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Social cognition in patients with schizophrenia spectrum and bipolar disorders with and without psychotic features☆

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ABSTRACT

Background: Social cognition may be critical to the impoverished social functioning seen in serious mental illness. However, although social-cognitive deficits are consistently demonstrated in schizophrenia spectrum disorders (SSD), studies in bipolar disorder (BD) have produced inconsistent results. This inconsistency may relate to symptom profiles of patients studied, particularly the presence or absence of psychotic features. Thus, we examined social cognition in bipolar disorder with psychotic features (BD+) versus without psychotic features (BD−) relative to SSD and controls.
Methods: A sample of 537 SSD patients, 85 BD+ patients, 37 BD− patients, and 309 controls were administered the MATRICS Consensus Cognitive Battery, including a social cognition measure, the managing emotions branch of the Mayer–Salovey–Caruso Emotional Intelligence Test (MSCEIT). Analyses of covariance compared MSCEIT performance between diagnostic groups while controlling for race, psychotropic medication status, and neurocognition.
Results: SSD but not BD− or BD+ patients showed significant MSCEIT deficits relative to controls.
Conclusions: MSCEIT deficits were found in SSD but not BD− or BD+, suggesting that social cognition may represent an underlying difference between SSD and BD. However, variance in MSCEIT performance among BD patients may also suggest latent BD subgroups characterized by social-cognitive deficits. Findings can help inform future investigations into how social cognition and social brain development differ between SSD and BD.

1. Introduction

Bipolar disorder (BD) patients suffer from multiple functional impairments including impoverished social functioning (Cannon et al., 1997; Pope et al., 2007), which have prompted investigations into social-cognitive deficits in BD. Social cognition is a multidimensional construct that includes identifying affect others display, inferring others' mental states, attributing meanings to others' actions, reading others' social cues, applying a working knowledge of social rules to socially reciprocate in kind, and managing one's own emotional responses to maintain interpersonal relationships (Green et al., 2008a; Green and Horan, 2010; Pinkham et al., 2014). Understanding bipolar patients' social-cognitive deficits may help illuminate mechanisms underlying impaired social competence in BD, which may help inform interventions to improve psychosocial functioning.

Yet to date, results of social cognition studies in BD have been inconsistent. While some studies have found social-cognitive deficits (Bora et al., 2005; Bozikas et al., 2006; David et al., 2014; Harmer et al., 2002; Hoertnagl et al., 2011; Inoue et al., 2004; Kerr et al., 2003; Lembke and Ketter, 2002; Martino et al., 2008, 2011; Soeiro-de-Souza et al., 2012a,b; Wolf et al., 2010), others have found mixed results with preserved aspects alongside deficits (Addington and Addington, 1998; Getz et al., 2003; McKinnon et al., 2010; Montag et al., 2010; Olley et al., 2005; Schaefer et al., 2010; Shamay-Tsoory et al., 2009; Summers et al., 2006; Vederman et al., 2012), and others have found no deficits relative to controls (Barrera et al., 2013; Lee et al., 2013; Van Rheenen and Rossell, 2014; Vaskinn et al., 2007; Venn et al., 2004). Social-cognitive differences have also been found between BD−I and BD−II, or between manic or depressive phases of BD (Derntl et al., 2009; Gray et al., 2006). As a result, the nature of social-cognitive deficits in BD remains inconclusive.

In contrast, schizophrenia spectrum disorders (SSD) have consistently shown substantial deficits in both social cognition and

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neurocognition (Corbera et al., 2013; Dawson et al., 2012; Eack et al., 2010; Fiszdon et al., 2013; Green et al., 2012; Kee et al., 2009; Kern et al., 2011; Kimhy et al., 2012; Thompson et al., 2012; Torgalsbøen et al., 2014; Tso et al., 2010). Findings suggest that such deficits remain stable across phases of psychosis (Green et al., 2012) and are linked to SSD patients' quality of life and community, work, and social functioning (Fiszdon et al., 2013; Horan et al., 2012; Kee et al., 2009; Maat et al., 2012; Shamsi et al., 2011; Shi et al., 2013; Tso et al., 2010). Moreover, results suggest that social cognition is more strongly linked with community functioning than neurocognition (Fett et al., 2011), and mediates the relationship between neurocognition and functional outcomes (Bell et al., 2009; Schmidt et al., 2011).

Recently, it has been suggested that inconsistent past findings in BD may reflect the impact of co-occurring psychotic symptoms on social-cognitive performance (Thaler et al., 2013a,b), especially considering ~60% of bipolar patients will also experience psychotic symptoms at some point during their lifetimes (Dunayevich and Keck, 2000). Specifically, bipolar patients with psychotic features (BD+) have shown greater social-cognitive deficits than those without psychotic features (BD−) (Thaler et al., 2013a,b). Although we previously reported (Burdick et al., 2011) no significant effect of a history of psychotic features in a bipolar sample substantially overlapping with the sample used in our current analyses, our prior work did not control for neurocognition and did not directly compare against SSD, which may have limited these past findings. In addition, although a study by Lee et al. (2013) also found no effect of a history of psychotic features on their BD patients' social-cognitive deficits, only 15 of their 68 patients (~22%) had a history of psychotic features, and thus this sample may have been underpowered to detect group differences. Therefore, it is possible that social-cognitive deficits may present in BD+ but not BD− when directly compared to SSD and controls.

Examining social cognition across SSD, BD+, BD−, and controls may illuminate whether a history of psychotic features impairs social cognition in BD relative to SSD. Findings could help identify BD patients in need of social-cognitive interventions and help elucidate if social cognition represents an underlying difference between BD and SSD. Thus, we compared social-cognitive performance differences among these diagnoses while controlling for neurocognition and psychotropic medication status.

2. Methods

2.1. Participants

Our sample consisted of 968 participants, with 537 SSD patients (447 schizophrenia and 90 schizoaffective disorder patients), 85 BD+ patients, 37 BD− patients, and 309 controls. For full demographics and clinical characteristics, see Table 1. All participants gave written informed consent to protocols approved by the Institutional Review Board of the North Shore-Long Island Jewish Health System (NSLIJ). Controls were recruited using newspaper and Internet advertisements, posted flyers, and personal referrals. Patients were referred from the outpatient and inpatient psychiatric departments of Zucker Hillside Hospital (ZHH), part of NSLIJ. Controls were excluded if diagnosed with past or present Axis-I disorders, had substance abuse within the past month, or had a history of learning disability, neurological disorder, or CNS trauma. Patients were excluded if they had a history of learning disability, neurological disorder, or CNS trauma.

2.2. Clinical assessments

2.2.1. Diagnostic assessment

Controls were administered the Structured Clinical Interview for the DSM-IV, non-patient edition (SCID-I/NP; First et al., 2001a) and patients were administered the patient edition (SCID-I/P; First et al., 2001b) by Ph.D. or Master's level psychometricians. Controls' SCIDs were compiled into narrative case summaries and no Axis-I pathology was determined by consensus of two senior ZHH faculty. Patients' SCIDs were supplemented by available records and compiled into narrative case summaries and diagnoses were determined by consensus of three senior ZHH faculty.

Table 1
Study group and subsample demographics and clinical characteristics.

	Healthy control	Bipolar disorder (BD) without psychotic features		Bipolar disorder (BD) with Psychotic features		SSD spectrum disorder	Statistic	*p*
Total sample *N*	309	37		85		537		
	–	BD 1 ($N = 17$)	BD 2 ($N = 20$)	BD 1 ($N = 85$)	BD 2 ($N = 0$)	–	–	–
Age: mean (SD)	40.52 (16.34)	41.24 (10.06)		40.61 (11.43)		43.26 (10.20)	$F(3,967) = 3.62$	$p < .05$
% Female	55.99%	59.46%		55.29%		26.82%	$\chi^2(3,967) = 86.01$	$p < .001$
% Caucasian	52.75%	54.05%		56.47%		37.80%	$\chi^2(6,967) = 39.85$	$p < .001$
Substance disorder history	0%	16.22%		27.05%		40.22%	–	–
Anticonvulsant use	0	18		32		186	–	–
Antidepressant use	0	15		19		161	–	–
Mood stabilizer use	0	11		21		40	–	–
Antiparkinsonian use	0	4		10		139	–	–
Anxiolytic use	0	5		8		56	–	–
Antipsychotic use	0	23		54		461	–	–
Sedative/Hypnotic use	0	4		6		26	–	–
Stimulant use	0	1		0		6	–	–
N of subsample with BPRS	–	21		60		492	–	–
	–	BD 1 ($N = 11$)	BD 2 ($N = 10$)	BD 1 ($N = 60$)	BD 2 ($N = 0$)	–	–	–
Brief psychiatric rating scale (BPRS): Mean (SD)	–	–		–		–	–	–
Thinking Disturbance	–	3.86 (1.11)		4.23 (2.04)		6.16 (3.04)	$F(2,572) = 17.15$	$p < .001$
Withdrawal/Retardation	–	3.05 (1.07)		2.75 (1.28)		3.66 (1.86)	$F(2,572) = 7.86$	$p < .001$
Hostile/Suspiciousness	–	4.86 (1.68)		4.45 (1.78)		4.99 (2.06)	$F(2,572) = 1.92$	ns
Anxious/Depression	–	6.48 (2.89)		5.02 (2.40)		4.85 (2.22)	$F(2,572) = 5.22$	$p < .01$
Activation	–	4.86 (2.01)		4.47 (1.92)		4.47 (1.86)	$F(2,572) = 0.43$	ns
BPRS Total Score	–	29.10 (5.80)		27.07 (7.58)		31.14 (7.44)	$F(2,572) = 8.58$	$p < .001$

2.3. Symptom assessment

Patients' clinical symptoms were assessed by trained Master's level psychometricians using the *Brief Psychiatric Rating Scale* (BPRS; Overall and Gorham, 1962), a semi-structured clinical interview assessing 18 symptoms within the past week.

2.4. Cognitive assessment

To assess neurocognition, all participants were administered the MATRICS Consensus Cognitive Battery (MCCB), which consists of 10 measures assessing seven domains: (1) processing speed, measured by the symbol coding subtest of the *Brief Assessment of Cognition in Schizophrenia* (BACS symbol coding; Keefe et al., 2004), the *Trail Making Test: Part A* (Trails-A; Army Individual Test Battery, 1944), and *Category Fluency: Animal Naming* (animal naming; Spreen and Strauss, 1998); (2) attention/vigilance, measured by the *Continuous Performance Test—Identical Pairs* (CPT-IP; Cornblatt et al., 1988); (3) working memory, measured by the spatial span subtest of the *Wechsler Memory Scale, Third Edition* (WMS-III spatial span; Wechsler, 1997) and *Letter–Number Span* (LNS; Gold et al., 1997); (4) verbal learning, measured by the *Hopkins Verbal Learning Test—Revised* (HVLT-R; Brandt and Benedict, 2001); (5) visual learning, measured by the *Brief Visuospatial Memory Test—Revised* (BVMT-R; Benedict, 1997); (6) reasoning and problem solving, measured by the *Neuropsychological Assessment Battery* mazes subtest (NAB Mazes; Stern and White, 2003); and (7) social cognition, measured by the managing emotions branch of the *Mayer–Salovey Emotional Intelligence Test* (MSCEIT; Mayer, 2002).

The MSCEIT has evidenced robust psychometric properties, with demonstrated applicability in BD (Burdick et al., 2011; Lee et al., 2013), SSD (Green et al., 2008b), and controls (Kern et al., 2008), good internal consistency (Eack et al., 2010; Mayer et al., 2003), good sensitivity to deficits in SSD and BD (Burdick et al., 2011; Kee et al., 2009; Van Rheenen and Rossell, 2014), and good validity in SSD, as SSD patients' MSCEIT performance has been significantly linked to social functioning (Shamsi et al., 2011) and residential independence (Lin et al., 2012), and has been improved by social-cognitive interventions (Eack et al., 2007).

2.5. MCCB scoring

While published norms were available for each MCCB domain (Kern et al., 2008), our sample of healthy control participants was better demographically matched to our bipolar and schizophrenia spectrum patient samples, as the Kern et al. (2008) data were not stratified by race. Specifically, the Kern et al. (2008) healthy control sample consisted of 76% Caucasian participants, whereas our healthy control participants consisted of 52.75% Caucasian participants, which was better matched to our BD −, BD +, and SSD patients (being 54.05%, 56.47% and 37.8% Caucasian, respectively). Thus, we chose to calculate Z-scores using means and standard deviations derived from our healthy control data, which provided improved demographic matching on race. Using our healthy control data, age and sex were regressed out of each MCCB raw score to produce standardized residuals that were then transformed to Z-scores. A neurocognition score was calculated by averaging across the 6 nonsocial MCCB domains. Next, MSCEIT and neurocognition Z-scores were inspected for outliers over ±3 standard deviations from the mean. There were no outliers for neurocognition, but 4 extreme low MSCEIT scores were identified and excluded (i.e. one BD +, two SSD, and one control case). Resulting age- and sex-corrected Z-scores consisted of (1) an MSCEIT Z-score and (2) a neurocognition Z-score. Both were normally distributed (K–S = 0.83 and 1.16 for neurocognition and MSCEIT, respectively, both p's > 0.05). Since we chose to derive MCCB scores using our own healthy control data, we have provided T-score means and standard deviations for all our diagnostic groups using the MCCB published norms in Table 2 for descriptive purposes and for ease of comparison to previous work on the MCCB.

2.6. Data analysis

Diagnostic groups were first compared on demographics using chi-squares for sex and race and analysis of variance for age. Demographic variables that significantly differed between groups were corrected for or included as covariates.

To investigate whether MSCEIT deficits would be demonstrated in all BD patients relative to SSD and controls, we first compared SSD patients, all BD patients, and controls using analysis of covariance (ANCOVA) controlling for race, neurocognition, and psychotropic medication status. Next, to examine the impact of psychosis history on MSCEIT performance in BD, we conducted a follow-up ANCOVA comparing SSD, BD +, BD −, and control groups, controlling for race, neurocognition, and psychotropic medication status.

Race consisted of a categorical variable with Caucasian, African-American, and other minority group. Psychotropic medication status consisted of 8 dummy-coded variables that each assessed the presence or absence of anticonvulsant, antidepressant, mood stabilizer, antiparkinsonian, anxiolytic, antipsychotic, sedative, or stimulant use. All patients were on at least one form of psychotropic medication and thus all 8 variables were included in all ANCOVAs. No controls had any history of psychotropic medication use.

3. Results

Preliminary analyses revealed the sex and race distributions significantly differed between diagnostic categories, with fewer females and more minorities in the SSD group compared to other groups (sex: $\chi^2(3,967) = 86.01$, $p < 0.001$; race: $\chi^2(6,967) = 39.85$, $p < 0.001$). Age also significantly differed between groups, with SSD patients slightly older than controls but no significant age differences between any other groups ($F(3,967) = 3.62$, $p < 0.05$). Thus, we corrected all MCCB subtests for age and sex and controlled for race in all ANCOVAs.

Results from the first ANCOVA found significant MSCEIT differences between SSD patients, all BD patients, and controls ($F(2, 964) = 24.85$, $p < 0.001$), controlling for race, neurocognition, and psychotropic

Table 2
MCCB domain scores by diagnostic group standardized using MCCB normative data.

MCCB domain	Healthy control ($N = 309$)	Bipolar disorder without psychotic features ($N = 37$)	Bipolar disorder with psychotic features ($N = 85$)	SSD spectrum disorder ($N = 537$)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Processing speed	48.63 (10.43)	40.19 (10.69)	38.12 (9.05)	29.80 (14.03)
Attention vigilance	46.97 (10.66)	39.76 (12.91)	36.89 (12.86)	32.13 (12.36)
Working memory	44.82 (10.44)	37.57 (13.04)	37.47 (10.51)	31.76 (13.16)
Verbal learning	42.59 (8.28)	39.38 (8.96)	37.76 (7.29)	34.50 (7.36)
Visual learning	41.57 (11.74)	34.84 (12.27)	35.80 (12.46)	30.31 (12.23)
Reasoning/problem solving	45.68 (10.38)	42.22 (8.72)	41.01 (8.75)	37.49 (8.33)
Social cognition	49.06 (10.98)	47.46 (11.34)	44.08 (13.50)	35.67 (12.05)
Overall composite score	42.68 (10.68)	34.00 (11.60)	31.67 (11.35)	22.55 (12.13)

medication status. Follow-up pairwise comparisons using Fisher's LSD found no significant MSCEIT differences between BD patients and controls, but rather only significant differences between SSD patients and controls and between BD and SSD patients. Significant covariates included neurocognition (F (1,964) = 92.29, $p < 0.001$) and antidepressant use (F (1,964) = 11.59, $p < 0.001$), while all other covariates were non-significant (all p's > 0.05).

Results from the second ANCOVA also found significant MSCEIT differences between the BD +, BD −, SSD, and control groups (F (3,964) = 16.83, $p < 0.001$), controlling for race, neurocognition, and psychotropic medication status. Follow-up pairwise comparisons using Fisher's LSD found no significant MSCEIT differences between BD +, BD −, and controls. Rather, the only significant difference was between SSD and all other groups. Significant covariates similarly included neurocognition (F (1,964) = 91.99, $p < 0.001$) and antidepressant use (F (1,964) = 11.02, $p < 0.001$), while all other covariates were non-significant (all p's > 0.05). For a plot of MSCEIT means across all four diagnostic groups, see Fig. 1.

4. Discussion

This study's primary aim was to examine social cognition across SSD, BD +, and BD − patients relative to controls. In particular, given ~60% of BD patients also experience psychotic symptoms at some point during their lifetimes, often during acute phases of illness (Dunayevich and Keck, 2000), and recent studies have found BD + patients to show significant social-cognitive deficits (Thaler et al., 2013a,b), our study aimed to elucidate whether BD + patients would show impaired MSCEIT performance relative to BD − patients and controls, as well as whether deficits would be consistent with SSD patients. However, in contrast to recent studies showing social-cognitive deficits in BD + (Thaler et al., 2013a,b), but consistent with past work showing no social-cognitive deficits in BD (Barrera et al., 2013; Lee et al., 2013; Vaskinn et al., 2007; Venn et al., 2004), our analyses found significant MSCEIT deficits in SSD but not BD + or BD − patients relative to controls. These results suggest that social cognition may represent a key underlying difference between SSD and BD.

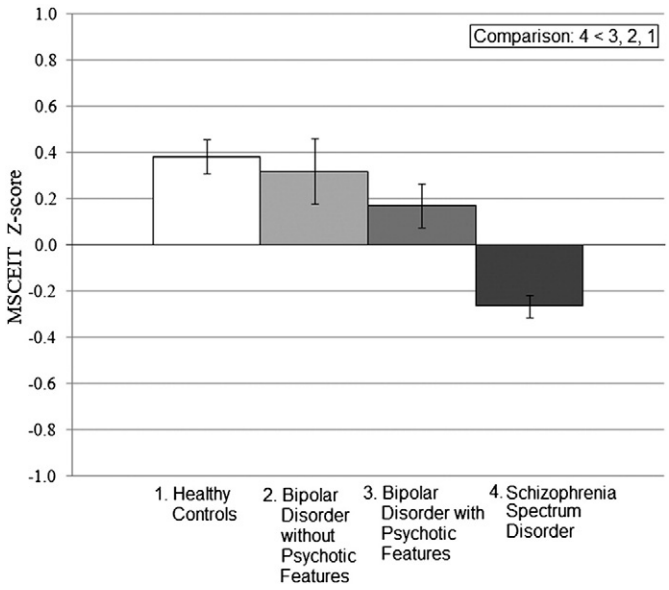

Fig. 1. Social cognition performance across diagnostic categories. *Note*: Error bars represent ±1 standard error. MSCEIT = the managing emotions branch of the Mayer–Salovey–Caruso Emotional Intelligence Test, the social cognition measure of the MATRICS Consensus Cognitive Battery (MCCB). All MCCB scores including the MSCEIT are age- and sex-corrected, and the comparison of MSCEIT performance across diagnostic groupings controlled for race and neurocognition.

Another possible explanation is that social-cognitive impairments of BD patients are more heterogeneous than those found in SSD. Consistent with this, our sample of BD + and BD − patients both had larger estimates of standard error on the MSCEIT than the other diagnostic groups, which might indicate the presence of latent bipolar subgroups that differ in their social-cognitive performance. To investigate this possibility, we examined a histogram of MSCEIT Z-scores for easily visible subgroups within our bipolar sample (both BD + and BD −). The distribution was unimodal and normally distributed (K–S = 0.83, $p > 0.05$), which was not suggestive of latent bipolar subgroups. It is still possible that there are latent subgroups of BD patients with different levels of social-cognitive performance, but that properly identifying these subgroups requires the use of more advanced statistical techniques such as hierarchical cluster analysis. Although outside the scope of the present study, future studies should use larger bipolar cohorts and more sophisticated analytic methods to investigate this possibility of latent subgroups of bipolar patients with differential social-cognitive performance.

An important limitation of our study was the lack of multiple measures of social cognition and the limited scope of the MSCEIT as social cognition measure. Specifically, the MSCEIT has evidenced some weaknesses in its ability to fully tap into the multifaceted construct of social cognition (Burdick et al., 2011), and Lee et al. (2013) used a battery that included multiple aspects of social cognition. Unfortunately, multiple social-cognitive measures were not available for our study. However, one benefit of the MSCEIT is that it is particularly well-suited to our study's transdiagnostic focus. Ceiling effects are a key problem for many social-cognitive measures, with controls often performing at over 90% accuracy (Dodell-Feder et al., 2013). As a result, past studies may have lacked sensitivity to subtle social-cognitive differences between BD patients and controls. In addition, the factor structure of social cognition in populations suffering from psychotic symptoms is still largely unknown and many measures have inadequate psychometric properties (Green et al., 2008a). In contrast, the MSCEIT was both developed for non-clinical populations and was the chosen consensus metric of the MCCB for studying serious psychiatric disorders (Kern et al., 2008; Nuechterlein et al., 2008), Thus, while limited in scope, the MSCEIT is apt for studying social-cognitive deficits across SSD and BD subtypes and for verifying past evidence of social-cognitive deficits in BD + patients (Lee et al., 2013; Thaler et al., 2013a,b). Nevertheless, social cognition remains a multifaceted construct that is not yet fully understood, and it is possible that BD patients may show deficits in social-cognitive domains not fully captured by the MSCEIT. Future studies are needed to develop and validate standardized, multifaceted social-cognitive batteries sensitive to subtle differences across clinical and nonclinical populations. Large-scale studies are also needed to assess performance on these batteries across all SSD and BD subtypes.

Our findings were also limited by differences in demographic distributions between study groups. Specifically, our SSD group was slightly older and had fewer females and more minorities compared to our other groups. However, we controlled for race in all analyses and MCCB subtests were all age- and sex-corrected, which helped attenuate any demographic differences between groups.

Although it may be argued that differences in symptom severity of our patient groups contributed to our findings, we ran a follow-up ANCOVA controlling for the total symptom severity score of the BPRS in a subsample of 570 patients who had been administered the BPRS (i.e., 21 BD −, 59 BD +, and 490 SSD patients). Results confirmed our findings, with no significant MSCEIT deficits in BD − or BD + but significant deficits in SSD relative to controls. Nevertheless, BPRS ratings were unfortunately not available for every patient and thus the possibility clinical symptoms impacted our findings cannot be completely ruled out. Moreover, differences in symptoms are often accompanied by differences in medication status. However, our analyses controlled for 8

classes of psychotropic medications and although including these 8 covariates had the potential to adversely affect statistical power, our large sample size withstood the inclusion of these covariates while maintaining excellent power ($1 - \beta = 0.9$).

In summary, our evidence supports past work showing social-cognitive deficits in SSD but not BD+ or BD− relative to controls (Barrera et al., 2013; Lee et al., 2013; Vaskinn et al., 2007; Venn et al., 2004). While these data suggest that social cognition may represent an underlying difference between SSD and BD, the large variance in social-cognitive performance among the bipolar groups is also suggestive of heterogeneity in performance and may indicate a latent bipolar subgroup characterized by social-cognitive deficits. Results highlight the importance of developing standardized social-cognitive batteries for use across BD and SSD and emphasize the need for future work on social brain development in clinical populations.

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Contributors

Dr. Burdick, Dr. Malhotra, and Dr. DeRosse were responsible for protocol and study design and management of data collection. Dr. Nitzburg and Dr. DeRosse undertook the literature search and statistical analyses of the present study. All authors have contributed to and have approved the final manuscript.

Conflict of Interest

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

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