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## Schizophrenia Research

journal homepage: [www.elsevier.com/locate/schres](http://www.elsevier.com/locate/schres)

## Impact of social anxiety on social cognition and functioning in patients with recent-onset schizophrenia spectrum disorders

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### ARTICLE INFO

#### Article history:

Received 17 September 2012

Received in revised form 16 January 2013

Accepted 18 January 2013

Available online xxxx

#### Keywords:

First-episode

Mentalizing

Theory of mind

Social knowledge

Emotion recognition

Social phobia

### ABSTRACT

Schizophrenia patients display important rates of comorbid social anxiety disorder (SAD) but few studies have directly examined how SAD affects the presentation of schizophrenia, notably social cognition deficits and functioning.

**Aims:** To compare social cognition performance of schizophrenia patients who meet the diagnostic criteria for a comorbid SAD (SZ+) relative to patients without such comorbidity (SZ−) and to determine if the impact of social cognition performance on functioning is moderated by that comorbidity.

**Method:** Social cognition performance (emotion recognition, social knowledge, and mentalizing), a control non-social reasoning task, as well as clinical symptoms and functioning were assessed in 26 patients with comorbid SAD (SZ+), 29 SZ− and 84 healthy controls.

**Results:** Patient groups significantly differed from each other on social knowledge performance, but not in levels of symptoms or overall functioning. Relative to healthy controls, SZ+ were impaired uniquely on mentalizing, whereas SZ− showed a more encompassing social cognition deficit that included mentalizing, social knowledge and non-social reasoning impairments. Mentalizing was the best predictor of functioning across both patient groups. Importantly, non-social reasoning negatively influenced mentalizing and in turn functioning only in the SZ− group.

**Conclusions:** The overall pattern of results indicates common mentalizing deficits in SZ+ and SZ−; however, these deficits appear linked to different underlying deficits and different pathways to functional impact in the two patient subgroups. This study highlights some distinctive characteristics of schizophrenia patients with comorbid SAD and signals a need for further investigations into the sources of the mentalizing and functioning impairments in SZ+ patients.

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### 1. Introduction

People with schizophrenia (SZ) display important rates of comorbid anxiety disorders (Kendler et al., 1996; Cassano et al., 1998; Cosoff and Hafner, 1998; Pallanti et al., 2004; Voges and Addington, 2005) and yet we know little about the relationship of these comorbid disorders with other aspects of schizophrenia such as positive or negative symptoms, cognitive deficits, and functioning. Among anxiety disorders, social anxiety disorder (SAD) seems particularly important as it was identified as the most prevalent comorbid anxiety disorder in people with SZ in a recent meta-analysis (Achim et al., 2011a), with a pooled prevalence

of 14.9%, a rate much higher than in the general population (3.6%) (Somers et al., 2006). Comorbid SAD appears early in the progression of SZ and can be detected with similar rates in first-episode psychosis (13.4%) as in more chronic SZ patient samples (15.2%) (Achim et al., 2011a). In addition, even higher rates of SAD (25.6%) have been observed in SZ in studies that relied on expanded clinical assessment tools, suggesting that SAD often goes undetected in SZ when standard diagnostic tools such as the Structured Clinical Interview for DSM Disorders (SCID) are used (Achim et al., 2011a). Another reason for studying SAD in SZ is the emerging evidence regarding the negative impacts of this comorbidity on SZ patients, including poorer levels of functioning and decreased self-esteem (Pallanti et al., 2004; Voges and Addington, 2005). Given that cognitive abilities and especially social cognition performance are recognized as the most important predictors of functioning in people with SZ (Brune et al., 2007; Fett et al., 2011), it opens the possibility of a relationship between social cognition deficits and comorbid SAD in these patients.

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Social cognition can be defined as the collection of cognitive processes (e.g. mentalizing, social knowledge, emotion recognition) that allow us to understand others and that guide social interactions (Green et al., 2008). Social cognition deficits have been consistently observed in SZ and are now recognized to be at the core of the disorder (Sprong et al., 2007; Bora et al., 2009; Kohler et al., 2010). Impairments in social cognition may also give rise to high levels of social anxiety symptoms and increased risk of developing a comorbid SAD (Jacobs et al., 2008). In this case, social cognition deficits and social anxiety disorders would represent a common risk factor for poor functioning in SZ and worse social cognition performance would be expected in SZ patients that are affected with comorbid SAD. Though this hypothesis seems logical and has obvious implications for treatment, no study has yet looked at social cognition abilities or its impact on functioning as a function of comorbid SAD diagnosis in people with SZ. To be considered as meeting full diagnostic criteria for SAD, a patient with schizophrenia has to present with social anxiety symptoms that are not strictly dependent on psychotic symptoms (for instance ideas of reference or persecutory delusions). The few studies that have assessed the relationships between social cognition and social anxiety symptoms (Lysaker et al., 2010a,b; Achim et al., 2011b) have not made this distinction, which could explain the mixed results from these studies.

The first aim of the current study is to compare the pattern of social cognition performance in patients with SZ spectrum psychotic disorders that also meet full criteria for a comorbid social anxiety disorder (SZ+) relative to schizophrenia patients that do not (SZ-) and healthy controls. Given the established relationship between social cognition and functioning (Brune et al., 2007; Fett et al., 2011), a second aim is to assess the potential moderating influence of comorbid SAD on cognitive pathways to functioning impairments in people with schizophrenia. The first aim was tested using between group comparisons whereas the second aim was tested using moderation analyses that allowed us to determine whether social cognition performance has a greater impact on functioning in SZ+ relative to SZ- patients. We initially hypothesized that meeting criteria for a comorbid SAD would be associated with poorer social cognition performance (Jacobs et al., 2008) and that these deficits would affect functioning to a greater extent in SZ+ patients given that cognitive models of social anxiety (Clark and Wells, 1995; Wells et al., 1998; Spurr and Stopa, 2002) emphasize the biased assessments of others' thoughts in this population, with a recognized impact on social behavior and social interactions (Spurr and Stopa, 2002, 2003).

## 2. Materials and methods

### 2.1. Participants

Fifty-nine patients with recent-onset SZ spectrum disorders (mean age = 25.7, 51 males) were recruited from the Clinique Notre-Dame-des-Victoires of the Institut Universitaire en Santé Mentale de Québec. Diagnoses included SZ (n = 41), schizoaffective disorder (n = 10), delusional disorder (n = 6) and psychosis not otherwise specified (n = 2). Our decision to include patients with this range of early diagnoses was based on previous reports that these diagnoses fall within the SZ spectrum when diagnoses are reassessed later in the course of the illness or based on family studies (Kendler et al., 1995; Schimmelmann et al., 2005; Malla et al., 2006). Patients were excluded from the study if they presented with mental retardation (estimated IQ below 70) or with a neurological disorder, or if their treating psychiatrist judged that they were not stable enough to provide informed consent for the study. All patients but one were taking a second-generation antipsychotic. In addition, 16 received antidepressants and 8 received benzodiazepines either on a daily basis or as needed. The duration of psychosis since first antipsychotic treatment ranged from 1 to 68 months, with a mean of 22 months.

Eighty-seven (87) healthy control subjects were recruited from ads in local media or public places. They were excluded if they presented with a psychosis, mood disorder or neurological disorder, had a first-degree relative with psychosis, or were taking a psychoactive medication according to our screening or SCID-NP (First et al., 1998). This led to the exclusion of three participants with a simple phobia, one of whom additionally showed agoraphobia. The 84 remaining healthy participants were included in our analyses (mean age = 24.0, 58 men).

### 2.2. Clinical assessment of comorbidities

Clinical assessments were performed with a comprehensive semi-structured interview based on the SCID-IV (First et al., 1998), which includes all the SCID questions further supplemented with questions from several other instruments that provide a detailed coverage of the full range of symptoms that the patients present with and the relationships between these symptoms and the conditions being considered. All the added instruments (see Supplementary material for a complete list) have been validated. The resulting semi-structured interview (Roy et al., 2011) notably includes all the questions from the Liebowitz Social Anxiety Scale (LSAS) inserted in the SCID module on social anxiety disorder to further assess social anxiety symptoms (Liebowitz, 1987). All interviews were conducted by a trained research assistant and were subsequently reviewed by one of the authors (MAR) who is an experienced psychiatrist.

### 2.3. Assessment of symptoms and functioning

Positive symptoms, negative symptoms and general psychopathology symptoms were assessed using the PANSS (Kay et al., 1987), a 30-item scale widely used to assess symptoms of schizophrenia. Global level of functioning was assessed with the SOFAS (American Psychiatric Association, 2000), which produces a single score reflecting current levels of social and occupational functioning. The treating psychiatrists rated these scales based on all available information including interviews with the patients, information from the clinical interview used for the current project (it includes all the PANSS questions as well as a module on functioning), information from family members or from other members of the staff at the Clinique Notre-Dame-des-Victoires.

### 2.4. Social cognition assessment

Social cognition was assessed with the Batterie Intégrée de Cognition Sociale (BICS), a social cognition test battery with good psychometric properties that includes three main measures of social cognition (Achim et al., 2012):

- 1) The *mentalizing test* relies on written scenarios. Open questions test the ability to infer the target character's mental states (26 mentalizing questions).
- 2) The *social knowledge task* also presents hypothetical situations, but no specific character is presented and nothing is being expressed. Instead, participants have to determine how people in general would feel or react in 14 different situations.
- 3) The *emotion recognition* task consists of consecutive presentations of 14 standardized facial affect stimuli (Ekman and Friesen, 1976). For each item, participants select the corresponding emotion from a given list of labels (happy, surprise, sad, angry, disgust, fear or neutral).

In addition, the BICS includes a control task that assesses *non-social reasoning* from 6 stories that are interspersed within the mentalizing test.

## 2.5. Analyses

Patients were first classified into two groups according to whether they met (the SZ+ group) or not (the SZ− group) full diagnostic criteria for a comorbid SAD (American Psychiatric Association, 2000) based on our clinical interview. Demographic and clinical variables for these two groups were compared using t-tests or Mann–Whitney tests, as appropriate.

Next, normality of the distributions was corrected with a square root transformation for mentalizing and non-social reasoning (see Table 2 for uncorrected means), following which the two patient groups were compared to each other and to the controls on all BICS measures through analyses of variances performed with gender, age and education level used as covariates.

Then, given the expected impact of social cognition on functioning (Fett et al., 2011; Achim et al., 2012), we examined these effects and assessed whether comorbid SAD acts as a moderator 1) of the direct effect of each BICS measure on functioning (Fig. 1A) or 2) of the indirect effect of non-social reasoning, social knowledge or emotion recognition on functioning through their respective effect on mentalizing (Fig. 1B). These analyses were performed following Hayes' method (Hayes, 2012) for path analysis, which relies on standard path analysis techniques (see tested models in Fig. 1) as well as bootstrapping tests (here 10,000 iterations) to assess the significance of the full path for models such as ours that include indirect effects.

Additionally, relationships between LSAS ratings and BICS measures were examined across all patients using Pearson correlations to determine whether comorbid SAD diagnosis and social anxiety symptoms showed similar effects on social cognition performance in people with SZ. The same correlations were also examined separately for each patient group.

Finally, we explored the correlations between LSAS ratings and the suspiciousness/persecution item from the PANSS (item p6), also performed separately for each patient group, which could help

highlight the nature of the social anxiety symptoms observed in SZ− and SZ+ patients.

## 3. Results

### 3.1. Comorbidity status

Of the 59 patients, 29 showed neither prior nor current comorbid SAD and were included in the SZ− group, 26 currently met all diagnostic criteria for comorbid SAD and were included in the SZ+ group, whereas the remaining four patients had a history of comorbid SAD that no longer met full diagnostic criteria. These four patients were excluded from further analyses as there was no a priori reason to include them with either the SZ− or the SZ+ group, and not enough patients in that situation to form a separate group.

As shown in Table 1, the SZ+ group was older than the SZ− group ( $p=.018$ ) despite similar duration of psychosis ( $p=.390$ ). SZ+ patients showed higher social anxiety symptom ratings on the LSAS than SZ− patients ( $p<.001$ ), but the two patient groups did not differ with respect to severity of PANSS positive symptoms, negative symptoms or general psychopathology, and both groups showed similar levels of functioning (see Table 1).

### 3.2. BICS performance

As shown in Table 2, SZ+ and SZ− patients differed significantly only in terms of their performance on the social knowledge task, with the SZ− group producing a lower performance ( $p=.038$ ).

Comparisons of each patient group with the controls confirmed that only the SZ− group showed a social knowledge impairment ( $p=.038$ ) whereas the SZ+ group showed normal performance on that measure. Interestingly, both the SZ− and SZ+ groups showed impaired mentalizing performance relative to healthy controls (both  $ps<.001$ ) while non-social reasoning was only impaired in the SZ− group ( $p=.048$ ).

### 3.3. BICS performance and functioning in schizophrenia

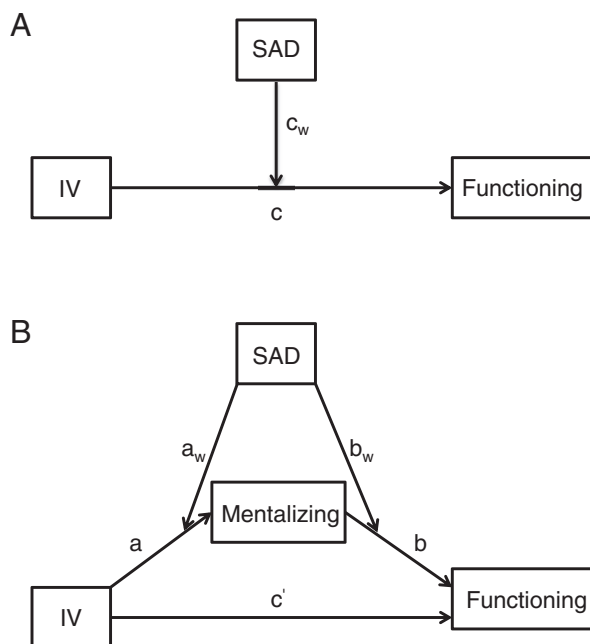
As shown in Table 3 and Fig. 2A, only mentalizing was a significant direct predictor of functioning ( $p=.004$ ), and the moderating effect of comorbid SAD on that relationship did not reach significance ( $p=.227$ ).

As presented in Table 3 and Fig. 2(B, C and D), our moderated mediation analyses revealed indirect effects of both non-social reasoning and social knowledge on functioning through mentalizing (a and b are significant in both cases). However, a significant moderating effect of SAD was uniquely observed on the path from non-social reasoning to mentalizing ( $a_w$ ,  $p=.021$ ), and assessment of the significance of the indirect effect (ab path) separately for each patient group using bootstrapping procedures and percentile-based confidence intervals revealed a significant indirect effect in SZ− (95% CI = 1.2 to 13.1) but not in SZ+ (95% CI = −0.8 to 4.0).

### 3.4. Social anxiety symptoms and BICS performance

None of the correlations between LSAS ratings and BICS measures reached significance (all  $ps>.26$ ), be it across all SZ patients or when assessed separately for the SZ− or the SZ+ groups.

Within the SZ− subgroup, 9 patients reported social anxiety symptoms that could correspond to DSM-IV criterion A for SAD (important fear of social or performance situations) but were nonetheless included in the SZ− group as these fears were limited to the topic of their psychotic symptoms and hence did not meet all other diagnostic criteria for SAD (i.e. social anxiety symptoms were not independent of psychotic symptoms). The social cognition performance of these 9 SZ− patients did not differ from the other 20 SZ− patients (all  $ps>.30$ ) while a trend



**Fig. 1.** Models testing for a potential moderation by comorbid social anxiety disorder of the direct and indirect effects of our target variables on functioning. A. Illustration of our moderation model. B. Illustration of our moderated mediation model. For each independent variable (IV), the coefficients and level of significance of all model parameters are reported in Table 3. SAD = social anxiety disorder, a dichotomous variable that distinguishes SZ− and SZ+ patients. IV = independent variable from the BICS, including mentalizing, social knowledge, emotion recognition and non-social reasoning for model A, and social knowledge, emotion recognition and non-social reasoning for model B.

**Table 1**  
Demographic and clinical characteristics (mean and SD) of the schizophrenia groups.

	SZ +	SZ –	SZ + vs. SZ –	Controls	SZ + vs. Controls	SZ – vs. Controls
N/%	26/44.1%	29/49.2%		84		
Age	27.0 (4.2)	24.0 (4.8)	t = –2.44, p = .018*	24.0 (3.6)	t = 3.54, p = .001*	t = 0.03, p = .979
Gender (men/women)	21/5	26/3	Z = –0.93, p = .355	58/26	Z = –1.26, p = .207	Z = –2.82, p = 0.23*
Education <sup>a</sup>	3.8 (1.1)	4.1 (0.9)	t = 0.98, p = .333	2.96 (1.0)	t = 3.74, p < .001*	t = 5.38, p < .001*
Duration of psychosis (months)	23.6 (18.9)	19.6 (15.7)	t = –0.87, p = .390			
PANSS						
Positive	14.6 (3.6)	14.8 (5.0)	t = 0.18, p = .857			
Negative	16.7 (5.7)	17.0 (7.1)	t = 0.18, p = .859			
General	32.2 (6.6)	30.6 (9.0)	t = –0.75, p = .455			
LSAS <sup>b</sup>	57.8 (21.2)	32.8 (18.7)	t = –4.62, p < .001*			
SOFAS <sup>b</sup>	56.9 (12.1)	56.7 (12.8)	t = –0.07, p = .943			

SZ – = patients with schizophrenia without a comorbid social anxiety disorder; SZ + = patients with schizophrenia with a comorbid social anxiety disorder; PANSS = Positive and Negative Symptom Scale; SOFAS = social and occupational functioning assessment scale, LSAS = Liebowitz social anxiety scale.

<sup>a</sup> Education categories from the Hollingshead two-factor index of social position (Miller, 1991) on a 7-point scale. Lower numbers indicate higher education. A score of 4 is equivalent to completed high school whereas a score of 3 is equivalent to a technical course or an unfinished bachelor degree.

<sup>b</sup> The SOFAS was missing for one SZ – patient and the LSAS for another SZ – patient.

\* Indicates a significant effect (p < .05).

for lower social knowledge performance was observed in these 9 patients relative to the SZ + group (p = .08).

### 3.5. Social anxiety symptoms and suspiciousness/persecution

The correlation between social anxiety symptoms and the suspiciousness/persecution item from the PANSS (item p6) was significant in the SZ – group (r = .41, p = .029) but clearly absent in the SZ + group (r = –.08, p = .706), despite higher average p6 ratings in the SZ + relative to the SZ – group (respectively 3.2 and 2.4, t(53) = 2.8, p = .008).

## 4. Discussion

This study investigated the clinical and social cognitive characteristics of patients with recent-onset schizophrenia spectrum disorders who also met full DSM-IV criteria for a comorbid social anxiety disorder (SZ + group) compared to schizophrenia patients without comorbid SAD (SZ – group) and healthy controls. Our thorough clinical evaluation allowed us to observe social anxiety symptoms meeting diagnostic criteria for a comorbid SAD in 44.1% of our patients. This high prevalence relative to other studies supports our previous finding (Achim

et al., 2011a) that supplementing the SCID with additional tools improves the detection of comorbid anxiety disorders in SZ.

Consistent with previous studies (Pallanti et al., 2004; Michail and Birchwood, 2009), our SZ + and SZ – groups did not differ in their levels of PANSS symptoms, but our results revealed different patterns of social cognition impairment. Contrary to our expectations, the SZ + patients were impaired only on mentalizing whereas the SZ – group showed a more encompassing social cognition deficit that included mentalizing impairments but also social knowledge and general reasoning impairments. Importantly, the two patient groups significantly differed from each other with respect to their social knowledge performance, with better performance in the SZ + group. The finding that social knowledge was intact in the SZ + group is intriguing as these patients, who are additionally burdened by SAD, showed less rather than more encompassing social cognition deficits. Interestingly, we showed that social knowledge performance specifically varied as a function of SAD diagnosis, regardless of levels of social anxiety symptoms measured using the LSAS. This pattern of results is consistent with the idea that schizophrenia patients showing social anxiety symptoms that meet full diagnostic criteria (i.e. the social anxiety symptoms that they report are not strictly dependent on psychotic symptoms) form a distinct subgroup of patient. Our results suggest

**Table 2**  
Performance (mean and SD), between group analyses, and post-hoc group comparisons.

	SZ –	SZ +	Controls	SZ + vs SZ –	SZ + vs Controls	SZ – vs Controls	Between group
Social knowledge (/14)	9.6 (2.7)	11.0 (1.6)	11.2 (1.7)	F = 4.54, p = .038*	F = 1.03, p = .312	F = 4.39, p = .038*	F = 4.41, p = .014*
Emotion recognition (/14)	10.6 (1.8)	10.6 (1.7)	11.0 (1.3)	F = 0.08, p = .779	F = 1.846, p = .177	F = 1.44, p = .233	F = 1.49, p = .230
Mentalizing (/52) <sup>a</sup>	37.2 (8.6)	38.0 (7.1)	44.7 (4.4)	F = 0.07, p = .793	F = 11.91, p = .001**	F = 12.05, p = .001**	F = 9.54, p < .001**
Non-social reasoning (/12) <sup>a</sup>	9.8 (1.8)	10.4 (1.6)	10.9 (1.3)	F = 2.42, p = .126	F = 1.91, p = .170	F = 4.02, p = .048*	F = 3.03, p = .052

\*p < .05; \*\*p < .005; SZ – = patients with schizophrenia without a comorbid social anxiety disorder; SZ + = patients with schizophrenia with a comorbid social anxiety disorder; CO = healthy controls. There were two patients for whom the combined story task was not available due to incomplete testing, one in the SZ – group and one in the SZ + group.

<sup>a</sup> Even though uncorrected means are reported here, the analyses for mentalizing and non-social reasoning were performed on scores that had previously been transformed (square-root transformation) to correct the normality of the distributions. Group comparisons were performed with gender, age and education level as covariates. The same analyses performed without demographic covariates led to a similar pattern of results with the exception that the overall group effect for non-social reasoning reached significance (p = .007).



**Table 3**  
Results for the direct and indirect effect models.

Model and parameter (see Fig. 2)	Unstandardized path coefficient	SE	t	p
<i>Direct effect models</i>				
MTZ-functioning				
c	6.43	2.15	2.99	.004**
c <sub>w</sub>	−4.16	3.39	−1.22	.227
SK-functioning				
c	0.67	0.89	0.76	.453
c <sub>w</sub>	−1.72	1.82	−0.95	.349
ER-functioning				
c	0.17	1.31	0.13	.899
c <sub>w</sub>	1.79	1.95	0.91	.365
NSR-functioning				
c	6.43	3.36	1.92	.061
c <sub>w</sub>	−7.49	4.64	−1.62	.112
<i>Indirect effect models</i>				
SK-MTZ-functioning				
a	0.16	0.07	2.48	.017*
a <sub>w</sub>	0.07	0.13	0.55	.585
b	7.19	2.32	3.10	.003**
b <sub>w</sub>	−4.41	3.42	−1.29	.203
c'	−0.71	0.80	−0.88	.383
ER-MTZ-functioning				
a	0.06	0.11	0.55	.582
a <sub>w</sub>	0.04	0.16	0.23	.823
b	6.32	2.17	2.91	.005**
b <sub>w</sub>	−4.27	3.42	−1.25	.218
c'	0.64	0.93	0.69	.494
NSR-MTZ-functioning				
a	0.91	0.24	3.74	<.001**
a <sub>w</sub>	−0.80	0.33	−2.40	.021*
b	6.63	2.40	2.77	.008**
b <sub>w</sub>	−4.32	3.53	−1.22	.227
c'	−0.48	2.45	−0.20	.846

MTZ = mentalizing; SK = social knowledge; ER = emotion recognition; NSR = non-social reasoning.

\* p < .05.

\*\* p < .01.

that this subgroup of patients could show a greater sensitivity to others, showing symptoms of social anxiety but also making them less prone to social knowledge deficits. Though the literature does not provide much evidence regarding social knowledge performance in people with a primary SAD (Plana et al., in preparation), enhanced empathy has been reported in primary SAD patients (Tibi-Elhanany and Shamay-Tsoory, 2011). Given that a good number of our patients were receiving antidepressants or benzodiazepines and that these patients were more likely to be in the SZ+ group, an alternative possibility is these treatments have an effect on social cognition and eventually on functioning, which could explain why our SZ+ patients showed better social knowledge performance and also why they did not show additional functioning difficulties such as in previous studies (Pallanti et al., 2004; Voges and Addington, 2005). Further work will definitely be required to understand the distinctions between SZ+ and SZ− patients, including their treatment needs to achieve optimal functioning, and to determine whether SZ+ patients are better conceived as having two relatively independent diagnoses or rather represent a subgroup of schizophrenia patients that differ from both SZ− patients and patients with a primary SAD (i.e. SAD not in the context of a psychotic disorder).

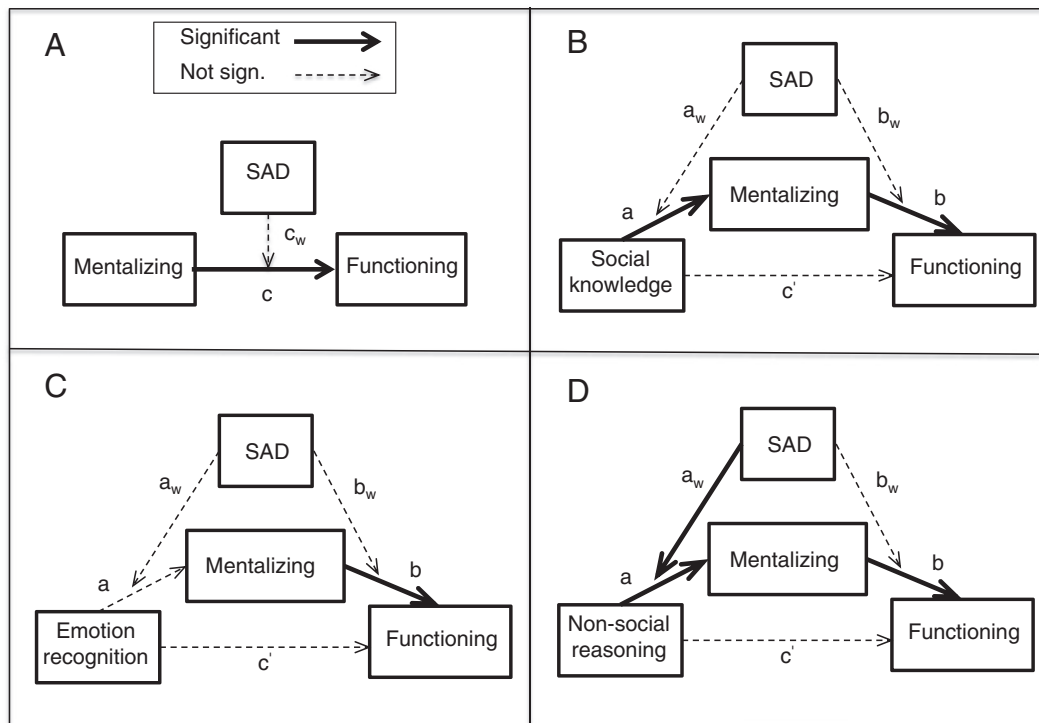
The fact that we observed similar levels of functioning in SZ− and SZ+ patients could also be related to the fact that different aspects of functioning may be differentially affected in SZ− and SZ+ and that the SOFAS, a very global measure, was not sufficiently sensitive to detect such differences. Pallanti et al. (2004) previously reported worse functioning in SZ+ relative to SZ− in some areas of functioning (e.g. work, socialization and personal well-being) but not all (e.g. they

found better family adjustment). More comprehensive measures of functioning should thus be used in future studies.

Even though our two patient groups did not differ in their levels of functioning, a major finding from the current study is that non-social reasoning had an important indirect effect on functioning through its effect on mentalizing in SZ− patients, but not in SZ+ patients. Furthermore, though social knowledge influenced mentalizing across both patient groups, it was only impaired in the SZ− group and was thus more likely to show a negative (rather than positive) influence on mentalizing and functioning in SZ− patients. Overall, non-social reasoning and social knowledge deficits are two target processes linked to mentalizing impairments and in turn to impaired functioning in SZ−, whereas the specific processes at play in the mentalizing deficits of SZ+ patients and that negatively influence their functioning remain unidentified. Since attribution biases have been reported in people with a primary SAD (i.e. SAD not in the context of SZ...) (Clark et al., 1997; Amin et al., 1998; Plana et al., in preparation), one possibility is that such biases are also present in SZ+ and negatively affect mentalizing and functioning. In any case, our study suggests that different pathways lead to mentalizing and functioning deficits in SZ+ and SZ− patients. Overall, this study thus highlights the relevance of taking social anxiety into account to further our understanding of the pathways to mentalizing and functioning impairments in people with SZ and suggests that further investigations into the neurocognitive processes at play are warranted. If the distinctions between the cognitive deficits of SZ+ and SZ− patients can be confirmed, treatments that target cognitive or social cognitive deficits in patients with schizophrenia (e.g. Moritz and Woodward, 2007; Horan et al., 2009; Aghotor et al., 2010; Horan et al., 2011; Moritz et al., 2011) should be adapted in consequence.

Another interesting observation is that no significant relationship emerged between LSAS scores and social cognition performance in this study. This observation is consistent with a previous study that found no significant relationship between social anxiety symptoms and emotion recognition performance in SZ (Lysaker et al., 2010b). We have previously observed a significant negative relationship between LSAS ratings and empathic perspective-taking, defined as the tendency to take the perspective of others in social situations (Achim et al., 2011b). However, in that study ratings on perspective-taking were acquired using a self-rating questionnaire and it is possible that perceived abilities differ from actual abilities in SZ patients. Generally, social anxiety symptom ratings should be interpreted with care in SZ as high ratings can sometimes be strictly observed in the context of psychotic suspiciousness or other psychotic symptoms (Lysaker et al., 2010a). The relationship between suspiciousness ratings and LSAS scores that was here uniquely observed in our SZ− group supports the idea that social anxiety symptoms are of a different nature in SZ+ and SZ− patients. These results also support the validity of our classification of patients into the SZ+ and SZ− groups, since patients for whom high levels of social anxiety symptoms could be explained by psychotic suspiciousness were appropriately included into the SZ− group.

The limitations of this study include the lack of sensitivity of our emotion recognition test, which did not allow us to highlight an emotion recognition deficit that has often been reported in SZ (Chan et al., 2010; Kohler et al., 2010). Emotion recognition deficits have however not been systematically observed in recent-onset or first-episode SZ patients and could be related to duration of psychosis (Achim et al., 2012). The relationship between emotion recognition and comorbid SAD in SZ thus deserves further investigation in more chronic samples or using more sensitive measures. A related limitation is that our functioning measure, the SOFAS, did not allow us to investigate separate areas of functioning. Another limitation is that the design and the sample size of this study did not allow us to assess the effect of adjunct medication such as antidepressants or benzodiazepines on social cognition or functioning. Also, the small sample size of this study did not provide optimal



**Fig. 2.** Models testing for a potential moderation by comorbid social anxiety disorder of the direct and indirect effects of our target variables on functioning. Illustration of the results for our direct effect and indirect effect models, for which all parameters are presented in Table 3. A. Results for the model testing the direct effect of mentalizing on functioning, a significant direct relationship that is not moderated by the presence or absence of a comorbid social anxiety disorder (SAD). B. Indirect effect of social knowledge on functioning through mentalizing, a significant indirect effect that is not moderated by the presence or absence of a comorbid SAD. C. Indirect effect of social knowledge on functioning through mentalizing, showing no effect of our emotion recognition measure on our mentalizing task, and no moderation by the presence or absence of a comorbid SAD. D. Indirect effect of non-social reasoning on functioning through mentalizing, a significant indirect effect that is however significantly moderated by the presence or absence of a comorbid SAD. SAD = social anxiety disorder, a dichotomous variable that distinguishes SZ– and SZ+ patients. The parameters (a,  $a_w$ , b,  $b_w$ , c,  $c_w$  or  $c'$ ) are presented in Table 3 for all our models.

power to detect moderation effects and did not allow us to use structural equation modeling to assess all potential direct and indirect effects in a single, more comprehensive analysis.

In conclusion, our results support the distinction of the SZ– versus SZ+ groups and highlight the importance of taking the prevalent SAD comorbidity into account given that SZ+ patients differed from SZ– patients at least in terms of their pattern of social cognition performance and pathways to functional impact. Such deficits have become important targets for new treatments and our results provide potentially helpful information for the design of adapted treatment for SZ patients presenting with or without a comorbid SAD.

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.schres.2013.01.012>.

#### Role of funding source

This work was supported by a Canadian Institutes of Health Research (CIHR) post-doctoral fellowship to AMA and a CIHR operating grant (#MOP-77647) to MAR. PJ was supported by a salary grant from CIHR.

#### Contributors

Amélie M Achim was implicated in all phases of the project including the design of the study, the conception and validation of the tools used in this study, the testing and the analyses. She also wrote the manuscript.

Rosalie Ouellet was also implicated in the conception and validation of the social cognition test battery, she performed some testing and edited the manuscript.

Marie-Audrey Lavoie did some testing, helped with the analyses and edited the manuscript.

Chantal Vallières was implicated in the recruitment phase, she did some testing and edited the manuscript.

Philip L. Jackson was implicated in the project design, provided support for all aspects of the project and edited the manuscript.

Marc-André Roy was implicated in the design of the tools, particularly for clinical assessment, provided support throughout the project and helped AMA with the writing of the manuscript.

#### Conflict of interest

None.

#### Acknowledgments

We would like to thank the clinical and research team from Clinique Notre-Dame des Victoires for their support.

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