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How psychosis shapes the impact of childhood trauma on social cognition in bipolar disorder: the role of Theory of Mind

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How psychosis shapes the impact of childhood trauma on social cognition in bipolar disorder: the role of Theory of Mind

Running title: Psychosis, Trauma, and ToM in Bipolar Disorder.

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Abstract

Objective: Childhood trauma and psychotic symptoms are prevalent in bipolar disorder (BD) and independently linked to social cognition impairments, particularly Theory of Mind (ToM). However, it remains unclear whether trauma and psychosis exert cumulative effects on ToM performance in BD.

Methods: We conducted a cross-sectional study involving 54 euthymic individuals with BD reporting at least moderate childhood trauma (Childhood Trauma Questionnaire; CTQ). Participants completed the Reading the Mind in the Eyes Test

(RMET) to assess ToM. Clinical and sociodemographic data were collected. Participants were stratified by lifetime psychotic symptoms for between-group comparisons.

Results: Individuals with a history of psychotic symptoms (n=18) reported significantly higher total CTQ scores (Mean=61.0, SD=15.7) than those without psychotic symptoms (Mean=44.5, SD=13.1; p=0.028) and exhibited significantly lower ToM accuracy (Mean RMET=23.0, SD=4.5 vs. 25.4, SD=3.4; p<0.001, d=0.58).

Conclusions: In BD, the co-occurrence of childhood trauma and lifetime psychosis is associated with more pronounced ToM deficits. These results highlight the need for trauma-informed approaches and early cognitive interventions targeting social cognition in individuals with BD and psychosis history.

Keywords: Bipolar disorder, psychosis, childhood trauma, theory of mind, social cognition.

Introduction

Bipolar disorder (BD) is a chronic and disabling mood disorder affecting up to 2% of the population [1]. It remains one of the most debilitating psychiatric conditions, marked by persistent impairments across neurocognitive domains such as attention, memory, and executive function, even during euthymia [1, 2]. Many individuals fail to achieve or maintain full remission [3], which underscores the importance of identifying modifiable factors that shape the course and outcome of illness. Among these factors, exposure to childhood trauma is notably prevalent: up to 75% of individuals with BD report early-life adversity, a rate nearly double that of the general population [4]. While genetic and neurobiological factors have long been studied, increasing evidence highlights psychosocial and environmental influences, particularly childhood trauma, as critical determinants of illness onset, relapse risk, and functional impairment [5]. Meta-analytic data indicate that emotional abuse is the most frequent trauma subtype in BD [6].

Exposure to childhood trauma has enduring effects that extend into adulthood, including heightened vulnerability to mood, anxiety, substance use, and stress-related disorders, as well as adverse physical health outcomes [5]. Several self-report instruments have been developed to assess these experiences, among which the *Childhood Trauma Questionnaire* (CTQ) is the most widely used and validated

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[7,8]. Using this measure demonstrated that childhood trauma is associated with poorer social functioning in individuals with severe mental disorders, including BD, both during acute and remitted phases [9]. Broadly, trauma exposure appears to influence cognitive and psychosocial trajectories across psychiatric conditions [5, 10]. Although BD, major depressive disorder (MDD), and schizophrenia differ in symptomatology and treatment response [11], they share overlapping neurodevelopmental vulnerabilities such as structural and functional brain abnormalities and similar profiles of cognitive and social cognitive impairment [12-14]. Understanding both shared and disorder-specific mechanisms is essential to developing personalized interventions that improve cognitive and functional outcomes in BD. Notably, trauma exposure in BD is linked to poorer clinical outcomes, including elevated risk for suicidality and substance misuse [15-18].

Social cognition (SC), defined as the set of mental operations involved in perceiving, interpreting, and generating responses to social stimuli [19], has emerged as a key construct in explaining the link between trauma and functional outcomes in BD [15]. The DSM-5 identifies SC as one of six core neurocognitive domains, encompassing subdomains such as Theory of Mind (ToM), emotion recognition, emotion perception, social perception, and attributional style [20]. One of the main core domains of SC, ToM, refers to the ability to infer and attribute mental states such as beliefs, intentions, and emotions to oneself and others [21, 22]. ToM enables individuals to interpret social cues and predict others' behavior. Deficits in ToM were first identified in individuals with psychotic symptoms in the 1990s, with early studies focusing on schizophrenia and autism spectrum disorders. Research examining ToM impairments in BD began in the early 2000s [20, 22], and a recent meta-analysis suggests that ToM deficits are present in roughly one-third of individuals with BD [23]. Longitudinal data using the *Reading the Mind in the Eyes Test* (RMET) show that BD patients consistently perform below healthy controls, with deficits remaining stable over time [24]. Neuroimaging studies further implicate prefrontal and temporo-parietal networks in ToM deficits, regions also sensitive to early stress, suggesting a potential neural overlap between trauma exposure and impaired mental state inference[24, 25].

Within BD, psychotic symptoms constitute another important dimension that may modulate cognitive outcomes. Meta-analytic data suggest lifetime prevalence rates around 60–70% for BD I and 20–30% for BD II, with higher rates observed

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during manic or mixed episodes [4, 26, 27]. The presence of psychosis has been associated with earlier illness onset, greater chronicity, and poorer functional and educational outcomes [27, 28]. Evidence also suggests that individuals with BD who experience psychotic symptoms exhibit more pronounced ToM and social cognitive impairments than those without such features [13, 14]. This pattern may reflect partially shared neurobiological mechanisms, including disrupted fronto-limbic integration and altered salience attribution, which are also implicated in both trauma exposure and psychotic phenomena.

Despite strong evidence linking childhood trauma to both psychotic symptoms and social cognitive deficits independently, their interactive effects on ToM in BD remain underexplored. The present study, therefore, aimed to test two hypotheses in euthymic adults with BD: (1) higher levels of childhood trauma would be associated with poorer ToM performance, and (2) this association would be stronger among individuals with a history of psychotic symptoms. All participants endorsed some degree of childhood trauma; analyses thus focused on trauma severity, categorized as high or low according to established criteria [15, 29]. By investigating these interactions, this study seeks to fill a gap in the literature regarding the clinical heterogeneity of BD. Understanding how psychosis and childhood trauma jointly influence social cognition allows for the identification of a greater cognitive vulnerability in specific subgroups. These findings are fundamental to the development of improved rehabilitation strategies and to a deeper clinical understanding of these complex cases.

Methods

Study Design

This cross-sectional analysis of baseline data collected between January 2022 and December 2023 included 54 euthymic adults diagnosed with BD who were consecutively recruited from the outpatient Bipolar Disorder Program at the Institute of Psychiatry, University of São Paulo. This consecutive sampling approach targeted a representative clinical population within the specialized program. Euthymia was operationalized as scores < 8 on the Young Mania Rating Scale (YMRS) and < 12 on the Montgomery-Åsberg Depression Rating Scale (MADRS) obtained at the baseline assessment visit. Participants were 18-65 years old and met DSM-5 criteria for BD (Type 1 or Type 2) confirmed with the Mini-International Neuropsychiatric

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Interview (MINI)[30]. Exclusion criteria comprised (1) current substance-use disorder or use of psychoactive substances within 6 months, (2) neurological or developmental disorders, (3) intellectual disability, (4) inability to provide informed consent and (5) patients with other primary psychotic disorders. The study adhered to the Declaration of Helsinki and was approved by the Research Ethics Committee of the Faculty of Medicine, University of São Paulo (approval no. 4.040.551). Written informed consent was obtained from all participants.

Procedures

Data collection was conducted in a single session lasting approximately 60 to 90 minutes in a quiet room at the outpatient clinic. The data presented in this study are part of a larger clinical protocol investigating neuropsychological markers in BD. Upon arrival, participants provided written informed consent and completed a sociodemographic questionnaire. Subsequently, a trained psychologist administered the MINI to confirm diagnostic eligibility and assess the lifetime history of psychosis. Clinical stability was verified through the YMRS and MADRS scales. After confirming eligibility, participants performed a broader neuropsychological battery; however, for this study, we analyzed the results from the Reading the Mind in the Eyes Test (RMET), followed by the self-administered Childhood Trauma Questionnaire (CTQ). To minimize the impact of fatigue, short breaks were offered between assessments.

Tools

Sociodemographic and Clinical Questionnaire

A semi-structured questionnaire was used to collect sociodemographic data (age, sex, and years of education) and clinical information (duration of illness and medication use).

Childhood Trauma Assessment

Childhood trauma was assessed using the Brazilian version of the Childhood Trauma Questionnaire (CTQ), which comprises five subscales measuring emotional, physical, and sexual abuse, as well as emotional and physical neglect [7]. The Brazilian version of the CTQ has demonstrated good internal consistency and construct validity in clinical and non-clinical samples [7, 8]. The CTQ includes 28 statements describing traumatic childhood experiences, with participants rating the

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frequency of each experience on a 5-point Likert scale ranging from 1 ("Never") to 5 ("Very often"). An example item is: "*I had to wear dirty clothes*." Total scores range from 5 to 125, with higher scores indicating greater exposure.

Following established clinical thresholds[15, 29], the presence and severity of childhood trauma were evaluated across five sub-dimensions using validated cut-off scores: physical abuse (≥10), sexual abuse (≥8), emotional abuse (≥13), emotional neglect (≥15), and physical neglect (≥10). Each sub-dimension was assessed to characterize the trauma profile of the sample. For analytical purposes, CTQ total score was used as a continuous measure of overall trauma severity, as recommended for samples with high trauma prevalence[15, 29]. All participants endorsed at least one CTQ item above the minimal threshold. In the current sample, the CTQ demonstrated excellent internal consistency (Cronbach's α = 0.89 for total score).

Theory of Mind Assessment

Theory of Mind was evaluated using the Brazilian Portuguese version of the Reading the Mind in the Eyes Test (RMET) [31]. This test assesses the ability to infer others' mental states based solely on photographs of the eye region. The Brazilian Portuguese version of the RMET is a valid and reliable measure of Theory of Mind in Brazilian samples [31]. Each of the 36 photographs depicts the eyes of male or female actors expressing a specific mental state, accompanied by four adjectives. Participants are instructed to choose the adjective that best describes what the person in the photograph is thinking or feeling. The total score is calculated by summing the number of correct responses, with a maximum possible score of 36. The test typically takes about 15 minutes to complete.

Psychotic Symptoms

Lifetime history of psychotic symptoms was assessed through clinical interview and confirmed using the Mini-International Neuropsychiatric Interview (MINI), a structured diagnostic tool based on DSM-5 criteria [30]. Participants were classified into two groups according to the presence or absence of psychotic symptoms at any point during illness.

Statistical Analysis

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Statistical analyses included descriptive statistics, hypothesis testing, and correlation analyses. Means, standard deviations, and frequency distributions were calculated for sociodemographic and clinical variables. Comparisons between participants with and without psychotic symptoms were conducted using Student's t-tests or Mann–Whitney U tests for continuous variables and effect size (Cohen's d or rank biserial correlations), depending on the distribution assessed by Shapiro-Wilk test. Categorical variables were analyzed using Fisher's exact test or Pearson's chi-square test, as appropriate. Statistical significance was set at $p < 0.05$. Post-hoc sensitivity analysis indicated adequate power ($1-\beta = 0.80$) to detect medium-to-large effects ($d \geq 0.60$) given our sample size. All analyses and visualizations (e.g., bar charts, scatterplots) were performed using JAMOVI software, version 2.3.28.

Results

The final sample comprised 54 euthymic adults with bipolar disorder. The mean age was 39.8 years (SD = 10.5), and the majority were female (69.6%) and single (58.2%). Participants had an average of 15.4 years of education (SD = 2.4). Detailed sociodemographic characteristics are presented in Table 1.

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Table 1. Sociodemographic Characteristics by Presence of Psychotic Symptoms

	Full Sample (N=54)				Psychotic Symptoms										
					No (N = 36)				Yes (N = 18)						
	M	SD	N	%	M	SD	N	%	M	SD	N	%	t / X^2	p	ES
Age (years)	38.0	10.5			37.4	10.3			45.3	9.2			2.76	0.008	0.80
Gender															
Female			39.0	69.6			25.0	65.8			13.0	34.2	0.044	0.833	
Male			17.0	30.4			11.0	68.8			5.0	31.3			
Education (years)†	15.4	2.4			15.7	2.2			14.6	2.6			252.5	0.298	0.18
Marital Status															
Married			17.0	30.9			12.0	70.6			5.0	29.4			
Divorced			3.0	5.5			2.0	66.7			1.0	33.3			
Separated			1.0	1.8			0.0	0.0			1.0	100.0			
Single			32.0	58.2			21.0	70.0			9.0	30.0	4.824	0.484	
Cohabiting			1.0	1.8			1.0	100.0			0.0	0.0			
Widowed			1.0	1.8			0.0	0.0			1.0	100.0			

Note. †Mann–Whitney U test. M = Mean; N = Count; SD = Standard Deviation; % = Percentage; t = Student's t-test; χ^2 = Chi-square test; ES = Effect size (Cohen's d for parametric testes); p = p-value. X^2 and p-values refer to overall group comparisons for categorial variables.

Additionally, potential confounding effects of sex and age on primary outcomes were explored; no significant differences were found between males and females regarding RMET accuracy (p = 0.782) or CTQ total scores (p = 0.250), and age did not significantly correlate with RMET performance (p > 0.05). Consequently, these variables were not included as covariates in the final analysis.

Group Differences by History of Psychotic Symptoms

Among the 54 participants, 18 individuals (33.3%) reported a lifetime history of psychotic symptoms. Compared to participants without psychotic symptoms (n = 36), those with such history were significantly older (Mean = 45.3, SD = 9.2 vs. Mean = 37.4, SD = 10.3), t (52) = 2.76, p = 0.008, d = 0.80. A chi-square analysis confirmed that individuals aged ≥ 40 years were significantly more likely to report psychotic symptoms, χ^2 (1) = 10.8, p = 0.001. There were no significant group differences in gender distribution (χ^2 = 0.044, p = 0.833), education (U = 252.5, p = 0.298), or marital status (Fisher's exact test, p = 0.484) (Table 1).

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Clinical Characteristics

The average number of mood episodes was 11.3 (SD = 9.0), and participants had experienced an average of 1.1 hospitalizations (SD = 2.0). Overall symptom scores confirmed euthymia (YMRS Score: Mean = 1.4, SD = 2.0; MADRS Score: Mean = 4.6, SD = 3.0).

Participants with psychotic symptoms reported a higher number of medications (Mean = 2.4, SD = 1.0) than those without (Mean = 1.8, SD = 1.0), with a trend toward significance (U = 208.5, p = 0.053, r = 0.319). No significant group differences were observed for the number of episodes, hospitalizations, YMRS, or MADRS scores (Table 2).

Table 2. Clinical Characteristics by Presence of Psychotic Symptoms

	Full Sample		No Psychotic Symptoms		With Psychotic Symptoms				
Measure	**M**	**SD**	**M**	**SD**	**M**	**SD**	**U**	**p**	$ES_{(r)}$
Number of Medications	2.0	1.0	1.8	1.0	2.4	1.0	208.5	0.053	0.319
Number of Episodes	11.3	9.0	11.7	9.0	10.4	9.0	242.0	0.646	0.083
Number of Hospitalizations	1.1	2.0	0.7	1.0	1.7	2.0	208.5	0.109	0.257
YMRS Total	1.4	2.0	1.5	2.0	1.0	1.0	292.5	0.551	0.097
MADRS Total	4.6	3.0	4.7	2.0	4.5	3.0	315.5	0.882	0.026

Note. M = Mean; SD = Standard Deviation; YMRS = Young Mania Rating Scale; MADRS = Montgomery–Åsberg Depression Rating Scale; U = Mann–Whitney U test statistic; p = p-value; $ES_{(r)}$ = Rank biserial correlation (effect size for the Mann–Whitney U tests).

Associations Between Childhood Trauma, Theory of Mind, and Psychotic Symptoms

Significant group differences emerged in both childhood trauma exposure and Theory of Mind performance (Table 3; Figure 1):

- On the Reading the Mind in the Eyes Test (RMET), participants without psychotic symptoms scored higher (Mean = 25.4, SD = 3.4) than those with psychotic symptoms (Mean = 23.0, SD = 4.5), t (52) = 2.26, p = 0.028, d = 0.65.

The sample exhibited a high prevalence of childhood adversity, with a mean CTQ total score of 50.0 (SD = 15.9). When applying established clinical cut-offs to individual sub-dimensions (15, 29), all participants (100%) exceeded thresholds on at least one trauma type, indicating clinically significant exposure. Specifically, the

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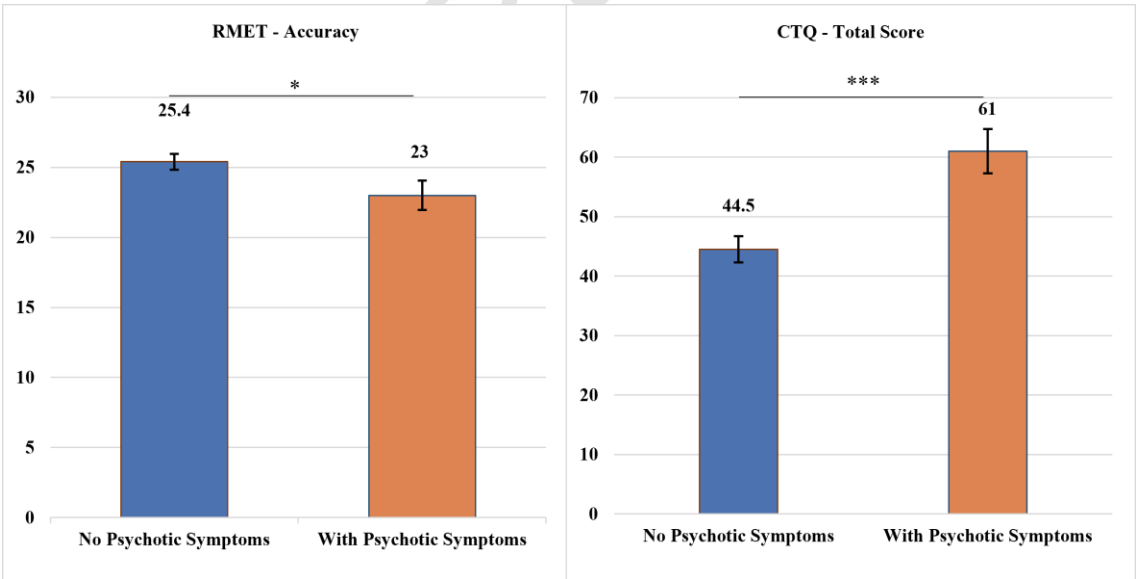
most frequently endorsed trauma types were emotional neglect (94% of participants) and emotional abuse (88%), consistent with prior BD samples. Given the universal presence of significant trauma exposure in our sample, we utilized CTQ total score as a continuous measure of trauma severity rather than creating artificial categorical groups. Individuals with psychotic symptoms reported significantly higher CTQ total scores (Mean = 61.0, SD = 15.7) compared to those without (Mean = 44.5, SD = 13.1), U = 135, $p < 0.001$, $r = 0.58$.

Table 3. Scale Scores by Presence of Psychotic Symptoms

	Full Sample		No Psychotic Symptoms		With Psychotic Symptoms				
Measure	**M**	**SD**	**M**	**SD**	**M**	**SD**	**t/U**	**p**	**ES**
RMET – Accuracy	24.6	4.0	25.4	3.4	23.0	4.5	2.262	**0.028**	0.65
CTQ – Total†	50.0	15.9	44.5	13.1	61.0	15.7	135	**0.0009**	0.58

Note. †Mann–Whitney U test. M = Mean; SD = Standard Deviation; RMET = Reading the Mind in the Eyes Test; CTQ = Childhood Trauma Questionnaire; U = Mann–Whitney U test statistic; p = p-value; ES = Effect size (Cohen's d for parametric tests; rank biserial correlation for nonparametric tests).

Figure 1. RMET performance and CTQ total scores in participants with and without lifetime psychotic symptoms. Data are presented as means. Error bars represent standard deviations.



Note. †RMET: Reading the Mind Test; ‡CTQ: Childhood Trauma Questionnaire. Asterisks indicate significant differences between groups (*$p < 0.05$; ***$p < 0.001$).

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Discussion

This study explored the associations among childhood trauma, ToM, and lifetime psychotic symptoms in a clinical sample of euthymic individuals with BD. Because all participants reported clinically significant trauma exposure, we were able to focus on trauma severity as a continuous dimension rather than presence versus absence of adversity. We found that BD patients with a history of psychotic symptoms were older, reported higher childhood trauma severity, and demonstrated poorer ToM performance than those without psychosis.

While illness burden markers such as the number of episodes and hospitalizations were comparable between groups, individuals with psychotic symptoms were prescribed more medications, suggesting greater illness complexity or treatment resistance. A particularly novel finding was the association between older age and psychotic symptoms in BD. This contrasts with prior longitudinal research indicating that the likelihood of psychosis decreases over the illness course [27]. This discrepancy may reflect: (a) selection bias, whereby older patients with more severe, psychosis-complicated illness remain engaged in specialized tertiary care; (b) cohort effects in diagnosis and symptom reporting; or (c) genuine chronicity-related emergence of psychotic features in a subgroup of patients. Alternatively, psychotic symptoms may be underreported or misattributed in younger individuals early in their illness trajectory. Future longitudinal research tracking the same individuals over time would help clarify this relationship.

Notably, we did not observe significant effects of sex or age on ToM performance or trauma severity in our sample. This contrasts with large-scale population studies showing sex and age differences in RMET accuracy[32]. The absence of these effects in our study may reflect the clinical homogeneity of our sample; all participants had BD with significant trauma exposure, which may have attenuated demographic variability typically observed in a community sample.

Consistent with our hypothesis, individuals with psychotic symptoms exhibited significantly lower ToM performance (Cohen's d = 0.65), a medium-to-large effect. This aligns with robust meta-analytic evidence indicating that ToM is impaired in BD across mood states [33] and persists during euthymia as a trait-like vulnerability [24]. Though generally milder than those observed in schizophrenia, social cognition impairment, particularly in ToM, are consistently found in BD and appears magnified in individuals with psychotic features[33, 34]. Our findings this literature by

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demonstrating that even during clinical stability, a history of psychosis is associated with more pronounced ToM deficits.

The overall pattern observed in our sample suggests a cumulative burden: individuals with both high childhood trauma severity and a history of psychosis performed worst on ToM tasks, supporting the hypothesis that these risk factors interact rather than operate independently. This observation accords with developmental models proposing that early adversity disrupts the neurocognitive maturation of systems supporting social cognition, thereby increasing vulnerability to both ToM deficits and psychotic phenomena [33].The strong association between psychotic symptoms and higher childhood trauma scores (r = 0.58) further supports existing literature linking early-life adversity with more severe clinical trajectories in BD [34, 35]. Meta-analytic studies indicate that childhood trauma significantly elevates the likelihood of psychosis across diagnostic categories [36]. Some studies suggest dose-response effects: greater numbers of adverse childhood experiences have been linked to increased likelihood of mood-congruent hallucinations[35], and higher psychosis risk with multiple trauma types [37]. However, findings are not entirely consistent; some investigations report no clear association between trauma number and psychosis history[38, 39] underscoring the importance of considering trauma type, timing, and developmental context. Our findings support a developmental pathway in which early trauma, particularly emotional neglect and abuse (the most prevalent types in our sample), disrupts the maturation of neural circuits underlying social cognition[40, 41]. Neuroimaging evidence indicates that childhood trauma is associated with altered brain activity in regions supporting ToM, including prefrontal and temporo-parietal networks[25]. Impaired mentalizing capacity may contribute to maladaptive interpersonal schemas, difficulties accurately interpreting others' intentions, and ultimately, paranoid ideation and psychotic misinterpretations[14]. This mechanistic model warrants further investigation through longitudinal neuroimaging studies. Several limitations must be acknowledged. First, the modest sample size (n=54) and gender imbalance (70% female) limit generalizability and statistical power to detect small effects or interaction terms. Second, trauma was assessed via retrospective self-report, which may be subject to recall bias, though the CTQ is a well-validated instrument with demonstrated reliability in clinical populations. Third, we assessed ToM using a single affective mentalizing task (RMET), which may not capture cognitive ToM or other social cognition domains such as attributional style or

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emotion regulation. Fourth, the cross-sectional design precludes causal inferences regarding the temporal relationships among trauma, psychosis, and ToM deficits. Finally, the absence of a trauma-free comparison group limits our ability to establish whether observed effects are specific to trauma severity or reflect broader BD-related impairments. However, this design reflects the clinical reality that trauma exposure is nearly ubiquitous in BD populations seeking tertiary care, supporting the value of dimensional trauma assessment. Future research should incorporate broader social cognition batteries, neuroimaging markers, healthy controls, and longitudinal designs to clarify developmental pathways and potential mediating mechanisms.

From a clinical perspective, these findings underscore the value of integrating trauma-informed approaches into routine BD assessment and treatment. We recommend: (a) systematic screening for childhood trauma severity using validated instruments such as the CTQ; (b) incorporation of brief ToM measures (e.g., RMET) into neuropsychological assessment batteries, particularly for patients with a history of psychotic symptoms; and (c) targeted psychosocial interventions to enhance social cognition in high-risk subgroups. Evidence-based approaches such as metacognitive training[42, 43] and social cognition and interaction training[44, 45] have shown initial promise in improving functional outcomes and some social cognition measures in BD. Tailoring such interventions to BD patients with both trauma histories and psychotic features may improve psychosocial functioning and potentially reduce relapse risk, though this hypothesis requires empirical testing. Future intervention research should examine whether strengthening ToM abilities can reduce functional impairment and lower vulnerability to psychotic symptoms in this high-risk subgroup.

This study makes several unique contributions to the literature. First, unlike previous research examining trauma or psychosis in isolation, we specifically investigated their interactive effects on social cognition in BD, a gap identified in recent reviews. Second, our exclusive focus on euthymic patients ensures that observed ToM deficits reflect trait-like vulnerabilities rather than state-dependent mood effects, strengthening inferences about enduring cognitive profiles. Third, the universal presence of significant trauma exposure in our sample (100% exceeding clinical thresholds) reflects the clinical reality of tertiary BD populations and supports dimensional rather than categorical approaches to trauma assessment. Finally, by

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combining validated trauma assessment, diagnostic confirmation of psychosis history, and performance-based ToM measurement in a clinically stable sample, we provide robust evidence for the cumulative impact of these risk factors on social-cognitive functioning.

In conclusion, our findings demonstrate that in BD, the co-occurrence of childhood trauma and lifetime history of psychosis is associated with more pronounced deficits in ToM. These results emphasize the importance of incorporating trauma-informed and social cognition-focused approaches into clinical care. Routine assessment of trauma history and ToM performance may enhance clinical understanding and support more targeted, personalized treatment planning. Longitudinal studies are needed to determine whether improving ToM through targeted interventions contributes to better long-term functional outcomes and reduced psychotic vulnerability in this high-risk population.

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Disclosure

The authors declare no conflicts of interest related to the publication of this article.

Data availability

Due to legal and ethical considerations, our research data is not publicly available. Interested parties may access the data by contacting the first author directly.

Author contributions

LSR and MECB were responsible for conceptualization, methodology, validation, and writing – original draft and review & editing. AGG contributed to formal analysis and writing – review & editing. RC, CN, MSF, TBB, and TC participated in methodology, data curation, and writing – review & editing. CCAR and BL contributed to supervision, conceptualization, and writing – review & editing. All authors have read and approved the final version submitted and take public responsibility for all aspects of the work.

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