Social Cognitive Impairments and Psychotic Symptoms: What Is the Nature of Their Association?

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Social cognitive deficits are associated with psychotic symptoms, but the nature of this association remains unknown. This study uses a genetically sensitive cross-trait cross-sibling design to investigate the nature of the overlap between both phenotypes. A sample of 1032 patients, 1017 of their healthy siblings, and 579 control subjects were recruited within the Dutch Genetic Risk and Outcome in Psychosis (GROUP) study. Participants completed a battery of cognitive tests, including 2 social cognitive tests on theory of mind (ToM) and emotion recognition. Within siblings, symptoms were assessed with the Structured Interview for Schizotypy—Revised. The Positive and Negative Syndrome Scale was used to assess patients' symptoms. Within patients, social cognitive performance was consistently and significantly associated with disorganized and, to a lesser degree, with negative symptoms. Associations with positive symptoms were significant, but smaller. Suggestive of a shared etiology, both social cognitive factors showed significant familial clustering. The associations between patients' ToM and subclinical symptoms in siblings were nonsignificant, suggesting that their overlap within patients is due to individual rather than shared familial factors. Indicative of a shared etiology, familial covariation was present between patients' emotion recognition ability and disorganized and, albeit to a lesser degree, positive but not negative subclinical symptoms in siblings.

Key words: schizophrenia/social cognition/family study/cross-sibling design

Introduction

Schizophrenia is accompanied by significant functional impairment in different social cognitive domains.[1,2] The impairments are associated with psychotic symp-toms.[3–5] This association may reflect a shared etiopathology with a possible genetic basis. Alternatively, both traits may be on a causal pathway such that the psychotic symptoms are the consequence of impaired social cognition (or vice versa) or both traits may be secondary to another, possibly disease-related factor (eg, general cognitive impairment). These explanations are not mutually exclusive, and the present study set out to investigate the evidence for a shared familiar etiology. If symptoms and social cognition covary because of a similar familiar etiology, social cognitive abnormalities could be useful intermediate phenotypes in the search for the genetic causes of the symptoms of psychosis.

There is some evidence in favor of a genetic etiology of the social cognitive impairment in psychosis. For example, higher rates of social cognitive impairment have been reported in first-degree relatives as compared to the general population.[6–10] However, higher rates of subclinical symptoms in relatives and familial clustering of symptoms have also been reported.[11] There is evidence that in nonclinical individuals higher positive schizotypy is associated with worse theory of mind (ToM) performance.[12] Other studies suggested that associations between social cognitive performance and symptoms are also present during prodromal states and remission of the illness.[13,14] Studies to date have not been able to rule out alternative explanations for the symptom-cognition association, for example that the presence of social cognitive deficits is secondary to subclinical or residual psychotic experiences. Therefore, the current study applied a genetically sensitive cross-trait cross-sibling design to investigate the nature of the association between social cognitive abnormalities and psychotic symptoms.

Social cognition is a multidimensional construct,[15] and the nature of the associations between symptoms and

social cognition may differ across social cognitive domains.[16] Here, we focused on 2 core domains of social cognition that are impaired in schizophrenia and have previously been suggested to play a role in the formation of psychotic symptoms: (1) ToM and (2) emotion processing.[7,16] We used a cross-trait cross-sibling design (see figure 1) in a large sample of patients with nonaffective psychosis, their unaffected siblings and healthy controls to investigate social cognitive impairment and psychotic symptoms and the nature of their association. First, the associations between the different social cognitive functions and symptom clusters were analyzed within patients and their relatives to confirm the assumption of overlap between the 2 domains. Second, familial clustering of social cognitive functioning was investigated using within-trait cross-sibling analyses. Finally, we investigated all cross-trait cross-sibling associations between subclinical symptomatic expression in siblings and social cognitive functioning in patients. The presence of such associations suggests a common familial etiology of both traits. Alternatively, finding symptom-cognition associations within affected individuals only suggests that the frequently reported overlap between symptoms and social cognitive deficits is due to individual (eg, illness related) factors rather than shared familial etiology.

Method

Procedure and Sample

The data pertain to baseline measures of the ongoing longitudinal multicenter study "Genetic Risk and Outcome in Psychosis" (GROUP). The sample was recruited in the Netherlands and Belgium. Participants with nonaffective psychosis were identified via clinicians working in regional psychotic disorder mental health services. Family members were recruited through participating patients. Healthy volunteers were recruited through random community mailings in the catchment area. The full GROUP sample consisted of 1120 patients with a nonaffective psychotic disorder, 1057 of their siblings, 919 of their parents, and 590 unrelated controls from the general population. The current inclusion criteria were (1) age between 16 and 60, (2) good command of the Dutch language, and (3) being able and willing to give informed consent. Patients had to meet the DSM-IV-TR[17] criteria for a nonaffective psychotic disorder, as assessed by the Comprehensive Assessment of Symptoms and History Interview.[18] Additional inclusion criteria for the control group were not having a (1) lifetime psychotic disorder and/or (2) a first-degree family member with a lifetime psychotic disorder. The project was approved by the local Research Ethics Committee, and all participants gave written informed consent in accordance with the committee's guidelines.

Measures of Social Cognition

Degraded Facial Affect Recognition Task. The Degraded Facial Affect Recognition Task (DFAR)[19] uses photographs of 4 different actors (2 male and 2 female) depicting the 4 emotions: angry, happy, fearful, and neutral. The task comprises 64 trials consisting of 16 face presentations in each emotion category. The emotions were shown with 100% and 75% intensity in order to increase the difficulty of the task. Subjects were asked to indicate the emotional expression of each face with a button press and to respond as accurately as possible. Outcomes were the proportion correctly recognized as neutral, happy, fearful, and angry emotions and the overall proportion correct.

Hinting Task. ToM was assessed with the Hinting Task (HT).[6,9,20] The task tests the ability of subjects to infer the real intentions behind indirect speech utterances. It comprises 10 short passages presenting an interaction between 2 characters that end with 1 of the characters dropping a hint. The subject is then asked what the character really meant. Correctly identified hints are scored with 2 points. In cases of an incorrect response, a more obvious hint is added. A subsequent correct response is scored with 1 point; an incorrect response is scored as 0. The outcome range is 0–20.

Measures of Neurocognition

Benton Facial Recognition Test. The short form of the Benton Facial Recognition Test,[21] a measure of the ability to match unfamiliar faces, was used to assess whether deficits in facial affect recognition are not mediated by differences in general facial-recognition ability.

Wechsler Adult Intelligence Scale. The Arithmetic, Digit Symbol-Coding, Block Design, and Information subtests of the Wechsler Adult Intelligence Scale III were administered as an indicator of IQ.[22,23]
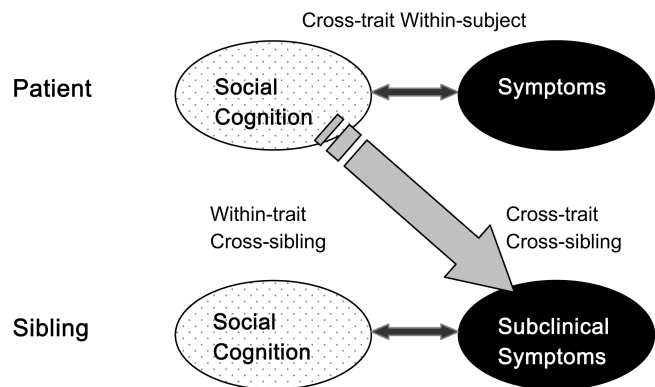

Fig. 1. Cross-trait Cross-sibling design.

Symptom Assessment

The Positive and Negative Syndrome Scale. The Positive and Negative Syndrome Scale (PANSS[24]) has been used to assess symptoms in patients. Originally, the PANSS consisted of a positive and negative syndrome scale and a general psychopathology scale. However, a specific model has been formulated on the social cognitive basis of disorganized symptoms[25] and research to date shows the most robust association between poor mental state attribution and disorganization symptoms.[26,27] Recently, Van der Gaag and colleagues[28] developed a more fine-grained model of symptoms. Among other factors, the model captures disorganized symptoms. The positive, negative, and disorganized symptom factors of the model were used in the current analyses.

Structured Interview for Schizotypy—Revised. The Structured Interview for Schizotypy—Revised (SIS-R)[29,30] was administered to assess subclinical symptoms in siblings. It is a semistructured interview that contains 20 schizotypal symptoms and 11 schizotypal signs. Guided by our a-priori theoretical considerations for the classification of symptoms within patients and previous research,[25] we reduced the item scores to the 3 dimensions of (1) positive (referential thinking, magical ideation, illusions, and suspiciousness), (2) negative (social isolation, social anxiety, introversion, and restricted affect), and (3) disorganization schizotypy (goal directness of thinking, loosening of associations, and oddness).

Analyses

Statistical analyses were performed using STATA version 11.0 statistical software. Linear regression analyses were used to investigate group differences and within-group symptom-cognition associations within siblings and patients. It has been argued that social cognitive impairment in schizophrenia is nonspecific and that any association with symptoms may be due to confounding by neurocognitive impairment.[31,32] We therefore adjusted all between group analyses for IQ (for intercorrelations between IQ and the social cognitive tasks see table 1). The potentially confounding factors age and gender were controlled in all between group analyses. Between group linear regression analyses on DFAR performance were also adjusted for general face recognition ability. Some families contributed more than 1 patient or sibling. All possible patient-sibling pairs were included in the analysis. To account for the observations of multiple siblings within 1 family, multilevel random regression analyses (XTREG) were used to analyze within-trait cross-sibling associations, ie, the familial clustering of cognitive performance. Cross-trait cross-sibling associations between cognitive performance in patients and subclinical symptoms in siblings were analyzed using the same routine. All cross-trait cross-sibling analyses were adjusted for the corresponding trait within the patient and sibling group. Effect sizes are expressed as the standardized regression coefficient β for linear regression analyses and the regression coefficient *b* with the 95% CI's.

Results

Sample

The current study incorporated a subset of participants from the full GROUP sample. This subsample included 1032 patients, 1017 of their healthy siblings, and 579 controls. Sample characteristics and test statistics are displayed in table 2.

Social Cognition. Patients had a worse HT performance than controls and siblings. The means of controls and siblings differed into the expected direction but this difference was not significant. Patients also performed worse on the DFAR than controls and siblings. Again, the performance of controls and siblings differed into the expected direction but was not significant. All groups recognized happy emotion best, followed by neutral, angry, and fearful emotion with the lowest rate of correct recognitions. Analyses per emotion category showed no differences between controls and siblings for any of the 4 categories. Patients did not perform worse than controls and siblings in recognizing neutral and happy emotions. They did, however, perform significantly worse than controls and siblings with respect to angry and fearful emotions (see table 3). All analyses were controlled for age, gender, and IQ. DFAR analyses were also controlled for general face recognition ability.

Cross-Trait Within-Group Analyses

Within patients, HT performance was consistently and significantly associated with disorganized and, to a lesser extent, with negative symptoms. The association with positive symptoms was smaller but also significant. DFAR performance was significantly associated with disorganized and, to a lesser extent, with negative and positive symptoms. Within siblings, HT performance was weakly but significantly associated with subclinical disorganized symptoms only. The associations

Table 1. Intercorrelations Between the (Social) Cognitive Tasks by Group

	Overall		Controls		Siblings		Patients	
	HT	DFAR	HT	DFAR	HT	DFAR	HT	DFAR
DFAR	.23		.12		.10		.25	
IQ	.35	.21	.17	.09	.23	.13	.37	.22

Note: DFAR, Degraded Facial Affect Recognition; HT, Hinting Task.

Table 2. Demographic and Clinical Sample Characteristics

Variable	Controls $N = 579$ Mean (SD)	n	Siblings $N = 1017$ Mean (SD)	n	Patients $N = 1032$ Mean (SD)	n	Test Statistic	P Value
Age (y)	30.4 (10.6)[a]	579	27.8 (8.2)[b]	1017	27.3 (7.2)[c]	1032	$F_{(2, 2624)} = 26.84$	<.001
Male (%)	45[a]		46[a]		77[b]		$\chi^2(2) = 257.53$	<.001
Education (%)							$\chi^2(16) = 246.54$	<.001
None/primary only	2.4	576	7.4	994	13	1008		
Lower secondary	14.8		19.7		30.7			
Lower vocational	15.6		22.4		17.4			
Higher secondary	31.7		20.5		25.5			
Higher vocational	25.5		18.5		9.1			
University	10		11.5		4.3			
IQ	109.7 (15.1)[a]	579	102.7 (15.6)[b]	1017	94.9 (16)[c]	1032	$F_{(4, 2623)} = 95.40$	<.001
HT (range 0–20)	19.1 (1.3)[a]	573	18.8 (1.7)[a]	1009	17.5 (2.8)[b]	1008	$F_{(5, 2584)} = 114.25$	<.001
DFAR total	73.2 (9.1)[a]	542	72.5 (9.4)[a]	943	68.6 (10.7)[b]	934	$F_{(6, 2398)} = 63.61$	<.001
Neutral	81.3 (15.1)		80.4 (15)		77.8 (17.5)		$F_{(6, 2398)} = 17.41$	<.001
Happy	87.4 (11.1)		88.2 (10.7)		86.8 (12.7)		$F_{(6, 2398)} = 14.17$	<.001
Angry	70.5 (18.6)		68.8 (19.3)		62.3 (20.9)		$F_{(6, 2398)} = 30.61$	<.001
Fearful	53.9 (18.1)		52.7 (19.7)		47.5 (19.7)		$F_{(6, 2398)} = 34.17$	<.001
SIS-R								
Disorganized	.03 (.12)[a]	571	.05 (.19)[b]	1006			$F_{(4, 1573)} = 6.78$	<.001
Negative	.45 (.43)[a*]	571	.5 (.47)[b]	1006			$F_{(4, 1574)} = 1.91$	.005
Positive	.31 (.35)[a]	571	.38 (.42)[b]	1006			$F_{(4, 1574)} = 14.72$	<.001
PANSS								
Disorganized					16.7 (6.2)	971		
Negative					15 (6.6)	970		
Positive					13.9 (6.6)	979		

Note: Different superscripts indicate significant group differences with $P < .05$. Group differences on the specific emotion categories of the DFAR are displayed in table 3. *Indicates $P < .06$. DFAR, Degraded Facial Affect Recognition; HT, Hinting Task; PANSS, Positive and Negative Syndrome Scale; SIS-R, Structured Interview for Schizotypy—Revised.

with subclinical negative and positive symptoms were nonsignificant. DFAR performance was significantly associated with subclinical negative symptoms only. The associations with subclinical disorganized and positive symptoms were nonsignificant (see table 4).

Within-Trait Cross-Sibling Analyses

The analyses between at least 674 patient-sibling pairs showed significant within-trait familial clustering of HT performance, DFAR performance, and **IQ** (see table

4). All within-trait cross-sibling analyses were controlled for age, gender, and in case of the HT and DFAR for IQ.

Cross-Trait Cross-Sibling Analyses

None of the associations between HT performance in patients and subclinical symptoms in siblings were significant. DFAR performance in patients and subclinical disorganized symptoms in siblings were significantly associated. The associations between patients' DFAR performance and siblings' subclinical symptoms were

Table 3. Group Differences on the Social Cognitive Tests

	Controls vs Patients		Controls vs Relatives		Patients vs Relatives	
	β	P	β	P	β	P
HT	−.17	<.001	.01	.61	−.18	<.001
DFAR	−.13	<.001	−.03	.30	−.10	<.001
Neutral	−.04	.12	−.01	.80	−.04	.11
Happy	.02	.51	.04	.13	−.02	.36
Anger	−.13	<.001	−.04	.12	−.09	<.001
Fear	−.10	<.001	−.03	.21	−.07	.002

Note: β = adjusted for age, gender, IQ, and face recognition ability (for the DFAR only). Abbreviations are explained in the first footnote to table 1.

Table 4. Cross-Trait Within-Patients/Siblings, Within-Trait Cross-Sibling and Cross-Trait Cross-Sibling Analyses

Analysis	n	Association	β	b	P value	95% CI: lb/ub
Cross-trait within-patients analysis	948	HT-disorganized symptoms	−.33	−.73	<.001	−.91/−.56
	946	HT-negative symptoms	−.21	−.51	<.001	−.70/−.32
	957	HT-positive symptoms	−.11	−.27	<.001	−.46/−.07
	880	DFAR-disorganized symptoms	−.21	−.12	<.001	−.17/−.07
	875	DFAR-negative symptoms	−.11	−.07	<.001	−.12/−.02
	885	DFAR-positive symptoms	−.09	−.05	.008	−.11/−.001
Cross-trait within-siblings analysis	1001	HT-disorganized symptoms	−.09	−.01	.006	−.02/.001
	1001	HT-negative symptoms	−.04	−.01	.17	−.04/.01
	1001	HT-positive symptoms	−.04	−.01	.22	−.03/.01
	935	DFAR-disorganized symptoms	−.02	−.0004	.55	−.002/.001
	935	DFAR-negative symptoms	−.07	−.003	.04	−.01/.001
	935	DFAR-positive symptoms	−.02	−.001	.60	−.005/.003
Families*						
Within-trait cross-sibling analysis	755	HT		.08	<.001	.02/.13
	674	DFAR		.09	.002	.02/.17
	766	IQ		.42	<.001	.35/.51
Cross-trait cross-sibling analysis	715	HT patients-disorganized symptoms siblings		.001	.87	−.01/.01
	717	HT patients-negative symptoms siblings		.01	.30	−.01/.02
	721	HT patients-positive symptoms siblings		.002	.75	−.01/.02
	640	DFAR patients-disorganized symptoms siblings		.001	.02	−.0002/.003
	639	DFAR patients-negative symptoms siblings		.003	.10	−.002/.01
	643	DFAR patients-positive symptoms siblings		.003	.06	−.001/.01

Note: *All analyses are adjusted for age, gender, IQ, and the respective relevant traits (ie, social cognition in siblings and symptoms in patients); 95% CI, confidence interval for b; lb, lower bound; ub, upper bound; Abbreviations are explained in the first footnote to table 1.

marginally significant for positive subclinical symptoms and nonsignificant for negative subclinical symptoms (see table 4). All cross-trait cross-sibling analyses were controlled for age, gender, and IQ and the respective symptom domain and cognitive task across siblings.

Discussion

Our results showed no familial covariation of the associations between ToM performance and psychotic symptoms across siblings, suggesting that the overlap between the 2 phenotypes, which is seen in patients, does not reflect a shared familial etiology. However, indicative of a shared etiology, familial covariation was present between patients' emotion recognition ability and disorganized and, to a lesser degree, positive, but not negative subclinical symptoms in siblings.

Social Cognitive Impairment Over the Psychosis Continuum

In line with earlier evidence,[27] a worse performance in ToM and emotion recognition ability was weakly associated with a higher psychosis risk. Patients performed worse on the HT than siblings and controls and siblings mean values were intermediate. Yet, in contrast to what had been expected on the basis of previous research,[6,8,9,33,34] the difference between siblings and

controls was small and not significant. A similar pattern of results was present for emotion recognition. Despite their overall impairment, the valence-related performance pattern of patients was similar to that of siblings and controls, with a superior recognition of happy and neutral emotions as compared to fearful and angry emotions.[35] Analyses per emotion category indicated that the overall effect was mainly driven by differences in the recognition of angry and fearful emotion. The finding of unimpaired recognition of neutral and happy affect but impaired recognition of negative affect is consistent with previous reports on the disproportionate impairment in the identification of negative emotions and lends further support to emotion-specific processing deficits in schizophrenia.[33,34,36–38]

Symptoms and Social Cognition

To investigate the nature of the associations between symptoms and social cognitive impairment in patients and siblings, we carried out cross-trait within-group analyses first. Within patients, poorer ToM performance was consistently and significantly associated with all 3-symptom clusters. The strongest association was present with disorganized symptoms. The association with negative symptoms was intermediate, and the weakest association was present with positive symptoms. Emotion recognition was significantly associated with

disorganized and negative symptoms, albeit to a lesser degree than ToM. Within siblings, significant associations were present between ToM and subclinical disorganized symptoms but not negative symptoms. Emotion recognition, in turn, was significantly associated with subclinical negative symptoms but not disorganized symptoms. Associations between both social cognitive domains and positive symptoms were entirely specific to patients.

In the next step, we investigated whether social cognitive performance clusters within families. The within-trait cross-sibling analyses revealed considerable familial clustering of ToM and emotion recognition, although to a slightly lesser degree. The social cognitive clusters remained significant when controlled for IQ, supporting a substantial independence from the neurocognitive domain.[15]

The cross-trait cross-sibling investigations revealed no significant associations between ToM in patients and any subclinical symptoms in their siblings. No significant association was present between patients' emotion recognition and negative symptoms in siblings. However, indicative of a common etiological transmission, patients' emotion recognition ability was significantly associated with siblings' disorganized subclinical symptoms and, although to a lesser degree, with their positive subclinical symptoms.

The results confirmed previously observed associations between symptoms and social cognitive performance within the patients. Only 2 significant cognition-symptom associations were present within the sibling group. This finding may be due to constricted variation within siblings. Alternatively, other symptom-cognition associations may only come into effect once the disorder has been developed. Our findings corroborated the potential role of both social cognitive functions as intermediary phenotypes of the illness. Familial clustering indicates a possible shared a etiological basis underlying impaired social cognition in siblings and patients. Obviously, the 2 phenotypes may also have been acquired in a shared environment (eg, parental neglect). However, research suggests that the familial liability to schizophrenia strongly represents the influence of shared genes, so a partly genetic transmission of the phenotypes is therefore likely.[39,40]

The differential cross-trait cross-sibling associations between social cognitive functioning and the specific symptom clusters point toward partly differential etiological substrates. The etiology of emotion recognition deficits and symptoms seems to vary between the 3 clusters. Emotion recognition deficits may be useful endophenotypes in the search for the genetic causes of disorganized symptoms and possibly to a lesser degree of positive but not negative symptoms. Alternatively, it could be argued that these differences are due to a lower sensitivity of the SIS-R to positive and even more strongly to negative symptoms. However, previous evidence proved the SIS-R is sensitive to family-specific variation in positive and negative subclinical symptoms.[41]

Our results did not substantiate a shared etiology of ToM deficits and any of the 3 symptom clusters. In this case, the overlap seems to originate at an individual level. In line with previous research,[15] the present findings suggest a substantial heterogeneity and multicomponent structure of social cognition. Analogous to other (genetic) predispositions that appear in the phenotype under particular conditions (eg, sunburn and skin cancer), manifold factors may bring the associations between ToM impairment and symptoms to expression. A dynamic interplay of symptoms, cognitive processes, behavioral, and environmental factors may offer a suitable explanation for our findings. Patients' ToM deficits may play a role in the formation, exacerbation and maintenance of psychotic symptoms.[42,43] Specifically, impaired ToM could lead to a paranoid interpretation of other persons' intentions as malevolent. Also, impaired emotion recognition, although possibly to a lesser degree, may cause negative misinterpretations of social-emotional cues. Misperceptions may lead to the avoidance of social situations or contacts and problematic social behavior, which may be partially reflected in negative symptoms. Disorganized symptoms and social cognitive deficits, moreover, could be associated on a level of conceptual understanding rather than misinterpretation of social situations.

Strengths and Limitations

The current study used a uniquely large sample with adequate power to detect even delicate effects. Also, the patient–sibling based design has the important advantage of automatically controlling for confounds that are associated with the illness, such as residual symptoms or the effects of antipsychotic medication. Sibling-based designs also have the advantage of low confounding by unobserved factors that may affect case–control comparisons in unrelated subjects, such as shared socioeconomic and developmental conditions.

The findings should be considered keeping the following limitations in mind. First, our data cannot completely clarify whether any abnormalities in social cognition are present prior to the illness onset (ie, potentially causal) or whether they are covarying epiphenomena of the clinical picture. However, in line with previous research, our results indicate that the association between social cognitive impairment and symptoms is not only present during acute psychosis.[27] Second, we had to use different measures to assess the clinical phenotypes in patients and siblings. The SIS-R is not suitable for the use in patients because it may underlie ceiling effect. The PANSS interview, in turn, may be subject to floor effects when used in relatives. However, we aimed to establish concurrent validity between the 2 measures by structuring the items along the same symptom dimensions. Third, as reported by previous research, the social cognitive performance differences between siblings and controls were subtle.[34,44]

This may partly be due to the nature of the social cognitive tasks that we employed. The HT is specifically prone to ceiling effects. It also needs to be noted that our measure may only reflect a part of the broader domain that it belongs to. Other ToM tasks possibly tap into different mentalizing capacities and future studies should aim to include more tasks to get a better representation of the domain. Another possible explanation for the relatively small effects could be a self-selection bias, in which only relatively stable patients volunteer to participate in demanding research. Also, biased answering of siblings who are highly aware of the symptoms of psychosis and who may want to appear healthy may have reduced the effects. Obviously, it is difficult to translate statistical effect sizes and *P* values into clinical significance one to one. Even though effect sizes on some tasks are small, the work of our group and others[45,46] has shown that social cognitive performance in the domains of ToM and emotion perception and processing is associated with functional outcome, possibly to a higher degree than many other cognitive factors. These findings imply a substantial clinical significance of the degree of social cognitive impairment that is typically seen in schizophrenia.

Conclusions

ToM and emotion recognition impairments are associated with the liability to nonaffective psychosis and cluster within families. Our findings support the idea that both traits could be suitable intermediary phenotypes for genetic studies. The cross-trait cross-sibling analyses did not support a familial continuity between subclinical symptoms and ToM. However, a shared familial etiology may underlie the overlap between emotion recognition deficits and disorganized and positive psychotic symptoms. Emotion recognition deficits could therefore be useful intermediate phenotypes in the identification of the familial causes of specific symptoms of psychosis.

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