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Negative symptoms mediate the relationship between social cognition and functioning in schizophrenia: a pilot study

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Introduction

In spite of significant advances in pharmacological and psychological treatments, schizophrenia still ranks among the leading causes of disability worldwide (1).

People suffering from schizophrenia have significant impairment in major areas of everyday life, such as interpersonal relationships, work or school and even self-care. Poor functioning in schizophrenia has been associated with social cognition impairments and the presence of negative symptoms (2, 3).

Social cognition refers to the suite of cerebral mechanisms and mental operations that underlie social interactions, namely perceiving, interpreting, and generating responses to socially relevant stimuli, including the intentions, dispositions, and behaviors of others (4). It is typically broken down into four domains: emotion processing, social perception, attributional bias and theory of mind (5).

The concept of negative symptoms in Schizophrenia recently defined by the National Institute of Mental Health (NIMH) includes blunted affect, alogia, avolition, asociality, and anhedonia (6). In addition, studies investigating the factor structure of the construct have revealed that these symptoms usually cohere into two distinct, yet related, subdomains: diminished expression, consisting of affective flattening and alogia; and **amotivation**, consisting of avolition, asociality and hedonic deficits (7, 8).

Understanding the complex relationship between these determinants of real-world functioning in schizophrenia is crucial to drive delivered care into the ambitious and meaningful goal of recovery.

OBJECTIVE: The aim of this study was to **explore the relationships between** negative symptoms, social cognition and real-life functioning in people with schizophrenia.

Methods

PARTICIPANTS

15 patients (12; 80% male) with diagnosis of Schizophrenia according to ICD-10 criteria were recruited from the outpatient clinic of the Department of Psychiatry at Coimbra Hospital and University Centre. Mean duration of illness was $7.27 \pm 7,58$ years.

PROCEDURE/INSTRUMENTS

Patients were assessed cross-sectionally regarding relevant dimensions to our study using the following interview/self-report questionnaires:

Social Cognition

- Emotion Processing FEIT: Face Emotion Identification Test (9),
- Social Perception SPS: Social Perception Scale (10),
- Social Schema SCST-R: Schema Component Sequencing Test Revised (11),
- Social Attributions AIHQ: Ambiguous Intentions and Hostility Questionnaire (12),
- Theory of Mind RMET: Reading the Mind in The Eyes Test (13).

Negative Symptoms

CAINS: Clinical Assessment Interview for Negative Symptoms (8)

Psychosocial Functioning

PSP: Personal and Social Performance scale (14).

STATISTICAL ANALYSIS

SPSS 21.0 was used to perform Spearman correlations. Then, variables that proved to be both correlated with each other and with the main outcome (Psychosocial Functioning) were entered in a multiple mediation model, performed using PROCESS (15). Unconditional indirect effects were assessed via bootstrapping (5000 resamples).

Results

CORRELATIONS

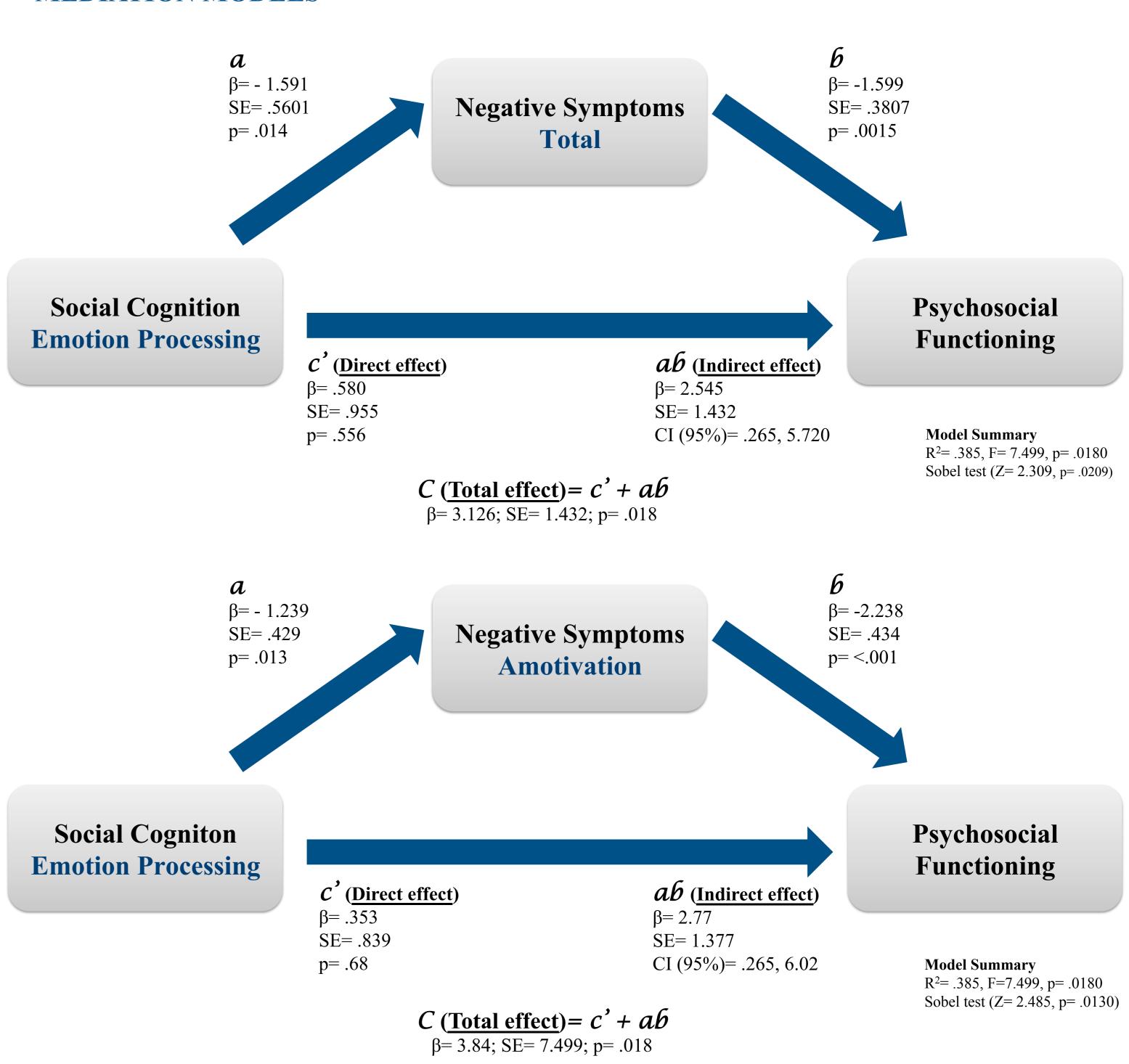
Spearman correlations are shown in Table 1.

Table 1: Spearman correlations

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		1	2	3	4	5	6	7	8	9
Negative Symptoms	1. Amotivation	1								
	2. Diminished Expression	.73**	1							
	3. Total	.98**	.81**	1						
Social Cognition	4. Emotion Processing	67**	Ns	75**	1					
	5. Social Perception	Ns	Ns	Ns	66*	1				
	6. Social Schema	Ns	Ns	Ns	Ns	Ns	1			
	7. Social Attribution	Ns	Ns	Ns	Ns	Ns	Ns	1		
	8. Theory of Mind	Ns	Ns	Ns	Ns	Ns	Ns	Ns	1	
Functioning	9. Psychosocial Functioning	87**	53*	76**	81**	.73**	Ns	Ns	.53*	1

**p<.01; p<.05; Ns: Non-significant

MEDIATION MODELS



N.B.: A model entering **Emotion Processing as a potential mediator** of the relationship between Negative Symptoms Total/Amotivation and Psychosocial Functioning was tested but was not significant: ab (Indirect effect, Negative Symptoms Total): β = -.1466; SE= .3715; CI (95%)= -.9027, .5617; ab (Indirect effect, Amotivation): β = -.1168; SE= .4296; CI (95%)= -.8861, 7408.

Conclusions and directions of future research

- Our results are in partial agreement with previous studies suggesting that both negative symptoms and impaired social cognition exert a deleterious impact on functional outcome.
- Emotion processing (social cognition) seems to drive negative symptoms, which play a role in mediating the deleterious impact of impaired social cognition on functional outcomes. Further exploration of this mediating role of negative symptoms has revealed that motivational deficits appear to be particularly important in explaining this relationship.
- Improving social outcomes seems to require a multi-faceted approach, which considers social cognition and negative symptoms.
- The complexity of the interplay between negative symptoms, social cognition and functioning should be addressed in future studies, as a greater understanding of underlying mechanisms is critical to development of effective treatments.
- These are preliminary results that need replication in a larger sample; this study is ongoing and we plan to provide more robust data in the near future.
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