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What is the Role of Social Cognition in the Relationship between Affect Regulation and Positive Symptom Severity in Psychotic Patients?

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Abstract

Impaired affect regulation has been associated with positive symptoms in patients suffering a psychotic disorder. Impaired social cognition may be a mechanism responsible for this association. This study examined the mediating and moderating effect of social cognition on the association between affect regulation and positive symptom severity. Eighty-five patients were included. Affect regulation was measured with the SOM-PSY ratio of the DSFM; social cognition was measured with the TAT (scored with the Social Cognition and Object Relations Scale) and the Hinting Task (HT); positive symptom severity was measured using the PANSS. The data collected was analysed using IMB SPSS version 25 with Hayes's PROCESS macro model 1 and 6. No mediating effect for none of the social cognition measures was found. This study did find that affect regulation was significantly associated with positive symptom severity but only with low levels of Theory of Mind(HT $\leq$ 17.56). So, instead of the expected mediation effect, a moderation effect of ToM on the relationship between affect regulation and positive symptom severity was found (this relation only accounts for 46% of the participants). Although more research is necessary to understand more about this moderation effect, it does implicate that addressing affect regulation in the treatment of psychotic patients may be effective as do stress-reducing interventions for psychotic patients scoring low on ToM tasks.

Keywords: psychosis, positive symptoms, social cognition, affect regulation, PSY-SOM ratio.

What is the Role of Social Cognition in the Relationship between Affect Regulation and Positive Symptom Severity in Psychotic Patients?

Suffering from a psychotic disorder, like schizophrenia and schizo-affective disorder, is a heavy burden, both for the patient, his/her environment and society. The health care costs of schizophrenia in the Netherlands alone are an estimated 835 million euros (Graaf, Have, & van Dorsselaer, 2010), not to mention other costs like social security costs due to the disabling character of many psychotic disorders. A study with over 30 thousand participants from all around the world found schizophrenia, the most common psychotic disorder, the most disabling disease in the world (Salomon et al., 2012). Based on a health screening (NEMESIS-2) of the Dutch population in 2010, an estimated 0,5% of the Dutch population in the range of 18 to 65 years of age had met the criteria for schizophrenia, schizophreniform disorder or schizoaffective disorder during the course of their lifetime (Graaf et al., 2010). Similar percentages were found in international studies, namely between 0,3-1,0% (Kahn et al., 2015; van Os & Kapur, 2009). Unfortunately much is still unknown about the nature and origin of psychotic disorders. Studying the pathogenic mechanisms of psychotic disorders, especially stress-related mechanisms like affect regulation and (impaired) social cognition, may improve understanding and treatment of psychotic disorder.

Psychotic disorders come with a range of symptoms, which are typically divided into three categories: positive symptoms, negative symptoms and cognitive impairments (American Psychiatric Association, 2000). The current study focuses on positive symptoms like hallucinations and delusions because they are generally viewed as the primary impairment in psychotic disorders. Negative symptoms and cognitive impairments occur in many other disorders, too (Andreasen et al., 2012).

Recent studies found that disrupted affect regulation may hold promise as a potential pathogenic mechanism in the aetiology of psychotic disorders. Gross (1999) refers to affect regulation as "ways individuals influence which emotions they have, when they have them and how they experience and express these emotions" (p.557). He suggests that affect regulation is on a continuum of conscious and unconscious processes, that vary in the degree to which they are regulated. A review of several daily diary method studies (Myin-Germeys & van Os, 2007) suggests that poor regulation of stress is an important vulnerability factor in the development of psychosis. Its authors concluded that there is convincing evidence that psychotic patients are emotionally more reactive to stress than non-patients (i.e., are less able to regulate their emotions when under stress, independent of cognitive impairments) and moreover that this increased stress reactivity is predictive of positive symptoms in psychotic patients. This is also supported by a study with 2524 adolescents and young adults that showed that poor affect regulation may contribute causally to the persistence and clinical relevance of reality distortion (van Rossum et al., 2009).

One way to regulate affect is to let mental stress flow out through the body (Eurelings-Bontekoe & Snellen, 2003). These authors suggest that the ratio between somatization[4] and psychopathology[5], can be seen as an affect regulation mechanism, in which psychological distress is translated into physical symptoms, so that they are not being "acted out" (Eurelings-Bontekoe & Koelen, 2007)[6]. A study showed that affect regulation through

[4] As measured by the somatization scale (SOM) of the Dutch Short Form of the MMPI (DSFM).

[5] As measured by the severe psychopathology scale (PSY) of the DSFM.

[6] Eurelings-Bontekoe and Snellen (2003) have developed "the theory driven interpretation of the DSFM". This is a model of structural diagnostics of personality characteristics that identifies underlying vulnerability factors that are independent of the diagnosed disorder. This method is mainly used to develop a working hypothesis on the structure of the personality and the inner dynamics of the individual patient, so clinicians can decide what treatment is best for the patient. Instead of interpreting the scales separately, Eurelings-Bontekoe and Snellen (2003) interpret the scales in combination with other scales based on psychodynamic theories of

somatization ensured that the most vulnerable patients with sleep disorders had fewer symptoms (Eurelings-Bontekoe, Koelen, Thijssen, de Ridder & Kerkhof, 2014). Eurelings-Bontekoe and Snellen (2003) also state that, to be able to talk about bodily symptoms, you need social cognitive capabilities, as emotional awareness, the ability to endure dependence on others and to mobilise help from others.

In addition to affect regulation, social cognition is another topic that is often associated with positive symptoms in psychotic disorders. Social cognition is a broad term and it is defined in various ways. A common definition is: the mental operations that underlie social interactions, including perceiving, interpreting, and generating responses to the intentions, dispositions, and behaviours of one's self and others (Green et al., 2008). Frith (1992) has suggested that impaired social cognition (impairments in the ability to represent mental states of self and others) may offer a potential explanation for (positive) psychotic symptoms. Due to this social cognitive impairment, psychotic patients have trouble discriminating the content of their own mind with that of the outside world. For example, delusions of persecution could be understood as misinterpretations of the intentions of other persons, guided by unrepresented suspiciousness; the inability to identify one's own thought as one's own can give rise to verbal hallucinations.

Two meta-analyses (Bora, Yucel, & Pantelis, 2009; Sprong, Schothorst, Vos, Hox, & van Engeland, 2007) have indeed shown significant impairments of social cognition in schizophrenic patients, for example impairments in the ability to understand indirect speech or the ability to understand that someone can hold a belief that is different from the actual

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personality. This theory-driven interpretation of the combination of scales exposes underlying vulnerability factors that cannot be determined by the individual scales alone. A study with 151 psychiatric patients found that the level of social cognitive capabilities depended on the personality organisation (Koelen, Eurelings-Bontekoe, van Broeckhuysen-Kloth, Snellen & Luyten, 2014).

state of affairs. Additionally, two articles reviewed the research on social cognition impairments in psychotic patients and found a link between social cognitive impairments and psychosis too (Debbané et al., 2016; Harrington, Siegert, & McClure, 2005).

Social cognition and affect regulation do not operate independently of each other. Fonagy and Bateman (2006) see social cognition as a versatile, dynamic ability, dependent on the capacity to regulate affect and stress. Social cognition is seen as a "higher order cognitive process", and like other higher cognitive functions, sensitive to excessive stress (Bateman & Fonagy, 2016). When the amount of stress exceeds social cognitive capabilities, "lower order cognitive processes" take over and produce affect-driven responses at the expense of more thoughtful responses (Arnsten, 2009). Fonagy and Luyten (2009) call these processes "pre-mentalizing modes". According to them, one form of a pre-mentalizing mode, especially pertinent to psychotic disorders, is the "psychic equivalence" mode, in which both inner and outer reality are treated as identical. If affect regulation mechanisms fall short and can no longer regulate the exceeding stress, the social cognitive capabilities do not function adequately and this increase the possibility to relapse into "psychic equivalence". Myin-Germeys and van Os (2007) describe a similar process. Poorly regulated affect, triggered by stressful events, activates biased evaluation processes and inappropriate adaptation of self / other schemes that leads to an externalizing attribution, preceding positive psychotic symptoms. These biased evaluation processes and inappropriate adaptation of self / other schemes can be seen as impairments in social cognition.

The mediating role of social cognition on the relationship between poor affect regulation and symptoms has previously been found in a study with adolescents with borderline traits (Sharp et al., 2011). The study found that when stress rises, the symptoms get worse because the stress exceeds social cognitive capabilities to regulate the stress. Even though borderline personality disorder (BPS) and psychotic disorders may seem quite

different, patients with BPS often experience psychotic symptoms (Auerbach, & Blatt, 1996: Yee, Korner, McSwiggan, Meares, & Stevenson, 2005). A study on both BPS and psychosis found that 17 % of the participants met the criteria for both disorders (Kingdon et al., 2010). Therefore, it is not unlikely that this mediating role of social cognition is present in psychotic disorders as well.

Concluding, research shows that poor affect regulation is associated with both positive symptom severity and impaired social cognition and that impaired social cognition is also associated with positive symptom severity. In a study with borderline patients, a mediating role of social cognition in the relationship between affect regulation and symptom severity was found (Sharp et al., 2011). However, to date no study has investigated the mediating role of social cognition in the relationship between (poor) affect regulation and positive symptoms. Insight into these processes can help to determine the focus of treatment.

In this study, we investigate what the role of social cognition is in the relationship between affect regulation and positive symptoms. Social cognition as a mediator could explain the negative relationship between affect regulation and positive symptoms. This means that we expect that: (1) there is a negative relationship between affect regulation and positive symptom severity; (2) affect regulation is positively related to social cognition; (3) social cognition is negatively related to the severity of positive symptoms; (4) social cognition as mediator explains (part of) the relation between affect regulation and positive symptom severity. If no mediating role is found, it is also possible that social cognition plays a moderating role. This means that the negative relationship between affect regulation and symptoms depends on the degree of social cognition. So, the last hypothesis is: (5) there is no moderation effect of social cognition on the negative relation of affect regulation on positive symptom severity.

Method

Participants

Participants are 90 patients of various outpatient sites of the Rivierduinen mental health institute and the Altrecht Institute for Mental Health Care in Zeist, The Netherlands. Patients are diagnosed with a psychotic disorder (DSM-IV (Diagnostical and Statistical Manual of Mental Disorders 4th edition) criteria) (American Psychiatric Association, 2000): schizophreniform, or schizoaffective disorder; delusional disorder; brief psychotic disorder; or psychotic disorder not otherwise specified. On page 14, sociodemographic and DSM-IV characteristics of the sample are shown in table 1.

Inclusion criteria:

- At least 6 months of prior treatment.
- No more than 10 years of treatment for psychotic disorders.
- Between 18 and 55 years of age.

Exclusion criteria:

- Intellectual disability and/or illiteracy.
- A lack of mastery of the Dutch language.
- Substance abuse to such an extent that inpatient detoxification is necessary. After detoxification the patient can participate.

Trial design

This study is a cross-sectional study into the role of social cognition in the relationship between independent variable; affect regulation and dependent variable; severity of positive symptoms. Social cognition is the mediator/moderator. Two covariates are included, namely age of onset and gender. The measures are self-assessment questionnaires as well as observer-rated, (semi)structured interviews.

Measurements and instruments

Affect regulation. Affect regulation is measured by the combination of the scales somatization (20 items) and severe psychopathology (13 items) of the Dutch Short Form of the MMPI (the Minnesota Multiphasic Personality Inventory) (DSFM) (Eurelings-Bontekoe, Onnink, Williams, & Snellen, 2008). The DSFM is a self-assessment questionnaire of 83 items divided into five scales representing 5 personality traits. All items can be answered with an 'agree' (2 points) or 'disagree' (0 points). Luteijn and Kok (1985) reported α coefficients between .76 and .83 for the somatization scale; and between .82 and .92 for the severe psychopathology scale. Somatization (SOM) measures the amount and degree of experienced bodily symptoms, and the ability to report and be aware of bodily symptoms. Severe psychopathology (PSY) measures anxiety tolerance and psychoticism. High scores on the PSY scale are hypothesized to point to an increased disposition to develop aggression, fear and suspicion with increasing stress. It is favourable if the body is able to regulate these affects by translating them in bodily symptoms instead of acting out aggression, fear and suspicion. Therefore, affect regulation is better if a high score on PSY goes together with a high score on SOM meaning that the participant is better able to translate psychological distress in bodily symptoms .

The theory driven profile approach distinguishes an unfavourable combination of SOM-PSY and a favourable combination of SOM-PSY (Eurelings-Bontekoe, & Koelen, 2007).

Unfavourable is:

- A PSY score of 0-3 (low) with a SOM score of 7 or lower (low), or
- A PSY score of 4-7 (average) with a SOM score of 11 or lower (below average), or
- A PSY score of 8 or higher (high to very high) with a SOM score of 23 or lower (above average and lower).

Favourable is:

- A PSY score of 0-3 (low) with a SOM score of 12 (below average) and higher, or
- A PSY score of 4-7 (average) with a SOM score of 19 (above average) and higher, or
- A PSY score of 8 (high to very high) or higher with a SOM score of 24 (high) or higher.

A frequently heard point of criticism on the theory driven interpretation of the DSFM is that, due to the categorization, a small difference in the score can make a big difference in the interpretation (Smid & Kamphuis, 2005). This is why we have chosen to make the dichotomous SOM-PSY combination a continuous variable. In order to make this combination a continuous variable, a ratio between SOM and PSY is calculated. Because it is not possible to calculate a ratio with scores of 0, both scales are first recoded with +1. The ratio is then calculated for every participant by dividing the SOM score with the PSY score. The higher the ratio between the two scales the more favourable the affect regulation is.

Positive symptom severity. A Dutch version of the Positive And Negative Syndrome Scale (PANSS) (Kay, Fiszbein, & Opfer, 1987) is used to assess positive psychotic symptom severity. This is an observer-rated instrument developed for the assessment of phenomena associated with schizophrenia during the two weeks prior to measurement. There are 30 items on a 7-point Likert scale. Subscale P (positive phenomena, seven items) is used measuring the following positive symptoms: delusions; conceptual disorganisation; hallucinations; excitement; grandiosity; suspiciousness and hostility. Inter-rater reliability was assessed by rating a taped interview by all observers and is considered high for the average of PANSS items (with an interclass correlation coefficient of 0.91).

Social cognition. Most research on social cognition is done with ToM (Theory of Mind) tasks (Debbané, 2016). However, additional aspects of social cognition might be relevant here as well. This is why this study assesses social cognition with two different

instruments. The Hinting Task (HT) is used to measure Theory of Mind (ToM), the ability to derive intentions from others (Corcoran, Mercer, & Frith, 1995). The test presents participants with 10 different interactions between 2 persons in which one person says something to the other and the participant has to decipher the implicit message. 2 points are given to each right answer of the participant, 1 point if a hint is needed and 0 points if the answer is incorrect. The HT is found to be a good measure with good evaluations of test-retest reliability, internal consistency, utility as a repeated measure and relation to functional outcome (Pinkham, Penn, Green, & Harvey, 2015).

The Thematic Apperception Test (TAT) as scored with the Social Cognition and Object Relations System (SCORS) by Westen (1995), has been proposed to measure most aspects of social cognition (Luyten, Fonagy, Lowyck, & Vermote, 2012). Six pictures of the TAT are shown to the participants, each with four questions. Their answers have been recorded and transcribed verbatim. These transcripts are scored with the SCORS, a narrative-based measure to assess four dimensions of social cognition: complexity of representations of people, understanding of social causality, affect-tone of relationships and the capacity for emotional investment. Each dimension is scored on a 5-point scale, with higher scores representing higher social cognitive functioning in that dimension.

The first two scales cover the cognitive aspects of social cognition and the last two scales cover the affective aspects of social cognition. Research (Shamay-Tsoory, Shur, Barcai-Goodman, Medlovich, Harari, & Levkovitz, 2007) shows that there is a difference in performance on affective and cognitive aspects of social cognition tasks in psychotic patients. Especially the affective aspects of social cognition are impaired in these patients. The TAT, scored with the SCORS, is found to be a valid and reliable way to measure social cognition (Hibbard, Mitchell, & Porcerelli, 2001; Meyer, 2004).

For the mediation analysis, the measures for social cognition are separated into 3 social cognition mediators: ToM, measured by the HT; the cognitive aspects of social cognition, measured by subscales complexity of representations of people and understanding of social causality of the TAT; and the affective aspects of social cognition, measured by subscales affect-tone of relationships and the capacity for emotional investment of the TAT.

For the moderation analyses, the TAT is excluded[7] from this analysis and only ToM, measured by the HT, is used as a social cognition moderator.

Demographic and illness parameters. Type of DSM-IV diagnose and age of onset are assessed with two items of the Comprehensive Assessment of Symptoms and History (CASH) and they address the DSM-IV diagnose, the period since first psychosis and the age of the first psychiatric problems. The CASH is a semi-structured interview assessing signs, symptoms and history of psychotic, manic and depressive syndromes and substance abuse. Gender and age are assessed with two items of the General Demographic Questionnaire (GDQ). This is an instrument to measure general demographics.

Analyses

A mediator and a moderator model was tested using IBM SPSS version 25 with Hayes's PROCESS macro model 1 and 6 (Hayes & Preacher, 2014). Affect regulation is the independent variable, as measured by a ratio of the somatization and the severe psychopathology scales of the DSFM. Positive symptom severity is the dependent variable, as measured by the positive phenomena scale of the PANSS. Social cognition is divided into three mediators for the mediation analyses: HT measuring ToM; two subscales of the TAT measuring the cognitive aspects of social cognition; and the other two subscales of the TAT

[7] Because of the lack of correlation between the cognitive and affective aspects of social cognition and the other variables and because a moderator analysis with 3 moderators is impossible within PROCESS, the TAT is excluded from the moderation analysis.

measuring the affective aspects of social cognition. For the moderator analyses, social cognition is only measured by HT[8], measuring ToM.

The mediation hypothesis is supported if: 1) there is a significant negative relation between affect regulation, ignoring the effect of social cognition, and positive symptom severity; 2) there is a significant positive relationship between affect regulation and social cognition; 3) there is a significant negative relationship of social cognition, controlling for affect regulation, on positive symptom severity; and 4) requisite 1,2 and 3 are met and the relationship of affect regulation, on positive symptom severity while taken into account the effect of social cognition, is significantly smaller than the relationship of affect regulation and positive symptom severity, ignoring the effect of social cognition.

The hypothesis of no moderation can be supported if: 5) there is no significant indirect effect of affect regulation with social cognition on positive symptom severity.

Possible confounders. Studies have shown that females have better social cognitive capacities then men (Krach et al., 2009). This is also found in patients with schizophrenia (Abu-Akel & Bo, 2013). A review of recent studies on the age of onset of the major mental illnesses suggest that the severity of psychosis is associated with earlier onset (McGorry, Purcell, Goldstone, & Amminger, 2011).

Results

In this study we investigated the role of social cognition in the relationship between affect regulation and positive symptoms.

Sample characteristics

Two participants were excluded because they dropped out before completing all questionnaires and three participants did not answer all the questions necessary for this study.

[8] Because of the lack of correlation between the cognitive and affective aspects of social cognition and the other variables and because a moderator analysis with 3 moderators is impossible within PROCESS, the TAT is excluded from the moderation analysis.

They were excluded from analyses. The final sample used for analysis consisted of 85 participants. The demographics are shown in table 1.

Table 1 Sociodemographic and DSM-IV characteristics of sample (N = 85)

Variable	Mean ± *SD*	Range
Age	31.6 ± 8.6	19 - 54
Age of first psychosis	26.1 ± 8.3	11 - 49
Variable	*n*	Percentage
Sex		
Male	55	64.7
Female	30	35.3
DSM-IV diagnosis		
Schizophrenia	54	63.5
Psychotic disorder NOS	13	15.3
Schizoaffective disorder	12	14.1
Brief psychotic disorder	4	4.7
Delusional disorder	2	2.4

Assumptions

Because for the mediation analysis other outliers have to be excluded from the data than for the moderation analysis, we first checked the assumptions with the complete data. After checking the assumptions for the complete data and after excluding the outliers relevant for each analysis individually, the data is checked again for assumptions.

The assumptions of multivariable normality (exceeding kurtosis: SOM-PSY ratio 9.3; cognitive aspects of social cognition, 5.1 and exceeding skewness: positive symptom severity, 1.21; ToM, -.96; age of onset, .82; and SOM-PSY ratio, 2.67) and homoscedasticity are violated. This means that the variables: positive symptom severity; ToM; age of onset; SOM-PSY ratio; and the cognitive aspects of social cognition are not normally distributed and the errors are not random.

In table 2 the correlations of the variables used in this study are shown. Positive symptom severity is only significantly related to affect regulation and age of onset and the affective aspects of social cognition are significantly related to the cognitive aspects of social cognition.

Table 2 Correlations of variables

	Positive symptom severity	Affect regulation	ToM	Cognitive aspects of social cognition	Affective aspects of social cognition	Age of onset
Positive symptom severity	1	-.33*	NS	NS	NS	.28*
Affect regulation		1	NS	NS	NS	NS
ToM			1	NS	NS	NS
Cognitive aspects of social cognition				1	.43**	NS
Affective Aspects of social cognition					1	NS
Age of onset						1

NS = not significant
*=p <.01
**= p < .001

Table 3 shows the results of the t-tests of the differences between men and women on the different variables. Only the difference between men and women on the positive symptoms severity is significant, with men having more positive symptoms than women.

Table 3 Comparing means of men and women on variables

		Gender			
		Men	Women	*t*-value	*p*
Positive symptom severity	M	10.13	12.40	2.35	.02
	SD	3.46	4.63		
Affect regulation	M	3.74	2.88	-1.26	.21
	SD	2.92	3.07		
ToM	M	17.63	16.76	-1.55	.13
	SD	1.83	2.76		
Cognitive aspects of social cognition	M	24.57	23.27	-1.86	.07
	SD	3.37	2.88		
Affective Aspects of social cognition	M	29.13	28.89	-.27	.79
	SD	4.17	3.84		

Age of onset	M	27.87	25.20	-1.42	.16
	SD	8.34	8.20		

Mediation analysis

Assumptions. An analysis of outliers was made resulting in the removal of three participants. The assumptions of multivariable normality (exceeding skewness of the variables: positive symptom severity, 1.1; ToM, -.92; and age of onset, .85 and exceeding kurtosis of the cognitive aspects of social cognition, 4.6) and homoscedasticity are violated. This means that the variables: positive symptom severity; ToM; age of onset; and cognitive aspects of social cognition are not normally distributed and the errors are not random.

Relationships. 1. There is a significant negative relationship between affect regulation and positive symptom severity, ignoring the effect of social cognition (b = -.36, t(3,78)= -2.10, p = .04,). The hypothesis that there is a negative relationship between affect regulation and positive symptom severity is supported.

2. There is no significant positive relationship of affect regulation and ToM (b = .19, t(3,78)= 1.68, p = .10), nor with the cognitive aspects of social cognition (b = .09, t(4,77)= .71, p = .48), nor with the affective aspects of social cognition (b = -.03, t(5,76)= -.18, p = .85). The hypothesis that affect regulation is positively related to social cognition is rejected.

3. ToM, controlling for affect regulation, is not negatively related to positive symptom severity (b = -.13, t(6,75)= -.74, p = .46), nor do the cognitive aspects of social cognition, controlled for affect regulation, relate negatively to positive symptom severity (b = -.06, t(6,75) = -.31, p = .76), nor do the affective aspects of social cognition, controlled for affect regulation, relate negatively to positive symptom severity (b = -.11, t(6,75) = -.88, p = .38). The hypothesis that social cognition is negatively related to the severity of positive symptoms is rejected.

4. Hypotheses 2 and 3 are not supported so there is no mediation effect for social cognition on the relation between affect regulation and positive symptom severity. This is confirmed by the lack of a significant indirect effect ((b = -.04, confidence interval 95% [.08, -.23]), the confidence interval contains 0, meaning that there is no significant indirect effect). The hypothesis that social cognition as mediator explains (part of) the relation between affect regulation and positive symptom severity is rejected.

Moderator analyses

Because the lack of correlation between the cognitive and affective aspects of social cognition and the other variables and because a moderator analyses with 3 moderators is impossible within PROCESS, the TAT is excluded from this analysis.

Assumptions. An analysis of outliers was made resulting in the removal of two participants. The assumptions of multivariable normality (exceeding skewness of the variables: positive symptom severity, .99; and SOM-PSY ratio, 1.91 and exceeding kurtosis of the variable: SOM-PSY ratio, 3.7) and homoscedasticity are violated. This means that the variables: positive symptom severity and SOM-PSY ratio are not normally distributed and the errors are not random.

Relationships. 5. There is a moderation effect ($F(5,77) = 6.98$, $p < .001$, $R^2 = .31$ (the interaction effect accounts for 4% of the variance)) for social cognition measured by HT, suggesting that ToM changes the strength of the relation between affect regulation and positive symptom severity. Affect regulation ($b = -3.62$, $t(77) = -2.41$, $p = .02$) is negatively related to positive symptom severity. ToM does not relate to positive symptom severity ($b = -.40$, $t(77) = -1.35$, $p = .18$). The interaction effect is $b = .19$, $t(77) = 2.21$, $p = .03$. Addition of the interaction was a change in the model of $F(1,77) = 4.87$, $p = .03$, R^2 change is = .04. With low (= 15) ($b = -.80$, $t(77) = -2.96$, $p = .004$) HT scores, affect regulation relates negatively to positive symptom severity, but not with average (= 18) ($b = -.24$, $t(79) = -1.44$, $p = .15$) or

high (=19.56) (b = .05, $t(77)$ = .22, p = .83) HT scores. With HT scores of 17.56 or lower (46% of the participants have a HT score of 17.56 or lower), affect regulation related negatively to positive symptom severity. The hypothesis that there is no moderation effect of social cognition on the negative relation of affect regulation on positive symptom severity is rejected.

Discussion

This study investigated what the role of social cognition is in the relationship between affect regulation and positive symptoms. This study did not find the expected mediating effect of social cognition on the relation between affect regulation and psychotic symptom severity. However, it did find a moderation effect, showing that affect regulation is negatively related to positive symptom severity for those with low levels of ToM.

There was a negative relationship between affect regulation and positive symptom severity, supporting the first hypothesis, but affect regulation was not positively related to social cognition, rejecting the second hypothesis. Social cognition was not negatively related to positive symptom severity, rejecting the third hypothesis. This means that the requisites for a mediation analysis were not met, rejecting the fourth hypothesis. Although not expected, there was a moderating effect of (a part of) social cognition on the negative relationship between affect regulation and positive symptom severity, but only with low levels of ToM (HT $\leq$ 17.56 (this relationship only applies to 46% of the participants)). The more unfavourable the affect regulation is, the more severe the positive symptoms are, with participants scoring low on social cognition, so the last hypothesis that there is no moderation effect is also rejected.

The current results replicate previous findings with reference to the negative relationship of affect regulation and positive symptoms (Myin-Germeys & van Os, 2007; van Rossum et al., 2009) and support the theory that poor affect regulation is related to symptom

severity (Eurelings-Bontekoe & Snellen, 2003; Eurelings-Bontekoe & Koelen, 2007; Eurelings-Bontekoe et al., 2014). The results do not find a relation between unfavourable affect regulation and social cognition as suggested by different researchers (Arnsten, 2009; Eurelings-Bontekoe & Snellen, 2003; Fonagy & Bateman, 2006; Fonagy & Bateman, 2016). An explanation for this discrepancy may be a mismatch in measurement level. The measures of social cognition used in this study are associated with the more explicit processes of affect regulation whereas the translation of psychological distress in bodily symptoms, measured by the SOM-PSY ratio, is a more implicit, self-oriented process of regulating affect. A different measurement of affect regulation like the Emotion Regulation Questionnaire (ERQ) (Gross & John, 2003) may show other results, more in line with the expectations. This study did not replicate the negative relation between social cognition and positive symptom severity as suggested by Frith (1992) and showed by meta-analyses (Bora et al., 2009; Sprong et al.,2007) and reviews (Debbané et al., 2016; Harrington et al., 2005). Maybe this is because social cognition is a very broad construct with a lot of different aspects (Fonagy and Luyten, 2009), and not all aspects of social cognition are related to functional outcome (Couture, Penn, & Roberts, 2006) or to each other (Fonagy & Bateman, 2016). Other measures for social cognition may find the expected relations.

Further research on the role of other measures of social cognition on the relationship between poor affect regulation and positive symptom severity is recommendable. Because there was no relation between affect regulation and social cognition nor a relation between affect regulation and positive symptom severity, the requisites for a mediation analysis were not met and therefore, there is no mediation effect of social cognition on the relation between poor affect regulation and positive symptom severity as suggested by Bradley (2003), Myin-Germeys and Os (2007) and Bateman and Fonagy (2012). It may be that this is because the development of the psychotic disorder is on a different level of development for each

participant. Depending on how developed the psychotic disorder is, the effect social cognition has on the relationship between poor affect regulation and positive symptoms could be different (Bradley, 2003). Even calculating the duration of illness, it is difficult to know where the participant is on the development because it is assumed that the development depends on the reactions of the environment of the participant and that is not measured. This is supported by a study that found a relation between expressed emotions of family members and positive symptoms (Cechnicki, Bielańska, Hanuszkiewicz, & Daren, 2013). A recommendation for further research is to include a measure for expressed emotion, follow the development of social cognition over time and stage the development of the disorder using a staging method as suggested by McGorry, Nelson, Goldstone and Yung, (2010). This may reveal a critical level in development of the psychotic disorder in which social cognition does mediate the relationship between affect regulation and social cognition.

There were no previous findings on a moderation effect of social cognition on the relation between affect regulation and positive symptom severity as found in this study. Debbané et al. (2016) did suggest social cognition as a moderator, protecting patients from the full onset of psychosis. This is supported in a study of Bartels□Velthuis, Blijd□ Hoogewys and van Os (2011), showing that social cognitive capacities reduced psychotic-like symptoms in youths. In another article (Debbané et al., 2016), the authors propose that impaired social cognition, especially self-monitoring deficits, has a moderating effect on the relation between stress and reality testing. Further research on a moderation effect of social cognition as a possible underlying mechanism between affect regulation and positive psychotic symptom severity is recommendable.

There are some limitations in this study. First, many assumptions were violated, impacting the ability to trust the results. However, using bootstrapping, no assumptions about the shape of the sampling distribution are necessary (Preacher, Rucker, &

Hayes, 2007). Nevertheless, the results have to be interpreted with caution. Second, although it is a clinically relevant group, the sample is small (85 participants) for the number of predictors (6), which may impact power of the analysis. But, Shrout and Bolger (2002) suggest that with small samples, bootstrapping is a good method to assess mediation. Third, the SOM-PSY ratio is not a commonly used measurement of affect regulation in science, although the interpretation of combining scales of the DSFM is often successfully used in clinical practice to predict treatment success (Mosterman, Eurelings-Bontekoe, & Hofstee, 2008). Fourth, most of the participants of this study have different pharmacotreatment histories that may have different effects on social cognition, affect regulation and positive symptom severity. This may make it difficult to measure the "pure" social cognition, affect regulation and positive symptom severity. For example, a study on the effects of antipsychotics on social cognition as well as on psychotic symptoms, found that antipsychotics effected both social cognition as well as psychotic symptoms, but independently (Mizrahi et al. , 2007).

Last, in the course of the research, it has become clear that the use of the TAT in this target group may not have been the right choice. To interpret the pictures of the TAT, imagination is important. In order to properly imagine, it is necessary to let your thoughts run free. We have noticed in the PANSS assessment that psychotic people are afraid of their own thoughts and have learned to ignore them. One patient formulated: "When suspicious thoughts arise I immediately think: "But I do not want to be like that", and then I ignore these thoughts". Thought suppression is a coping strategy used for thoughts with a high chance of social disapproval (Freeston & Ladouceur, 1993: Purdon & Clark, 1994), often used among psychotic patients (Shergill, Murray, & McGuire, 1998). This is probably the reason that patients invested little in the TAT and therefore there was little variation in the scores and

thus explaining the high correlation between the cognitive and affective aspects of social cognition as well as the lack of correlation with the other variables.

Although the current study has not been able to reveal a mediating effect of social cognition for the relation between affect regulation and psychotic symptom severity, it did show that affect regulation is negatively related to positive symptom severity for those with low levels of ToM (46% of the participants). With low levels of ToM (HT $\leq$ 17.56), affect regulation does predict positive symptom severity. Although further research is necessary, this supports the use of interventions addressing affect regulation and social cognition during the treatment of psychotic patients scoring low on ToM tasks. For this reason, Mentalization Based Therapy (MBT) (Brent, Holt, & Keshaven, 2014), may be a good treatment for those psychotic patients, because MBT focuses on reflecting on emotions while they are being experienced, instead of after they have been experienced, with the aim to improve affect regulation to a more favourable level. In addition, it also supports the deployment of family intervention programs (Pharoah, Mari, Rathbone, & Wong, 2010) where the focus is on reducing stress in the social environment of patients. Reducing stress, however, is in contradiction with the shift in mental health care, where more pressure is placed on patients on being able to participate in work for example. The question is whether this is not too stressful for this vulnerable group of patients.

Competing interests. The author declares that she has no competing interests.

Consent for publication. Consent to publish results and store data and materials for a maximum of 15 years is obtained from all participants. Consent to share raw data with outside parties was not be obtained.

Ethical considerations. This study is part of a study, approved by the Medical Research and Ethics Committee (MREC) of Maastricht University, registered under NL

47236 068/METC 13-03-066. Consent to participate is obtained from all participants of whom the data is used.

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