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Effects of Social Cognitive Impairment on Speech Disorder in Schizophrenia

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Disordered speech in schizophrenia impairs social functioning because it impedes communication with others. Treatment approaches targeting this symptom have been limited by an incomplete understanding of its causes. This study examined the process underpinnings of speech disorder, assessed in terms of communication failure. Contributions of impairments in 2 social cognitive abilities, emotion perception and theory of mind (ToM), to speech disorder were assessed in 63 patients with schizophrenia or schizoaffective disorder and 21 nonpsychiatric participants, after controlling for the effects of verbal intelligence and impairments in basic language-related neurocognitive abilities. After removal of the effects of the neurocognitive variables, impairments in emotion perception and ToM each explained additional variance in speech disorder in the patients but not the controls. The neurocognitive and social cognitive variables, taken together, explained 51% of the variance in speech disorder in the patients. Schizophrenic disordered speech may be less a concomitant of "positive" psychotic process than of illness-related limitations in neurocognitive and social cognitive functioning.

Key words: schizophrenia/speech disorder/language/social cognition/theory of mind/emotion perception

Introduction

Disordered speech is a symptom of schizophrenia that limits social functioning because it impairs the ability to communicate with others. Efforts to develop treatments targeting this symptom have been limited by an incomplete understanding of its causes or underpinnings. Researchers attempting to identify the processes underlying schizophrenic speech disorder have conceptualized and assessed it in several different ways, roughly categorizable as thought disorder, linguistic structural impairment, and communication failure perspectives. The thought disorder approach views speech disturbances as manifestations of disordered thought processes, and targets for measurement elements in speech that are believed to reflect elements of thought disorder such as associative loosening, illogicality, bizarre-idiosyncratic thinking, and poverty of thought (eg, refs. [1–3]). The linguistic perspective posits that disordered speech is the product of impairments in language-specific cognitive processes, ie, the ability to structure language, and it targets characteristics of language structure believed to reflect these deficiencies (eg, refs. [4–6]). The communication failure approach assesses speech disturbances pragmatically, in terms of how well or poorly meanings are conveyed rather than in terms of underlying thought disorder or linguistic structural breakdown (eg, refs. [7,8]). It measures the frequency of instances of unclear meaning, or unclear references broadly defined, in an individual's speech. Unclear references may reflect thought disorder and linguistic structuring deficits; but from the communication perspective, the focus of the assessment is on the extent to which the speech accomplishes its primary function, which is to communicate meaning. This approach has had several advantages in research, not the least of which is that speech disorder measured in this functional way generally has been more highly related to identifiable neurocognitive deficits than when measured in terms of signs of thought disorder or characteristics of linguistic structure. The frequency of unclear references has been related to impairments in certain basic neuropsychological functions, including sustained attention, verbal working memory, and sequencing ability.[9,10] These and related cognitive deficits have accounted for a significant proportion of the variance in frequency of communication failures.[10,11] However, much of the variance in communication failures is still unexplained.

Communication is an interpersonal process, and as such it requires certain social cognitive abilities. Many scholars of schizophrenic thought and language disturbance have noted that patients whose speech is disordered seem to be unaware of what the listener needs in order to understand the speech (eg, refs. [3,7,12]). This phenomenon has been described as a lack of awareness of the perspective of the listener. Two facets of social cognition that are impaired in schizophrenia, emotion perception (for review

see[13]) and theory of mind (ToM),[14,15] ie, comprehension of the thoughts, perceptions, and intentions of others, could contribute to such a lack of awareness and thus be implicated in communicative failures in speech. Although neurocognition may be defined very broadly to include all mental functions, in the present context, it is defined and operationalized more narrowly than that, in terms of intellectual functioning, attention, memory, organizational, and conceptual abilities. Impairments in these neurocognitive functions contribute to deficits in social cognition to some degree but do not fully explain them.[16,17] There is reason to believe that both types of impairments, neurocognitive and social cognitive, may contribute incrementally to the variance in communication failures in speech.

Previous studies have found relationships between performance on ToM tasks and severity of speech disorder,[18–22] but the associations have tended to be IQ related and could have reflected neurocognitive deficits, which do share some variance with social cognitive deficits. These studies did not assess the role of basic language-related neurocognitive impairments in the relationship between ToM and speech disorder. To our knowledge, there has been only one study that controlled for neurocognitive performance and then examined the effects of social cognitive impairment on speech disorder.[23] Neurocognitive test performance was found to be associated at moderate levels with speech disorder in that study, but social cognitive performance made little or no additional contribution beyond the effects of the neuropsychological tests. However, only one measure of social cognition was used, and it appears to have been somewhat limited in scope and to have had a restricted range. In addition, the measure of speech disorder, signs of "bizarre-idiosyncratic thinking" in proverb interpretations, may not have been optimal for the present purposes. As noted above, findings of associations between neurocognitive variables and speech disorder generally have been strongest when the speech disorder has been measured in terms of its functional effect, failure in the communication of meaning, rather than in terms of its underlying thought disorder.[10,24] Furthermore, one might expect social cognitive impairments in particular to be more directly relevant to communication failures than to formal thought disorder. Limitations in an individual's ability to interpret the emotions, thoughts, and intentions of others are likely to have a more direct impact on the ability to communicate clearly than on the degree to which the speech is bizarre and idiosyncratic.

The Present Study

The present study examined the unique, sequential, and combined contributions of verbal intelligence and neurocognitive functioning, emotion perception, and ToM to communication disturbances in the speech of schizophrenia patients, using several different measures of emotion perception and ToM. The main hypothesis was that deficits in the 2 social cognitive abilities would have additional negative impacts on patients' speech, beyond the effects of neurocognitive functioning. The same associations also were tested in a group of community control subjects to assess whether the associations were specific to schizophrenia or applicable more broadly.

Methods

Participants

Patients. Participants included 63 adult outpatients in treatment at a local public mental health clinic, who were assessed as part of a large ongoing research project.[25] The present cohort consisted of consecutive enlistees to the study who had *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) diagnoses of schizophrenia (n = 33) or schizoaffective disorder (n = 30), as determined by diagnostic interview and clinic records. All had been outpatients for at least the preceding 3 months and were considered by their clinicians to be clinically stable. Volunteers who met DSM-IV criteria[26] for current (past year) substance abuse or dependence were excluded, as were those with histories of head injury resulting in prolonged loss of consciousness, seizure disorder, mental retardation, alcohol dependence requiring detoxification, or inhalant abuse. Patients who were unable to comprehend the purpose or procedures of the study were not enrolled. Individuals whose primary language was not English also were excluded because this was a study of verbal communication. The resulting patient sample included individuals with a range of symptom severity, from the virtually asymptomatic to the actively psychotic, and a range in level of functioning. Some were gainfully employed, but the majority were supported by Social Security, many with payees to oversee the management of their money. Most lived alone and unsupervised in subsidized housing; others lived in group homes, nursing homes, or other supervised settings.

Controls. Control participants included 21 volunteers roughly matched to the patients on gender, race/ethnicity, and parent educational attainment. They were recruited by means of flyers to university support staff and signs posted in libraries and other community facilities. The same exclusion criteria were applied to controls as to patients. Individuals also were excluded from the control group if they endorsed a history of any psychotic symptoms. We did not exclude controls for any other reasons (eg, depression, anxiety, family history) because we wanted them to be as comparable to the patients as possible on variables other than schizophrenia.

All patient and control volunteers were paid for their participation. Participant descriptive information is provided in table 1.

Table 1. Participants

	Patients	Controls	Chi-Square	*t*	*P*
Number	63	21	—	—	—
Age	40 (SD = 8)	38 (SD = 9)	—	1.04	<.30
Male/female	42/21	10/11	0.46	—	<.50
African American/Caucasian/Other	39/22/2	12/9/0	1.37	—	<.30
Education (years)	12 (SD = 2)	15 (SD = 2)	—	−7.17	<.00
Parent education (years)	12 (SD = 3)	12 (SD = 1)	—	0.67	<.50
Global assessment of functioning	48 (SD = 14)	83 (SD = 9)	—	−9.20	<.00
Patient symptom severities					
PANSS positive symptoms	*M* = 18; SD = 6; range = 7(none)–34(moderate/severe)				
PANSS negative symptoms	*M* = 15; SD = 5; range = 8(none)–31(moderate/severe)				
PANSS general symptoms	*M* = 34; SD = 10; range = 16(none)–59(mild/moderate)				
PANSS total symptoms	*M* = 67; SD = 17; range = 34(none)–113(mild/moderate)				

Note: PANSS, Positive and Negative Symptom Scales.

Measures

Diagnosis and Symptoms. The Schedule for Affective Disorders and Schizophrenia diagnostic interview,[27] adapted slightly for use with DSM-IV criteria, was administered to all participants by graduate assistants trained in the measure. Diagnoses were determined by the first author, in consultation with the graduate assistants, using information from the interview and clinic records. Symptoms were rated for severity using the Positive and Negative Syndrome Scales (PANSS).[28]

Neurocognitive Tests. Premorbid verbal intelligence was estimated using the vocabulary test from the Shipley Institute of Living Scale, Part I.[29,30] Sustained attention was measured using the Continuous Performance Test—Identical Pairs (CPT-IP),[31,32] which requires participants to respond when 2 identical stimuli occur in a row. The stimuli are 2-, 3-, and 4-digit numbers. This test assesses attention and has a working memory component. The score used in the present study was the mean *d*-prime across the 2-, 3-, and 4-digit conditions. *D*-prime is a measure of sensitivity that takes into account both hits and false alarms. Working memory was assessed using the Digit Span test, forward, and backward.[33] Sequencing ability was measured as time to completion of the Trails B task,[34] which requires "follow the dots" type connecting of numbers and letters in alternating order. Conceptual sequencing was assessed with the Shipley Test, Part II (conceptualization subscale).[29,30] This test consists of partial sequences of numbers and letters, each sequence based on a different concept. The test requires subjects to deduce each concept and then generate additional numbers or letters to complete the sequence.

Social Cognitive Tests. Two facets of social cognition were assessed: emotion perception and ToM. Three measures of emotion perception were used. The first was an Eckman test,[35] in which 35 pictures of emotional faces were presented, and subjects were asked to identify the emotions being expressed, choosing from a list of 7 emotions. The second was the Bell-Lysaker Emotion Recognition Test (BLERT),[36] which consists of 21 audiovisual clips of a man making emotional statements. Each series of statements has the same verbal content but with variations in prosody, facial expression, and body language. After each clip, subjects choose the emotion being expressed from the same list of 7 possibilities as in the Eckman test. The third was the Profile of Nonverbal Sensitivity (PONS) test.[37] The short version (half-PONS) was used. The half-PONS consists of a series of 110 brief, 2-second video clips of a female actor moving and/or speaking. Scenes may include body movements, facial expressions, or content-filtered or spliced audio, or combinations of these. After each clip, the participant is asked to choose the answer (of 2) that most appropriately describes the activity depicted (eg, helping a lost child vs admiring the beauty of nature). Number of items correct was the score used for each of these 3 tests.

ToM was assessed using 2 measures. The first was Corcoran et al's Hinting Task,[38] which consists of 10 short written vignettes, each of which ends with one character dropping a hint to another character. Each vignette is given to the subject and also read out loud. The subject is asked what the character really means. A correct inference is given a score of 2 points. If no inference is offered, a second, more obvious hint is added, and the subject is again asked for the inference. A correct answer at this point receives a score of 1 point. The measure is British. We adapted it slightly by Americanizing the language. The second ToM measure administered was a test created by Sarfati and colleagues[39] that involves completing a set of 28 ToM cartoon stories. For each story, 3 pictures in sequence are presented depicting a character doing something purposeful. The subject is given 3 "ending" cards and is asked to select the correct one to finish the story.

A correct response requires that the subject make an accurate inference regarding the intentions of the character from his/her behavior in the previous pictures.

Speech Disorder. Ten-minute conversational speech samples were collected from each participant, on the topics of their self-perceptions, interests, and daily activities. The speech samples were audiorecorded and later transcribed for rating. Interviewers encouraged participants to do most of the talking but prompted them with comments or questions as needed to keep them talking and on topic and to steer them away from emotionally laden topics. Speech disorder was rated using the Communication Disturbances Index (CDI).[8] The CDI assesses references in speech, broadly defined, and is based entirely on failures in the transmission of meaning. Instances of unclear meaning are identified in the speech sample. These include structural and nonstructural failures. The structural or interclausal unclear references include: (1) references for which the intended referent is either ambiguous (2) or entirely missing; (3) words or phrases that are unclear because they have more than one possible definitional meaning, and the correct choice is not clear from the context; and (4) segments with unclear meaning due to a breakdown in grammar. The nonstructural or intraclausal unclarities include (1) words or phrases that are overly vague and (2) wrong word usage, in which the meaning is unclear because of a seemingly incorrect word choice. Lack of clarity of meaning is always the criterion. Vague words, instances of poor grammar, and so on, are only counted if they impair the conveyance of meaning. Instances of unclarity are counted, and the sum divided by number of hundred words (no. words/100) in the speech sample, to yield a frequency count. The CDI is described in more detail, and examples of each type of communication failure are provided in its original validation article.[8] Nonpsychiatric individuals make some unclear references, but schizophrenia patients as a group make them with much higher frequencies.[40] In patient samples, CDI ratings have correlated significantly with ratings of formal thought disorder[40] and linguistic structural breakdown.[24] CDI ratings have also been correlated with measures of attention, working memory, and sequencing. These latter correlations have been specific to the structural, or interclausal, types of unclear reference. In the present study, we used the total of all referential failures, structural and nonstructural, because we expected unclear references of all kinds to be more frequent in the speech of patients with social cognitive impairments that affect the ability to understand the perspective and responses of a conversational partner. In the present study, the ratings were done using both the transcripts and the audiorecordings. The rater attained good reliability with a second rater on a separate set of speech samples prior to completing the ratings for the present study, intraclass correlation (ICC) = .94.

Procedure

Participants were assessed in 3 sessions, each 1-week apart. Informed consent procedures, a diagnostic interview, symptom ratings, and collection of a speech sample were done in the first session, neurocognitive tests administered in the second session, and social cognitive measures in the third. All the patients completed all the measures. Two of the tests (CPT-IP and BLERT) were added to the protocol for control participants after data collection had started, so only 12 control participants completed those measures. The Hinting Test was not administered to controls because pilot testing of the measure showed a marked ceiling effect in controls.

Analysis

Patients were compared with controls on all the measures using *t* tests. Next, correlations of neurocognitive and social cognitive test scores with CDI ratings were computed in each group. Third, a regression was computed to test the sequential contributions of verbal intelligence and neurocognitive impairments, emotion perception, and ToM deficits to the variance in communication disturbances in the speech of the patients. A similar regression was computed with the control participant data, to test whether associations would be similar or different in the 2 groups. All tests of significance were 2-tailed.

Results

The CDI ratings were positively skewed, so they were log-transformed for the analyses. The distributions of all the other measures met assumptions of normality. The schizophrenia patients differed from the schizoaffective patients in having significantly higher CDI ratings (M (SD) = 2.18 (1.28) and 1.58 (0.73), respectively, t (61) = 2.27, $P < .03$) and worse performance on the CPT-IP (M (SD) = 5.08 (2.16) and 6.26 (2.11), respectively, t (61) = −2.18, $P < .04$) and the Hinting Task (M (SD) = 14.21 (4.37) and 16.87 (2.13), respectively, t (61) = −3.11, $P < .01$). They did not differ on any of the other neurocognitive or social cognitive measures. Because the differences were few and not large, the 2 patient groups were combined for the main analyses; however, a secondary analysis also was computed with only the schizophrenia patients.

Patients vs Controls

Means and SDs for all the measures are presented in table 2. Comparisons between patients and controls also are presented in table 2. The speech of the patients contained much more frequent instances of unclarity than the speech of the controls. Patients scored significantly worse than controls on all the neurocognitive measures except the digit span test ($P = .11$) and on all the social cognitive measures except the Sarfati test, on which there was a difference at the trend level ($P < .06$).

Table 2. Speech, Neurocognitive, and Social Cognitive Variables: Patients vs Controls

Measure	*N*, Patients/controls	Patients		Controls			
		M	SD	*M*	SD	*t*	*P*
CDI total	63/21	1.89	1.09	0.61	0.32	8.39	.00
Shipley-Part I	63/21	24.11	5.84	29.95	3.75	−5.38	.00
Shipley-Part II	63/21	16.95	8.59	27.55	7.25	−5.17	.00
CPT-IP, *d*-prime	63/12	5.64	2.20	9.79	1.92	−.86	.00
Digit span total	63/21	12.89	3.13	14.95	5.44	−1.69	.11
Trails B time (s)	63/21	119.37	60.30	85.57	43.03	2.42	.02
Ekman test	63/21	23.14	4.18	25.64	3.36	−2.52	.02
BLERT	63/12	13.51	3.70	17.42	2.23	−3.53	.00
Half-PONS	63/21	72.76	11.43	79.95	8.10	−2.66	.01
Hinting test	63/0	15.48	3.71	—	—	—	—
Sarfati ToM test	63/21	20.80	4.97	23.23	4.86	−1.97	.06

Note: CDI, Communication Disturbances Index; CPT-IP, Continuous Performance Test—Identical Pairs; BLERT, Bell-Lysaker Emotion Recognition Test; PONS, Profile of Nonverbal Sensitivity; ToM, theory of mind.

Psychotic Symptoms and Speech Disorder in Patients

Associations between psychotic symptoms and speech disorder were examined. Severity of delusions (per the PANSS) was correlated with CDI ratings at a low nonsignificant level, $r = .13$; severity of hallucinations was correlated at a modest but significant level with CDI ratings, $r = .33$, $P < .01$.

Neurocognitive and Social Cognitive Contributors to Speech Disorder in Patients

In the patient group, CDI ratings were associated with premorbid verbal functioning, as measured by the Shipley vocabulary test, and with most of the measures of attention, working memory, and sequencing. They also were associated with the measures of emotion perception and ToM. These correlations are presented in table 3.

A regression analysis examined the unique and combined effects of neurocognitive functioning, emotion perception, and ToM on patients' speech. The dependent variable was the CDI ratings. In the first step, verbal intelligence scores (Shipley-Part I) and the other neurocognitive test scores (CPT-IP, Digit Span, Trails B, and Shipley-Part II) were entered as a block. This step was significant, *R*-square = .407, $P = .000$. Second, the emotion perception measures (Ekman test, BLERT, and Half-

Table 3. Pearson Correlations of Cognitive and Social Cognitive Measures With Communication Failure Ratings in Patients and Controls

Measure	*N*, Patients/controls	Communication Disturbance Ratings			
		Patients		Controls	
		r	*P*	*r*	*P*
Premorbid verbal intelligence					
Shipley-Part I	63/21	**−.36**	.01	−.25	.28
Neurocognition					
Shipley-Part II	63/21	**−.58**	.00	**−.46**	.04
CPT-IP, *d*-prime	63/12	**−.32**	.01	.41	.21
Digit span total	63/21	−.15	.25	.30	.18
Trails B time (reversed)	63/21	**−.35**	.01	**−.52**	.02
Social cognition					
Ekman test	63/21	−.40	.00	−.14	.54
BLERT	63/12	**−.46**	.00	.01	.97
Half-PONS	63/21	**−.42**	.00	−.11	.62
Hinting test	63/0	**−.46**	.00	—	—
Sarfati ToM test	63/21	**−.32**	.01	**−.47**	.04

Note: Abbreviations are explained in the first footnote to table 2. Statistically significant values are in bold type.

Table 4. Regression of Neurocognitive, Emotion Perception, and ToM Test Performance on Communication Disturbances in Speech

Steps	*R*	*R*-Square	*R*-Square Change	*F*-Change	Significance of *F* Change
(a) 63 patients with schizophrenia or schizoaffective disorder					
1. Neurocognitive tests	.638	.407	.407	7.545	.000
2. Emotion perception tests	.728	.530	.123	4.437	.007
3. ToM tests	.768	.590	.060	3.684	.032
(b) 33 patients with schizophrenia					
1. Neurocognitive tests	.709	.503	.503	5.268	.002
2. Emotion perception tests	.794	.631	.127	2.645	.073
3. ToM tests	.874	.764	.133	5.913	.009
(c) 21 nonpsychiatric controls					
1. Neurocognitive tests	.747	.559	.559	5.061	.008
2. Emotion perception tests	.753	.567	.008	0.130	.879
3. ToM tests	.753	.567	.000	0.001	.980

Note: ToM, theory of mind; CPT, Continuous Performance Test. Step 1: Shipley Vocabulary, Shipley Abstraction, CPT—Identical Pairs, Trails B, and Digit Span. Step 2: Eckman Faces, Bell-Lysaker Emotion Recognition Test, and Profile of Nonverbal Sensitivity (half). Step 3: Sarfati Test and Hinting Test.

PONS) were entered as a block, to test whether they would contribute further to speech disorder beyond the effects of the neurocognitive variables. This step made a significant contribution, *R*-square change = .123, $P = .007$. In the third and final step, the ToM measures (Sarfati and Hinting Test) were entered. This step also added significantly to the equation, *R*-square change = .06, $P = .032$. To summarize, all 3 sets of variables contributed significant variance to communication failures, and together, they explained 51% of the variance in patients' CDI ratings. These findings are presented in table 4a.

When schizoaffective patients were removed from the analysis and the above regression repeated with the data from the schizophrenia patients only ($n = 33$), the associations were even stronger, see table 4b. Together, the variables explained 65% of the variance in CDI ratings.

Neurocognitive and Social Cognitive Contributors to Communicative Clarity in Controls' Speech

Similar analyses were conducted with the CDI ratings of the control participants. In the correlational analysis, CDI ratings were associated with performance on the Shipley-Part II, Trails B, and Sarfati tests but not with any of the other measures. The control group was smaller than the patient group, making statistical significance less likely; however, the majority of the correlations in the control group tended to be small as well as nonsignificant. These results are presented in table 3.

A regression was computed using the same procedure and steps as for the patients, except that the CPT-IP, BLERT, and Hinting Test were omitted because they had not been administered to all the control participants. In step 1, the vocabulary and neurocognitive test scores made a large contribution to the variance in CDI ratings, *R*-square = .559, $P < .008$. The emotion perception and ToM measures entered in steps 2 and 3 did not make contributions beyond the effects of the neurocognitive variables. These results are presented in table 4c.

Discussion

Impairments in emotion perception and ToM contributed to the frequency of communication failures in the speech of this sample of stable outpatients with schizophrenia and schizoaffective disorder. These effects were significant even after removal of the effects of verbal intelligence, attention, working memory, and sequencing ability. Furthermore, the effects of the 2 social cognitive variables were cumulative: Emotion perception performance added to the variance in communication failures, and ToM performance added on top of that. The neurocognitive and social cognitive impairments, taken together, explained a great deal of the variance in speech disorder in the patients. These strong associations are especially notable because multiple domains and measures of social cognition exist, and only a sampling was used in the present study. Other facets of social cognition might conceivably explain addition variance.

Social cognition was related to neurocognitive test performance in this study, but not very highly (*r*'s = .02 to .50, median $r = .28$). Of course, the test battery did not assess all facets of neurocognition or social cognition, and there may be facets that are more highly related to each other than the ones assessed here. However, our results are consistent with findings of previous studies using a variety of neurocognitive and social cognitive measures.[16,17] If social cognitive impairments are not largely the results of neurocognitive deficits, then what does cause them? They are impairments

relevant to interpersonal, or relational, functioning. Disordered relatedness has been described as a primary, quasi-independent dimension of schizophrenia,[41] as well as one of the most enduring deficits associated with the disorder. The inability to read others' emotions accurately and to interpret their beliefs and intentions appear to be either products or concomitants of schizophrenic disordered relatedness. Perhaps they are manifestations of what Bleuler[42] termed "autism" in his description of the basic pathologies of schizophrenia, a preoccupation with the self and self-generated material that results in a lack of awareness of and connectedness to others. The findings of the present study suggest that communication failures in the speech of schizophrenia patients may be a natural consequence of illness-related limitations in neurocognitive and relational functioning. CDI ratings were not associated significantly with severity of delusions and only modestly with hallucinations. Given the large proportion of variance accounted for by the cognitive and social cognitive measures, this kind of disorder in speech may actually be more highly related to neurocognitive and relational deficits than to "positive" psychotic process.

This study examined some of the process underpinnings of a schizophrenic symptom by correlating severity of the symptom, in this case speech disorder, with severity of process impairments, in this case neurocognitive and social cognitive deficits. Most of the participants in a patient group such as this have cognitive and social cognitive impairments and some level of speech disorder. The distributions of these variables are limited in such a sample, which diminishes the potential for finding large effects. To illustrate the point, in a (hypothetical) community sample including 63 schizophrenic and 6300 nonschizophrenic participants, which is roughly reflective of the distribution of schizophrenia in the general population, the associations among these same variables would almost certainly be considerably stronger; the impairments would cluster together more than they do in a sample of patients only. The limitations inherent in examining associations between process impairments and symptoms in a patient sample make it unlikely that the full strength of the real-life associations among the processes will be identifiable, even if strong relationships exist. Given this methodological limitation, the findings of the present study are quite notable.

Some of the tests (Shipley Part II, Trails B, and Sarfati) were related to speech disorder in the controls as well as the patients. Such findings are unusual in the literature, at least at this size and level of significance. One difficulty is an issue of scaling. Although the distributions of scores on cognitive and social cognitive tests may meet assumptions of normality within each group, ranges in scores are likely to be wider in patients than controls. This was the case in the present study. The relatively modest ranges of scores in controls probably made correlations more difficult to detect in this group. This makes the control findings especially notable. The presence of stronger associations than usual in the present study may be attributable to the measure of speech disorder used, which captures subtle as well as severe disturbances. The speech of nonpsychiatric participants rarely shows much evidence of formal thought disorder or linguistic failure, yet has a range in communicative efficacy that is captured by the CDI. As noted in the introduction, associations between cognitive test performance and speech disorder in patients have generally been relatively modest when the speech disorder has been assessed using measures of formal thought disorder or linguistic structural breakdown, compared with studies in which the speech disorder has been measured in terms of communication failure.[10,11,24] This may apply to the speech of nonpsychiatric individuals as well. In any case, the finding that communication failures in the speech of patients and controls share correlates supports the idea that schizophrenic speech disorder is at least in part the natural result of compromised cognitive functioning.

The ability to communicate is important to effective social functioning. Few if any interventions have been developed so far that have been demonstrated to improve patients' ability to communicate clearly. Cognitive remediation therapies have demonstrated some success in improving patients' attention and working memory (eg, refs. [43–45]), but to our knowledge, such interventions have not yet had much impact on speech disorder. The cognitive gains made in these programs may not be large enough to translate into improvements in speech disorder. Training in social cognition may be another method to try. Several social cognitive interventions have been developed in recent years (eg, refs. [45,46]), and some have been successful in improving their targeted capacities, although none to our knowledge have reported or even assessed for improvements in verbal communicative ability. Possibly, an effective program could be developed using a combination of cognitive remediation, social cognitive training, and individualized behavioral interventions (eg, ref. [47]) targeting the specific types of communication failures that occur most frequently in an individual's speech.

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