) and (A) Theory of Mind and (B) Emotion Processing performance in individuals with schizophrenia spectrum disorders (SSD; green) and healthy volunteers (HV; purple). Regression lines with 95 % confident intervals indicate significant negative associations between both social cognitive domains and aggression in the SSD group ($p < .05$) but not in HV. Higher scores indicate better performance for social cognitive measures.

Social cognitive impairments were robustly associated with self-reported aggression even after adjusting for demographic and clinical covariates. The attenuation of effects when adjusting for overall symptom severity may reflect conceptual overlap between symptom and aggression, rather than diminishing the mechanistic role of social cognition. Exploratory analyses using the BPRS three-factor structure further highlighted the specific role of hostility-related symptoms, including items related to irritability, suspiciousness, and uncooperativeness. This underscores the importance of these more distal characteristics as potent predictors of engagement in aggression, more so than other core features of SSD (e.g., hallucinations, delusions). Their predictive value may reflect shared behavioral features rather than an independent contribution. Notably, ToM remained a significant predictor even after adjusting for Hostility, reinforcing its role. These findings suggest that deficits in social cognition, particularly in mental state attribution, contribute to aggression in SSD beyond the influence of psychiatric symptom burden (O'Reilly et al., 2015).

Given that neither emotion processing nor ToM mediated the relationship between SSD diagnosis and increased aggression, our findings indicate that although social cognitive deficits correlate with aggression in individuals with SSD, other untested factors may explain the increased aggression observed in this population. It remains possible that our measures did not fully capture the most relevant aspects of social cognition in relation to aggression. For instance, the RAD, used to assess social perception, may lack sensitivity to features such as attribution bias (Pinkham et al., 2018), which has been previously associated with paranoia and unprovoked aggression (Iozzino et al., 2021).

Aggression in this population is often multifaceted and can arise from various factors, including feelings of persecution or defensive reactions. A recent qualitative study by Lambe and colleagues (Lambe et al., 2024) found that violence in forensic patients with psychosis can be triggered by sensitivities to perceived threats from others, as well as attempts to regulate distressing internal states related to psychotic symptoms, such as defensive aggression due to paranoia or violence in response to command hallucinations. As such, further quantitative research is needed to build and fit the theoretical model of aggression in SSD. As theoretical models of aggression emphasize multifactorial pathways that integrate cognitive, neurobiological, and environmental factors, future research should explore a broader range of potential contributors including biased social attributions and interpersonal threat sensitivity to further elucidate the mechanisms linking social cognition and aggression in SSD.

Limitations.

While our study offers valuable insights into the relationship between social cognition and aggression in individuals with SSD, several limitations should be acknowledged. Specific methodological shortcomings should be noted, in that the reliance on self-report measures for aggression may limit the objectivity of the findings. Self-reports of aggression may be influenced by social desirability bias, which may lead both individuals with SSD and healthy controls to underreport their aggressiveness (Vigil-Colet et al., 2012). Furthermore, although the inclusion of data such as clinical or forensic records would have improved the validity of self-reported aggression, such data were not available in our study and are associated with their own limitations (e.g., potential omission of less severe but more prevalent forms of aggression). Future research should aim to incorporate multiple sources of aggression assessment, including clinician-rated measures and historical records, to strengthen the validity of findings in this area.

Other limitations of research on this topic include variations in sample characteristics and insufficient consideration of the heterogeneity within the schizophrenia spectrum (Ahmed et al., 2018). Variability in both social cognitive and aggression scores within the SSD group may have reduced the power of group-based mediation analyses. These factors may contribute to inconsistencies in findings across related studies and highlight the need for more standardized approaches in future research. Longitudinal studies with larger, more diverse samples are needed to confirm current findings, explore causality, and address these methodological limitations.

The lack of significant mediation results suggests that other variables, such as substance use or broader cognitive biases (O'Reilly et al., 2015) may play a fundamental role in the aggression risk observed in individuals with SSD. Future studies should include dedicated assessments to measure attributional bias (Dodge, 2006) and incorporate biomarkers to further characterize these and other modifiable contributors to aggression in SSD. Moreover, the potential utility of cognitive remediation interventions in reducing the risk of violence in SSD (Darmedru et al., 2017) suggests that targeting both social and cognitive deficits may be important for developing comprehensive interventions to address aggression in this population. Innovative methods like digital phenotyping could provide real-time data on aggression-related behaviors (Barnett et al., 2018; Buck et al., 2019). By employing a whole-person, multi-modal approach, researchers can work toward a more

Table 4
Covariation models.

Covariation model	Variable	*b*	β	SE	*t*	*p*
RPAQ ~ Overall SC + Age	Overall SC	−1.1	−0.34	0.37	−2.98	0.004**
	Age	−0.07	−0.13	0.06	−1.12	0.268
RPAQ ~ Overall SC + Sex	Overall SC	−1.03	−0.31	0.37	−2.81	0.006*
	Sex	1.81	0.15	1.36	1.33	0.187
RPAQ ~ Overall SC + Education	Overall SC	−1.01	−0.31	0.42	−2.41	0.019*
	Education	−0.08	−0.03	0.34	−0.24	0.812
RPAQ ~ Overall SC + Ethnicity	Overall SC	−1.08	−0.31	0.37	−2.9	0.005**
	Ethnicity	−0.92	−0.06	2.09	−0.44	0.659
RPAQ ~ Overall SC + Race	Overall SC	−0.8	−0.29	0.39	−2.06	0.044*
	Race	3.25	0.17	1.9	1.71	0.092
RPAQ ~ Overall SC + BPRS	Overall SC	−0.8	−0.24	0.56	−1.42	0.165
	BPRS	0.26	0.32	0.15	1.68	0.102
RPAQ ~ Overall SC + SANS	Overall SC	−1.04	−0.3	0.59	−1.77	0.086
	SANS	0.07	0.04	0.34	0.21	0.838
RPAQ ~ Overall SC + MATRICS	Overall SC	−1.04	−0.29	0.5	−2.09	0.041*
	MATRICS	0.01	0.03	0.06	0.18	0.859
RPAQ ~ ToM + Age	ToM	−1.18	−0.35	0.34	−3.44	<0.001***
	Age	−0.07	−0.13	0.05	−1.27	0.206
RPAQ ~ ToM + Sex	ToM	−1.14	−0.33	0.34	−3.29	0.001**
	Sex	1.61	0.13	1.24	1.3	0.197
RPAQ ~ ToM + Education	ToM	−0.93	−0.27	0.4	−2.34	0.022*
	Education	−0.35	−0.14	0.29	−1.22	0.227
RPAQ ~ ToM + Ethnicity	ToM	−1.16	−0.33	0.35	−3.29	0.001**
	Ethnicity	0.03	−0.06	1.91	0.02	0.986
RPAQ ~ ToM + Race	ToM	−1.15	−0.34	0.35	−3.27	0.002**
	Race	0.42	0.03	1.27	0.33	0.742
RPAQ ~ ToM + BPRS	ToM	−0.96	−0.28	0.49	−1.95	0.058†
	BPRS	0.16	0.17	0.13	1.22	0.231
RPAQ ~ ToM + SANS	ToM	−1.03	−0.3	0.5	−2.08	0.043*
	SANS	0.16	0.08	0.29	0.56	0.576
RPAQ ~ ToM + MATRICS	ToM	−0.98	−0.29	0.45	−2.18	0.032*
	MATRICS	−0.01	−0.288	0.51	−0.376	0.708
RPAQ ~ EmoP + Age	EmoP	−1.62	−0.38	0.47	−3.46	<0.001***
	Age	−0.12	−0.19	0.07	−1.7	0.094
RPAQ ~ EmoP + Sex	EmoP	−1.38	−0.32	0.47	−2.97	0.004**
	Sex	2.37	0.17	1.55	1.53	0.131
RPAQ ~ EmoP + Edu	EmoP	−1.27	−0.3	0.51	−2.48	0.016*
	Education	−0.36	−0.12	0.38	−0.96	0.34
RPAQ ~ EmoP + Ethnicity	EmoP	−1.5	−0.35	0.47	−3.19	0.002**
	Ethnicity	−1.32	−0.06	2.42	−0.55	0.585
RPAQ ~ EmoP + Race	EmoP	−1.4	−0.33	0.47	−2.97	0.004**
	Race	1.69	0.12	1.59	1.07	0.29
RPAQ ~ EmoP + BPRS	EmoP	−1.19	−0.24	0.77	−1.54	0.132
	BPRS	0.37	0.32	0.18	2.04	0.049†
RPAQ ~ EmoP + SANS	EmoP	−1.77	−0.36	0.8	−2.22	0.033*
	SANS	−0.13	−0.05	0.4	−0.33	0.748
RPAQ ~ EmoP + MATRICS	EmoP	−1.44	−0.34	0.6	−2.42	0.018*
	MATRICS	0.01	−0.05	0.06	−0.02	0.988

Note: Associations between social cognition (Overall SC, Theory of Mind [ToM], and Emotion Processing [EmoP]) and self-reported aggression, controlling for demographic and clinical covariates. Each model included one covariate at a time. Unstandardized (*b*) and standardized (β) coefficients, standard errors (SE), *t*-values, and *p*-values are reported.

* $p < .05$.
** $p < .01$.
*** $p < .001$.

nuanced understanding of the factors that contribute to aggression in this population, ultimately informing the development of targeted, effective interventions.

5. Conclusions

Our study contributes valuable insights into the relationship between social cognitive domains and self-reported aggression in individuals with SSD, such that these individuals with SSD report higher levels of aggression and poorer social cognition than HVs, but we did not observe evidence that poorer social cognition statistically accounts for the link between SSD and aggression. This study aimed to address the limitations of previous research by employing a comprehensive set of tasks to examine the relationship between social cognition and self-reported aggression across multiple domains in outpatients with SSD and HV. This line of questioning holds the potential to inform prevention and intervention strategies, ultimately aiming to enhance support and understanding for individuals with SSD and reduce the burden on mental health and criminal justice systems. The results highlight the relationship between domain-specific social cognitive deficits and self-reported aggression in SSD, emphasizing the importance of targeting emotion processing and ToM skills in interventions aimed at reducing aggression in individuals with SSD, as improving these social cognitive abilities may facilitate more accurate social learning and the acquisition of prosocial behaviors. Future research should focus on addressing existing limitations and expanding our understanding of the biopsychosocial factors contributing to aggression in SSD, ultimately leading to the development of more targeted and effective interventions.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scog.2025.100361.

CRediT authorship contribution statement

Sarah A. Berretta: Writing – original draft, Visualization, Formal analysis. **Lindsay D. Oliver:** Writing – review & editing, Formal analysis. **Courtland S. Hyatt:** Writing – review & editing, Conceptualization. **Ricardo E. Carrión:** Methodology, Data curation, Conceptualization. **Katrin Hänsel:** Formal analysis. **Aristotle Voineskos:** Conceptualization. **Robert W. Buchanan:** Writing – review & editing, Conceptualization. **Anil K. Malhotra:** Funding acquisition, Conceptualization. **Sunny X. Tang:** Writing – review & editing, Supervision, Formal analysis, Conceptualization.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author(s) used Perplexity AI to receive feedback and suggestions on the manuscript draft. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

Declaration of competing interest

SXT owns equity and serves on the board and as a consultant for North Shore Therapeutics, received research funding and serves as a consultant for Winterlight Labs, is on the advisory board and owns equity for Psyrin, and serves as a consultant for Catholic Charities Neighborhood Services and LB Pharmaceuticals. RWB is a DSMB member for Merck, Newron, and Roche and has served on advisory boards for Acadia, Karuna, Merck and Neurocrine. AKM is a consultant for Acadia Pharmaceuticals, Genomind Inc., Informed DNA, and Janssen Pharmaceuticals. The other authors have no disclosures to report.

Acknowledgments

We thank the participants for their contributions. In addition, we thank Leily Behbehani and Majnu John for their support. Funding was received from NIMH: R01MH102313, R01MH102318, R01MH102324, K23 MH130750 (SXT).

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