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Commonalities in social and non-social cognitive impairments in adults with autism spectrum disorder and schizophrenia

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ABSTRACT

Autism spectrum disorder (ASD) and schizophrenia are both conditions that are characterized by impairments in social and non-social cognition, yet commonalities in the magnitude and domains of cognitive deficits across these two conditions remain unclear. This study examined neurocognitive and social-cognitive functioning in 47 outpatients with schizophrenia, 43 verbal adults with ASD, and 24 healthy volunteers. A comprehensive neuropsychological battery assessing processing speed, attention, memory, and problem-solving domains was administered along with a social-cognitive battery of emotion processing. Results demonstrated large and significant impairments in emotion processing and neurocognition relative to healthy individuals in participants with autism ($d = -.97$ and -1.71, respectively) and schizophrenia ($d = -.65$ and -1.48, respectively). No significant differences were observed between those with ASD and schizophrenia on any cognitive domain assessed, and the areas of greatest impairment were identical across both disorders and included slowness in speed of processing and an inability to understand emotions. These findings indicate a high degree of similarity in the cognitive challenges experienced by verbal adults with autism and schizophrenia, and the potential need for trans-diagnostic remediation approaches to enhance cognition in these conditions.

1. Introduction

Autism spectrum disorder (ASD) and schizophrenia are severe neurodevelopmental disorders that are both characterized by considerable impairments in social and emotional function and information processing (Lewis and Levitt, 2002; Volkmar et al., 2004). The earliest definitions of schizophrenia considered autism to be a childhood form of the illness, in part due to the gross cognitive and functional impairments observed in the two disorders (Bleuler, 1911). Although subsequent clinical evidence delineated unique aspects of the two conditions (Kanner, 1965), the notion that ASD and schizophrenia share some important commonalities in phenomenology (Couture et al., 2010), pathophysiology (Guilmatre et al., 2009; Pinkham et al., 2007; Sugranyes et al., 2011), and treatment (McCracken et al., 2002) has remained long after their distinction in modern psychiatric nosology.

Impairments in information processing, particularly social information processing, are some of the deficits most commonly noted to be shared between ASD and schizophrenia (e.g., Sugranyes et al., 2011). While many studies have demonstrated impairments in social domains within autism and schizophrenia samples (Baron-Cohen et al., 1985; Penn et al., 1997; Heinrichs and Zakzanis, 1998; Ozonoff et al., 2004), few studies have compared the domains and magnitude of cognitive impairment between these conditions. Early work by Schneider and Asarnow (1987) examined 11 individuals with childhood schizophrenia and 15 lower functioning children with autism, and found similar levels of impairment in processing speed and executive function. Another study found that a sample of 31 verbal adults with ASD had similar degrees of processing speed and comprehension as a psychometrically defined subsample of high-functioning inpatients with schizophrenia (Goldstein et al., 2002). With regard to social cognition, Pilowsky et al. (2000) identified similar theory of mind impairments in 12 individuals with childhood onset schizophrenia and 12 children with autism relative to healthy controls. Sasson et al. (2007) also found comparable levels of slowing in gaze orientation toward social stimuli among 10 adults with schizophrenia and 10 adults with autism, although the autism

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group demonstrated a differential slower speed of orienting toward emotional faces. Conversely, some have found greater impairments in facial emotion perception in children with autism compared to schizophrenia (Bolte and Poustka, 2003), and other studies have found impairments in this domain to be similar among adults with these two conditions (Couture et al., 2010). Several studies have also found overlap in the neurobiologic deficits present in ASD and schizophrenia, particularly with regard to functional abnormalities associated with social-cognitive impairment (Pinkham et al., 2007; Sugranyes et al., 2011).

Although there is growing evidence of meaningful overlap in social and non-social impairments among individuals with ASD and schizophrenia, these studies have mostly been characterized by small sample sizes, particularly in the patient groups, which may have precluded detecting differences in cognitive impairment across these disorders, and few studies have used rigorous methods for diagnosing ASD and schizophrenia. Furthermore, comparative studies focusing on differences in non-social cognitive impairment in higher functioning verbal adults with autism, who may be less cognitively disabled, have not been conducted. This study sought to examine neurocognitive impairments and social-cognitive deficits in emotion processing in rigorously diagnosed and well-characterized samples of verbal adults with autism and adults with schizophrenia compared to healthy individuals, in an effort to identify similarities and differences in cognitive function that could demonstrate a need for shared and/or unique approaches to the treatment of cognition in these populations.

2. Method

2.1. Participants

Participants consisted of 47 outpatients with schizophrenia, 43 adults with ASD, and 24 healthy control individuals participating in ongoing studies of cognitive enhancement therapy (CET; Hogarty and Greenwald, 2006) and the neural basis of social cognition at the University of Pittsburgh. Schizophrenia patients were included if they (1) were between the ages of 18 and 60; (2) were diagnosed with schizophrenia or schizoaffective disorder using the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002); (3) were receiving and adherent to antipsychotic medication; and (4) had an IQ ≥80. Adults with autism were included if they (1) were between the ages of 16 and 45; (2) met criteria for autism or autism spectrum using the autism diagnostic observation schedule (ADOS; Lord et al., 2000); (3) had an IQ ≥80; (4) had not been abusing substances within the past 3 months prior to study enrollment; (5) did not have a comorbid psychotic disorder according to the clinical record; and (6) demonstrated significant cognitive and social disability on the cognitive styles and social cognition eligibility interview (Hogarty et al., 2004). Cognitive and social disability criteria were part of the inclusion criteria for the ASD group to ensure that participants experienced sufficient disability to need treatment. Of the more than 150 potential ASD participants screened, none failed to meet cognitive and social disability criteria. Finally, healthy control individuals were included if they (1) were between the ages of 18 and 50; (2) were free from a current psychiatric diagnosis verified by the SCID; (3) did not have a family history of psychosis or ASD; and (4) were not abusing substances within the past 3 months.

Characteristics of enrolled participants are presented in Table 1. The average age for the overall sample was 29.32 ($SD = 10.23$) years, participants were predominantly male and mostly Caucasian. All individuals with schizophrenia were receiving antipsychotic treatment, and none of the adults with autism were taking antipsychotic medication. Patients in the schizophrenia sample tended to be significantly older, have lower IQ scores, and were less likely to be Caucasian than both autism and healthy control groups. In addition, as is common in the disorder, 28 of the patients with schizophrenia also met SCID criteria for substance abuse or dependence. Patients with schizophrenia were significantly less likely to be employed than adults with ASD, who were less likely to be employed than healthy individuals.

2.2. Measures

2.2.1. Neurocognition

Neurocognitive assessments included a broad array of standardized neuropsychological tests, most of which came from the MATRICS consensus cognitive battery, which is a comprehensive battery of cognitive assessments recommended by the National Institutes of Mental Health Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) initiative for use in clinical trials of cognitive enhancers in schizophrenia (Green et al., 2004). This battery consists of field standard measures of processing speed, attention/vigilance, working memory, verbal learning, visual learning, and problem-solving, and was augmented by the Wisconsin card sorting test (Heaton et al., 1993). For primary analyses, a composite index of neurocognition was formed, with higher scores indicating better cognition, by scaling test items to a common (z) metric, which demonstrated excellent internal consistency ($\alpha = .86$).

2.2.2. Social cognition

Assessments of social cognition focused on emotion processing and included the Mayer–Salovey–Caruso emotional intelligence test (MSCEIT; Mayer et al., 2003) and the Penn emotion recognition test (Kohler et al., 2003). The MSCEIT is a 141-item performance-based measure of the

Table 1
Demographic and clinical characteristics of adults with autism, schizophrenia, and healthy individuals.

	Healthy (HC; $N = 24$)		Autism (ASD; $N = 43$)		Schizophrenia (SZ; $N = 47$)		Analysis[a]	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	Direction
Age	26.25	5.52	24.86	5.75	34.96	12.48	<.001	HC, ASD < SZ
IQ	106.75	6.78	113.05	15.28	99.04	10.50	<.001	HC, ASD > SZ
	N	%	*N*	%	*N*	%	*p*	Direction
Male	16	67%	38	88%	34	72%	.075	
White	19	79%	36	84%	21	45%	<.001	HC, ASD < SZ
Attended college	22	92%	31	72%	33	70%	.113	
Employed	13	54%	17	40%	10	21%	.017	HC < ASD < SZ
Diagnosis								
Autism	–	–	26	60%	–	–		
Autism spectrum	–	–	17	40%	–	–		
Schizophrenia	–	–	–	–	22	47%		
Schizoaffective disorder	–	–	–	–	25	53%		

[a] χ^2 or analysis of variance test, two-tailed, for significant differences between autism, schizophrenia, and healthy control participants.

four domains of emotional intelligence outlined by Salovey and Mayer (1990). The test is performance-based in that participants are asked to solve emotional problems rather than self-report on their emotional abilities, is scored using consensus norms with a normative mean (*SD*) of 100 (15), and has been validated in normative (Mayer et al., 2003) and patient (Nuechterlein et al., 2008; Eack et al., 2010) populations. The Penn emotion recognition test was used to assess facial emotion perception, where participants are presented with 40 emotional (happy, sad, angry, and fearful) or neutral faces and asked to choose the appropriate emotional label for each face. The measure has been widely used and validated in previous psychiatric research (e.g., Kohler et al., 2003), and provides an assessment of both accuracy and response time. A composite index of social cognition was constructed for primary analyses by scaling items from these measures to a common (*z*) metric, which also demonstrated adequate internal consistency ($\alpha = .83$).

2.3. Procedures

Participants were recruited from support groups, community agencies, colleges and universities, community mental health centers, specialty clinics, online advertisements, previous studies, and local advocacy groups in the Pittsburgh region. Upon recruitment, participants were screened for eligibility by trained diagnosticians. Diagnostic interviews for ASD were administered using the ADOS by research staff in the Subject Assessment Core of the NIH-funded University of Pittsburgh Autism Center of Excellence, who were supervised by a study psychologist. Diagnostic interviews for healthy controls and outpatients with schizophrenia were carried out using the SCID by research staff who were trained and supervised by an expert diagnostician. After determining eligibility, participants were administered the aforementioned measures of neurocognition and social cognition by trained neuropsychological testers supervised by a study psychologist. For individuals participating in ongoing treatment studies of cognitive enhancement therapy, only cognitive data collected prior to initiating treatment in these studies were analyzed. Studies were reviewed and approved annually by the University of Pittsburgh Institutional Review Board, and all participants provided written informed consent prior to study participation.

2.4. Data analysis

Impairments in neurocognitive and social-cognitive domains among adults with ASD and schizophrenia relative to each other and to healthy volunteers were investigated using a series of general linear models. Primary models evaluated neurocognition and social cognition composite indexes, and secondary models examined the univariate components of these indexes when the overall composite suggested a significant difference between the study groups. All general linear models accounted for the potential confounding effects of group differences in age, gender, race, and IQ, and were adjusted for multiple pairwise group comparisons using Benjamini and Hochberg's (1995) correction. Substance use diagnoses were also considered as potential covariates in these models, but were collinear with group status, due to their exclusive presence in the schizophrenia group. Analyses conducted to examine potential differences in cognitive function among schizophrenia patients with and without a substance use diagnosis revealed no significant differences between these two groups in performance on any cognitive measure studied (mean $d = -.06$, range of $d = -.51$ to .32, all $p > .095$) and no significant relationships between a substance use diagnosis and composite neurocognitive ($\beta = -.15$, $p = .318$) or social-cognitive performance ($\beta = -.09$, $p = .476$), indicating that the presence of substance use problems in the schizophrenia sample was not likely to confound analyses of between-group differences in cognitive function.

3. Results

3.1. Impairments on composite indexes of social and non-social cognition

Investigation of overall performance differences on composite measures of neurocognition and social cognition between adults with ASD, schizophrenia, and healthy volunteers indicated that significant differences were observed in neurocognitive, $F(2, 107) = 26.75$, $p < .001$, and emotion processing social-cognitive function, $F(2, 107) = 7.51$, $p < .001$, between the study groups. Planned follow-up pairwise comparisons indicated that neurocognitive performance was greatly impaired in adult autism ($d = -1.71$, $p < .001$) and schizophrenia ($d = -1.48$, $p < .001$) participants compared to healthy controls, but that the magnitude of neurocognitive impairment was not significantly different between those with ASD and schizophrenia ($p = .710$). Similarly, both autism ($d = -.97$, $p < .001$) and schizophrenia ($d = -.65$, $p = .016$) participants also demonstrated significant impairments in emotion processing compared to healthy controls, but again no significant differences were observed in the magnitude of these impairments between the two patient groups ($p = .331$) (see Fig. 1).

Subsequent exploratory analyses revealed that while differences did exist in neurocognitive performance indicating greater ability in adults with ASD compared to schizophrenia when examining unadjusted models ($d = .90$, $p < .001$), adjusting for the discrepancy in IQ between these two groups was sufficient to reduce this between-group difference to non-significant levels ($d = .37$, $p = .083$). No significant differences in level of impairment on the social-cognitive composite were observed between ASD and schizophrenia participants in either unadjusted ($p = .053$) or IQ-adjusted ($p = .646$) analytic models. Further, moderator analyses examining the association between IQ and performance on neurocognitive and social-cognitive composites across the three study groups indicated that the contribution of IQ to performance on these domains was not significantly different between adults with autism, schizophrenia, and healthy volunteers, all $F(2, 105) < 2.56$, all $p > .08$.

3.2. Impairments across individual social and non-social cognitive domains

After finding significant and similar impairments on composite measures of neurocognitive and social-cognitive performance among adults with ASD and schizophrenia, analyses were then conducted on univariate components of these composite indexes to identify domains of greatest impairment and potential differences between those with autism and schizophrenia. As can be seen in Table 2, both patient groups demonstrated significant impairments relative to healthy individuals on nearly every neurocognitive domain assessed. The largest domain of neurocognitive impairment for both groups was speed of processing.

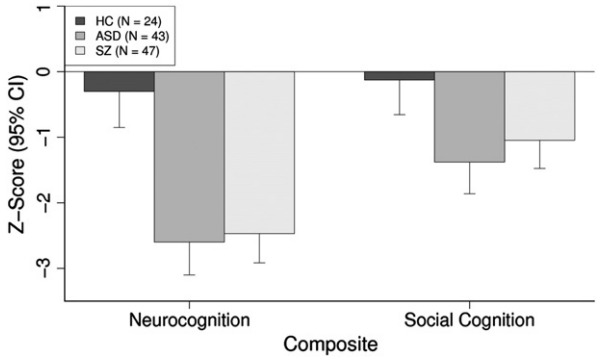

Fig. 1. Performance on composite indexes of neurocognition and social cognition among adults with autism, schizophrenia, and healthy individuals.

Table 2
Neurocognitive and social-cognitive performance across adults with autism, schizophrenia, and healthy individuals.

	Healthy (HC; $N = 24$)		Autism (ASD; $N = 43$)		Schizophrenia (SZ; $N = 47$)		Analysis[a]				
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	d_{ASD}	d_{SZ}	$d_{ASD\ vs.\ SZ}$	*p*	Direction
Neurocognition											
MCCB											
Speed of processing	79.04	19.15	34.92	31.63	32.80	28.35	−1.53	−1.48	−.06	<.001	HC > ASD, SZ
Attention/vigilance	52.29	27.89	36.01	32.06	27.03	24.14	−.57	−.81	−.25	.006	HC > ASD, SZ
Working memory	56.00	26.01	22.17	30.72	31.61	27.44	−1.29	−.86	.29	<.001	HC > ASD, SZ
Verbal learning	62.83	23.93	40.10	29.44	38.56	30.67	−.85	−.83	−.05	.001	HC > ASD, SZ
Visual learning	50.25	25.28	31.06	28.71	32.27	26.90	−.72	−.62	.04	.010	HC > ASD, SZ
Problem-solving	59.02	24.08	29.86	30.69	42.03	30.58	−1.05	−.56	.35	<.001	HC > ASD, SZ
WCST											
Perseverative errors	8.17	3.75	17.86	9.27	17.47	15.55	.84	.74	−.03	.002	HC < ASD, SZ
Non-perseverative errors	7.92	4.56	18.35	9.63	18.00	13.14	1.01	.89	−.03	<.001	HC < ASD, SZ
Social cognition											
MSCEIT	101.50	12.98	91.79	14.44	92.86	14.86	−.70	−.57	.06	.017	HC > ASD, SZ
Perceiving emotions	103.54	15.92	100.56	19.58	98.56	21.05	−.15	−.24	−.08	.635	–
Facilitating emotions	102.48	13.32	94.96	14.74	98.72	15.51	−.49	−.23	.20	.157	–
Understanding emotions	102.54	9.88	88.86	13.31	90.12	12.43	−1.17	−.98	.09	<.001	HC > ASD, SZ
Managing emotions	95.25	11.60	88.98	12.54	91.22	12.86	−.49	−.29	.14	.153	–
Penn emotion recognition test											
Total correct	34.25	2.40	30.77	4.14	32.44	4.34	−.84	−.40	.33	.005	HC > ASD
Reaction time (log)	7.55	.15	7.74	.22	7.67	.37	.73	.43	−.21	.019	HC < ASD

Note. Means are adjusted from general linear models accounting for age, gender, and IQ. The statistical significance of all pairwise comparisons is adjusted using Benjamini and Hochberg's (1995) correction.
MCCB = MATRICS consensus cognitive battery; MSCEIT = Mayer–Salovey–Caruso emotional intelligence test; and WCST = Wisconsin card sorting test.
[a] Omnibus *F*-test results for group differences from general linear models, two-tailed, adjusting for age, gender, race, and IQ.

No significant differences were found in the degree of neurocognitive deficit between autism and schizophrenia participants.

When examining the social-cognitive domains of emotion processing, areas affected were less uniform than basic cognitive processes. Overall emotional intelligence and emotional understanding as measured by the MSCEIT were significantly impaired in both adults with ASD and schizophrenia. Conversely, emotion recognition performance was only significantly impaired relative to healthy controls in the autism ($p = .004$), but not the schizophrenia group ($p = .126$), although the average performance advantage for those with autism versus schizophrenia was small ($d = .33$) and the two groups did not significantly differ in emotion recognition ability before ($p = .126$) or after correction for multiple inference testing ($p = .126$). The greatest domain of emotion processing impairment was emotional understanding in both autism and schizophrenia, and no significant differences in degree of impairment were found between adults with ASD and schizophrenia on any social-cognitive domain of emotion processing studied.

4. Discussion

Autism spectrum disorder and schizophrenia are distinct neurodevelopmental conditions that may share considerable impairments in social and non-social cognitive information processing (e.g., Sugranyes et al., 2011). This study examined neurocognitive and social-cognitive emotion processing impairments in carefully diagnosed samples of adults with ASD and schizophrenia, relative to each other and to healthy volunteers. Results revealed a high degree of similarity in cognitive impairment between those with ASD and symptomatically stable outpatients with schizophrenia, when compared to healthy individuals, with no significant differences observed between the two patient groups on any measure of social or non-social cognition. The largest domains of impairment shared between those affected by these conditions were speed of processing and emotion understanding, and when taken together, these findings indicate that large and similar impairments are present across diverse cognitive domains in verbal adults with autism and adults with schizophrenia.

It is interesting that while the sample of adults with autism studied was "higher functioning", as reflected by above-average levels of intelligence, impairments in non-social information processing domains were as large as adults with schizophrenia. In fact, even without adjusting for differences in IQ, adults with ASD continued to experience what would be considered large neurocognitive deficits ($d = -1.18$). After adjusting for IQ differences, neurocognitive differences between those with ASD and schizophrenia became negligible, and similar levels of impairment in emotion processing were observed between the two groups regardless of whether IQ was accounted for in analytic models. Such findings suggest that the greater verbal ability and intellectual capacity of high-functioning adults with ASD did not spare them from challenges in core domains of information processing. This dissociation may explain why clinicians, educators, and parents often over-estimate the understanding that adults with ASD have of information, events and people and underestimate their need for appropriate intervention. Overall, these findings indicate that interventions targeting broad social and non-social cognitive deficits may be needed for adults with ASD and adults with schizophrenia, and that comprehensive and integrated treatment approaches that target these domains, such as cognitive enhancement therapy (Hogarty et al., 2004), might offer the greatest opportunity for meaningful functional improvement, as we have demonstrated in schizophrenia (Hogarty et al., 2004) and preliminarily observed in ASD (Eack et al., in press).

Despite the pathophysiologic and treatment implications of this research, a number of important limitations should be noted. First, neurocognitive measures were taken primarily from a battery of cognitive tests specifically selected for patients with schizophrenia, and the MATRICS consensus cognitive battery has not been as thoroughly evaluated in adults with ASD, although this research suggests that it shows promise in this population. Second, intelligence levels could not be closely matched between the affected groups, in part due to the greater intellectual ability of higher-functioning adults with ASD. However, all final analytic models accounted for the effects of IQ, and while adults with ASD did display greater IQ scores on average as a sample, below-average intelligence scores (as low as 80) were present in individuals with autism and above-average intelligence scores (as high as 135) were observed in patients with schizophrenia. In addition, these findings may not be generalized to non-verbal adults with autism and comorbid intellectual disability. Third, substance use problems were uniquely present in the schizophrenia group, which could have affected differences observed with the healthy control group, although no significant differences on any of the cognitive

measures used in this study were observed between patients with schizophrenia with and without a substance use diagnosis. Fourth, the sample size of healthy volunteers was modest, which may have precluded detecting more modest deficits within each affected group, such as those observed in emotion perception in patients with schizophrenia. Finally, assessments of social cognition were limited to measures of emotion processing. Future research will need to cover a more diverse array of social-cognitive constructs.

In summary, this research indicates considerable similarity in social and non-social cognitive impairment in verbal adults with ASD and outpatients with schizophrenia, with particularly marked deficits in speed of processing and emotion understanding observed in both of these conditions. Intervention approaches that transcend diagnostic boundaries and integrate neurocognitive and social-cognitive remediation are needed to improve adaptive function and quality of life in these underserved populations.

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Contributors

This study was designed by Drs. Eack, Minshew, Newhill, Keshavan, and Phillips. Dr. Eack wrote the initial draft of the manuscript, Drs. Minshew, Newhill, Keshavan, Phillips, and Greenwald, along with Mrs. Hogarty, Ms. Bahorik, and Ms. McKnight provided critical revisions and feedback on both the manuscript and analyses. Ms. Bahorik and Ms. McKnight led cognitive data collection supervised by Dr. Greenwald. Dr. Minshew contributed to autism data collection and analysis, and Drs. Keshavan, Newhill, and Phillips contributed to schizophrenia data collection and analysis. Dr. Eack oversaw all data collection and analysis aspects of the study. All authors contributed to and have approved the final manuscript.

Conflict of interest

The authors report no conflicts of interest.

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