

Evaluation the effect of cognitive-behavioral psychotherapy skills on emotional intelligence

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Abstract: Cognitive-behavioral is a new developmentally sensitive psychosocial intervention for pediatric bipolar disorder that is intended for use along with medication. Participants included 34 male and female (mean age 12.20 years, SD = 2.98) who were treated in a specialty clinic. All higher quality studies reported positive effects favoring the cognitive-behavioral treatment program. Specifically, positive reductions in recidivism were observed for moral recognition therapy, reasoning and rehabilitation, and various cognitive-restructuring programs. The evidence suggests the effectiveness of cognitive skills and cognitive restructuring approaches as well as programs that emphasize moral teachings and reasoning.

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1. Introduction

Cognitive behavioral therapy (CBT) is a psychotherapeutic approach that addresses dysfunctional emotions, maladaptive behaviors and cognitive processes and contents through a number of goal-oriented, explicit systematic procedures. The name refers to behavior therapy, cognitive therapy, and to therapy based upon a combination of basic behavioral and cognitive principles and research. Most therapists working with patients dealing with anxiety and depression use a blend of cognitive and behavioral therapy. This technique acknowledges that there may be behaviors that cannot be controlled through rational thought. CBT is "problem focused" (undertaken for specific problems) and "action oriented" (therapist tries to assist the client in selecting specific strategies to help address those problems).

CBT is thought to be effective for the treatment of a variety of conditions, including mood, anxiety, personality, eating, substance abuse, tic, and psychotic disorders. Many CBT treatment programs for specific disorders have been evaluated for efficacy; the health-care trend of evidence-based treatment, where specific treatments for symptom-based diagnoses are recommended, has favored CBT over other approaches such as psychodynamic treatments.

CBT was primarily developed through an integration of behavior therapy (the term "behavior modification" appears to have been first used by Edward Thorndike) with cognitive psychology research, first by Donald Meichenbaum and several other authors with the label of cognitive behavior modification in the late 1970s. This tradition thereafter merged with earlier work of a few clinicians, labeled as Cognitive Therapy (CT),

developed by Aaron Beck, and Rational Emotive Therapy (RET) developed by Albert Ellis. While rooted in rather different theories, these two traditions have been characterised by a constant reference to experimental research to test hypotheses, both at clinical and basic level. Common features of CBT procedures are the focus on the "here and now", a directive or guidance role of the therapist, a structuring of the psychotherapy sessions and path, and on alleviating both symptoms and patients' vulnerability.

CBT has six phases:

- Assessment or psychological assessment;
- Reconceptualization;
- Skills acquisition;
- Skills consolidation and application training;
- Generalization and maintenance;
- Post-treatment assessment follow-up

Social functioning impairment is a hallmark of schizophrenia that is not amenable to most common forms of treatment (Swartz et al., 2007). Elucidating determinants of social functioning are thus a critical step in the development of effective interventions. Research in this area however, may be limited by a lack of information regarding the neural mechanisms that give rise to social impairment. Such information may elucidate the neurocognitive processes and neural substrates to be targeted for remediation that will most likely result in functional improvements.

Theory of mind (ToM), or the ability to attribute and reason about the mental states of others, is markedly impaired in individuals with schizophrenia (Bora et al., 2009 and Sprong et al., 2007). Critically, among individuals with schizophrenia, these deficits have been consistently linked to aspects of social functioning (Couture et al., 2006, Fett et al., 2011 and Horan et al., 2012), are more proximal to daily

functioning (Bora et al., 2006, Couture et al., 2011 and McGlade et al., 2008), and account for more of the variance in daily functioning than other aspects of the illness, including non-social aspects of cognition and symptoms (Brune et al., 2007, Fett et al., 2011, Kosmidis et al., 2011 and Pinkham and Penn, 2006). Though the neural bases of ToM have been fairly well characterized in healthy adults, encompassing right and left temporo-parietal junctions (RTPJ, LTPJ) and medial prefrontal cortex (MPFC) (Mar, 2011 and Van Overwalle, 2009), less is known about the ToM network in individuals with schizophrenia. The extant literature suggests functional and anatomical abnormalities in MPFC (Benedetti et al., 2009, Brune et al., 2011, Brunet et al., 2003, Das et al., 2012, Hooker et al., 2011, Lee et al., 2006, Lee et al., 2011, Walter et al., 2009a and Yamada et al., 2007). However, findings are inconsistent, and, surprisingly, few such studies investigate the relationship between neural activity for ToM and social functioning. Such data could provide biomarkers of social dysfunction, which may prospectively predict schizophrenia outcomes, and neural targets for remediation. Thus, the first aim of the current study was to characterize the neural bases of ToM in schizophrenia and investigate their relationship to aspects of social behavior using multiple methods of assessment.

Social anhedonia, characterized by a trait-like disinterest and lack of pleasure from social interaction, is another important determinant of social functioning in individuals with schizophrenia (Blanchard et al., 1998 and Cohen et al., 2005) and the general population (Blanchard et al., 2011). Individuals with high levels of social anhedonia, which is an enduring feature of schizophrenia-spectrum disorders (Blanchard et al., 1998, Blanchard et al., 2001 and Horan et al., 2008), are more likely to be socially isolated (Brown et al., 2007 and Kwapil et al., 2009), report less social support and social coping (Blanchard et al., 2011 and Horan et al., 2007), greater levels of perceived stress (Horan et al., 2007), and worse functioning within the family unit including less family cohesion, support, and more conflict (Blanchard et al., 2011). Much of the extant literature concerning the underlying mechanisms connecting social anhedonia to these aforementioned consequences for social functioning has focused on the role of diminished anticipatory reward for future social interaction (Barch and Dowd, 2010, Gard et al., 2007, Horan et al., 2008 and Kring and Elis, 2012), which may be a consequence of difficulty representing reward value and subsequently accessing these representations due to impairments in working and episodic memory (Gold et al., 2008 and Strauss and Gold, 2012). These findings have led to suggestions that targeting reward-related neural

circuitry, possibly through pharmacological means, may ameliorate symptoms of anhedonia and subsequently improve functional outcome (Juckel et al., 2006a, Juckel et al., 2006b, Schlagenhauf et al., 2008, Walter et al., 2009b and Waltz et al., 2007).

An alternative uninvestigated possibility that would carry important treatment implications is that social anhedonia impacts social functioning, at least partially, through its consequences on neural circuitry supporting social cognitive processes, such as ToM. Substantial evidence exists that repeated engagement in cognitive skills, as in cognitive remediation treatments for schizophrenia, improves neural function due to the brain's capacity for reorganization in response to environmental input (Buonomano and Merzenich, 1998, Eack et al., 2010, Hooker et al., 2012, Penades et al., 2013 and Subramaniam et al., 2012). Likewise, both animal and human studies have demonstrated that failure to engage in cognitive skills, via social deprivation, for example, can produce profound neurobiological alterations (Barr et al., 2004, Chugani et al., 2001, Geyer et al., 1993, Kaufman et al., 2000 and Suomi, 1997). Considering these findings in the context of social anhedonia, social isolation may remove opportunities to engage in ToM and this lack of engagement may precipitate a cascade of aberrant neuroplastic events that result in disruption to neural networks supporting social cognition (Hoffman, 2007). These disrupted neural networks may, in turn, manifest as difficulty in inferring the intentions and emotions of others, which then contributes to the interpersonal difficulties, compromised social networks, and further isolation that characterizes social functioning impairment in schizophrenia and individuals with high levels of social anhedonia.

If disruption to ToM-related neural circuitry accounts for the relationship between social anhedonia and social functioning, it would suggest ToM as a more proximal process to social functioning than social anhedonia. This has significant implications for schizophrenia as it means that neurocognitive improvements in ToM may more directly support improved social functioning. In other words, it would suggest ToM as a more effective treatment target than social anhedonia or anhedonia more broadly. Furthermore, the early presence of social anhedonia and accompanying withdrawal pre-illness onset (Gooding et al., 2005, Kwapil, 1998 and Tarbox and Pogue-Geile, 2008), and its temporal stability (Blanchard et al., 1998 and Blanchard et al., 2001), suggest that social anhedonia could be a marker of risk for disruption to ToM-related neural circuitry and subsequent social dysfunction. Thus, engaging and training ToM processes early in development in those exhibiting social withdrawal

may help to prevent social dysfunction (Cornblatt et al., 2012 and Tarbox and Pogue-Geile, 2008). In consideration of these issues, our second aim was to investigate this proposed relationship; that is, whether neural activity for ToM accounts for the link between social anhedonia and social functioning.

We addressed these aims using a well-validated ToM scanner task ubiquitously employed in the social neuroscience literature: the False-Belief Task (Saxe and Kanwisher, 2003). Between-group differences were examined in a priori regions-of-interest (ROIs) identified from the ToM literature and with whole-brain analyses. The use of ROIs increases the certainty that the neural activity under examination relates to the process of mental state attribution as opposed to illness-related deficits in peripheral cognitive processes (Poldrack, 2006 and Saxe et al., 2006). Furthermore, we investigate how neural activity for ToM relates to different aspects of social behavior (i.e., trait empathy/perspective taking, the ability to manage emotions in different social contexts, the quantity/quality of social interaction in a variety of social roles) with multiple methods of assessment (i.e., self-report, clinician-ratings, performance-based measures). Converging evidence from these assessments make it less likely for significant relationships to be an artifact of the method used (Hooker et al., 2011). Mediation models were used to test whether neural activity for ToM accounts for the relationship between social anhedonia and social functioning. We predict the following: 1) individuals with schizophrenia will exhibit reduced recruitment of the ToM network, specifically MPFC, compared to matched controls; 2) neural activity in these regions will predict social behavior across all participants; 3) neural activity in these regions will mediate the relationship between social anhedonia and social functioning.

2. Methods

2.1. Participants

Twenty individuals with schizophrenia or schizoaffective disorder (SZ) and 18 healthy controls (HC) were recruited from the Greater Boston Area and participated for monetary compensation (Table 1). Inclusion criteria for all participants included being between the ages of 18 and 65, English speaking, no neurological or major medical illness, no history of head trauma, no substance abuse within six months, and no current or past history of substance dependence. Inclusion criteria for SZ included a diagnosis of schizophrenia or schizoaffective disorder, no comorbid axis I disorders, and no history of electroconvulsive therapy. Inclusion criteria for HC included no current or past axis I disorder and no first-degree relative with a psychotic disorder. Efforts

were made to recruit an HC group that matched the SZ group in demographics and education. Thus, advertisements for HCs specified that we were particularly interested in participants who finished high school, but did not necessarily attend or complete college. Participants gave informed written consent in accordance with the Institutional Review Board at Harvard University.

2.2. Social anhedonia

Social anhedonia was assessed with the Revised Scale for Social Anhedonia (SocAnh) (Eckblad et al., 1982), which is widely used in the schizophrenia/schizotypy literature to assess this phenomenon (Gooding et al., 2005, Horan et al., 2008 and Kwapil, 1998). SocAnh consists of 40 self-reported items answered true/false (e.g., "Having close friends is not as important to me as many people say" [keyed true]; "If given the choice, I would much rather be with others than alone" [keyed false]). Higher scores denote greater disinterest/less pleasure in and lack of social interaction.

2.3. Social variables

2.3.1. Interpersonal Reactivity Index

The Interpersonal Reactivity Index (IRI) (Davis, 1980 and Davis, 1983) is a 28-item self-report measure that assesses different facets of empathy. We were interested in two subscales: the perspective-taking subscale (IRI-PT), which assesses an individual's tendency to engage in ToM, specifically through adopting another's perspective (e.g., "I sometimes try to understand my friends better by imagining how things look from their perspective."), and the empathic concern subscale (IRI-EC), which assesses an individual's tendency to consider the emotional states and feel sympathy for others (e.g., "I often have tender, concerned feelings for people less fortunate than me."). Each subscale consists of 7 items that are rated on a scale from 0 (does not describe me well) to 4 (describes me very well).

2.3.2. MSCEIT—Managing Emotions

Participants completed the Managing Emotions subtest of the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT-ME) (Mayer et al., 2003), a performance-based assessment of social cognition that measures an individual's understanding of how emotions affect behavior and how to best manage emotions in a variety of social contexts. Participants read short vignettes about story characters and judge how socially effective different actions would be for the story character in managing their emotions. This task was completed as part of the MATRICS battery.

2.3.3. Social Adjustment Scale—Self-Report

The Social Adjustment Scale—Self-Report (SAS) (Weissman et al., 1978) is a 54-item questionnaire that assesses functioning over the past

two weeks in six areas: work, social and leisure activities, relationship with extended family, role as a marital partner, parental role, and role within the family unit. Participants answer questions on a 5-point scale that are designed to assess performance, friction with others, interpersonal relationships, and satisfaction within each area. Raw scores were averaged across areas and converted to a gender-adjusted T score which represents overall social functioning. Lower scores represent better social functioning.

2.3.4. Global Functioning Social Scale

Participants were administered the clinician-rated Global Functioning Social Scale (GFS) (Cornblatt et al., 2007), which assess the quantity and quality of peer relationships on a scale from 1 (extreme dysfunction) to 10 (superior functioning).

2.4. fMRI task: False-Belief Task

Participants underwent functional magnetic resonance imaging while performing an optimized version of the False-Belief Task (Dodell-Feder et al., 2011). This task, as in other ToM tasks used in the literature (Happe, 1994 and Stone et al., 1998), requires participants to predict behavior based on mental states. More specifically, participants read short stories designed to fit one of two conditions: (1) False-Belief (FB) stories describe a protagonist's outdated (i.e., "false") belief, and actions based on that outdated belief (e.g., "The morning of the high school dance, Barbara placed her high heel shoes under her dress and then went shopping. That afternoon, her sister borrowed the shoes and later put them under Barbara's bed."), and (2) False-Photograph (FP) stories describe outdated physical states in the world through photographs and maps (e.g., "Old maps of the islands near Titan are displayed in the Maritime museum. Erosion has since taken its toll, leaving only the three largest islands."). Both stories require the representation of false content. The critical difference is that the false content pertains to beliefs in FB stories and physical states in FP stories. Following the presentation of each story, participants responded to a true/false question (half of which referred to the false representation and half referred to reality) with a button press (e.g., FB: "Barbara gets ready assuming her shoes are under the dress"; FP: "Near Titan today, there are many islands").

In the scanner, participants saw 10 stories per condition, divided into two functional runs (5 stories presented from each condition in each run). Stories were visually presented for 11 s, followed by the true/false question for 6 s, and finally 12 s of fixation on a center cross. Each functional run lasted 5 min and 2 s. Stories were presented according to two predetermined orders (divided evenly between

participants in each group), in which story order was pseudorandomized within and across runs. Stimuli were presented in white font on a black background with Matlab 7.6 using Psychophysics Toolbox extensions (Brainard, 1997 and Kleiner et al., 2007). Accuracy and reaction time (RT) data were collected.

2.5. fMRI data acquisition and analysis

fMRI data were collected on a 3T Siemens scanner at Harvard University with echo-planar images (47 sagittal slices, $3 \times 3 \times 3$ mm voxels, TE = 30 ms, TR = 2560 ms, flip angle = 85°). A high-resolution T1-weighted anatomical image was acquired with an MPRAGE sequence (176 sagittal slices, $1 \times 1 \times 1$ mm voxels). Data were analyzed with

SPM8 (<http://www.fil.ion.ucl.ac.uk/spm/software/spm8/>) within the general linear model (GLM) framework. Preprocessing occurred in the following steps: realignment to the mean functional image, co-registration to the anatomical image, normalization to MNI template space, and smoothing with an 8 mm Gaussian kernel. Data were high pass filtered at 128 s. Within each subject, hemodynamic response to each condition was estimated at the start of each story for the duration of the story and true/false judgment. Scans that were ± 3 SD from the mean global signal intensity or exceeded 3 mm in movement from the previous volume (identified with the Artifact Detection Tool, http://www.nitrc.org/projects/artifact_detect/) were entered as nuisance regressors to reduce noise.

2.5.1. Neural activity for FB > FP within each group

To verify the expected task-related activity, we first identified neural activity for FB > FP separately within HC and SZ with one-sample t-tests. The statistical threshold was set to $p < .001$, $k > 10$, uncorrected for multiple comparisons. Regions that survive correction for multiple comparisons at a voxel-wise $p < .001$, corrected at the cluster-level to $p < .05$, are marked with an asterisk

2.7. Mediation analysis

Finally, we performed a mediation analysis to investigate the hypothesis that neural activity for ToM accounts for the link between social anhedonia and our measures of functioning (SAS, GFS). Several paths between the variables are estimated in typical mediation analyses including the total effect of an independent variable X on a dependent variable Y (path c), which consists of the direct effect of X on Y after controlling for mediator M (path c') and the indirect effect of X on Y through M (i.e., the product of path X \rightarrow M and M \rightarrow Y; path ab) (Fig. 3A). A test of mediation is conducted by evaluating whether path ab is significantly different from zero; that is, whether there exists a significant difference between

the total effect (path c) and direct effect (path c') that accounts for M (Preacher and Hayes, 2004 and Preacher and Hayes, 2008). We used a non-parametric bootstrapping procedure, which is better suited for smaller sample sizes, to derive bias-corrected 95% CIs of the ab sampling distribution based on 5000 bootstrap samples (Preacher and Hayes, 2008). If the CI does not encompass zero, then the indirect effect (path ab) is significantly different from zero ($p < .05$), indicating that neural activity accounts for a statistically significant portion of the relationship between social anhedonia and social functioning. Contrast values extracted from the independent ROIs were used as the measure of neural activity. Data from all participants were used in this analysis.

3. Results

3.1. Participant characteristics and False-Belief Task performance

SZ and HC did not differ in demographic characteristics or IQ (Table 1). SZ performed worse on all neurocognitive domains assessed in the MATRICS, except working memory, with effect sizes ranges from .36 (visual learning) to .85 (speed of processing). However, only the differences in speed of processing, and verbal learning (at a trend level) were statistically significant. Notably, the difference between SZ and HC on SocAnh was significant only at a trend level and smaller than what has been observed by other investigations (Horan et al., 2008). Behavioral data on the scanner task were not collected for 3 SZ participants and 2 HC participants due to technical error. SZ and HC did not differ in accuracy in either condition. SZ did not differ from HC in RT to the FP question, but were significantly slower to the FB question.

4. Discussion

Using a well-validated ToM scanner task and multiple-methods for assessing different aspects of social behavior, we found that individuals with schizophrenia exhibit reduced recruitment of MPFC for ToM. Neural activity in MPFC correlated with understanding how to manage emotions (MSCEIT-ME), the tendency to engage in perspective-taking (IRI-PT), and both self-reported (SAS) and clinician-rated (GFS) measures of social functioning. Finally, mediation analysis provided evidence that social anhedonia influences social functioning through its effect on ToM-related neural circuitry.

Similar to other investigations (Brune et al., 2011, Brunet et al., 2003, Das et al., 2012, Hooker et al., 2011, Lee et al., 2006, Lee et al., 2011 and Walter et al., 2009a), the ROI analysis revealed significantly

less MPFC activity for ToM in individuals with schizophrenia versus matched healthy controls. Though the whole-brain analysis did not yield any group differences at a corrected threshold, we observed largely converging evidence of reduced MPFC activity in the schizophrenia group at an uncorrected threshold. Importantly, we found several relationships between neural activity in MPFC and RTPJ for ToM and aspects of social behavior, which were largely unchanged when controlling for the effects of age and IQ. More specifically, neural activity in MPFC correlated with trait perspective-taking on the IRI-PT, MSCEIT-ME performance, self-reported social functioning on the SAS, and clinician-rated social functioning on the GFS, such that greater activity in these regions was associated with better social cognition and social functioning, respectively. RTPJ activity correlated with MSCEIT-ME, SAS, and IRI-PT at a trend level. We note that several of these relationships may have been influenced, in part, by group differences on the social variables (e.g., MSCEIT-ME, SAS, and GFS), which could have led to clustering of data points by group and an inflated correlation coefficient. Furthermore, most of these relationships were observed across all participants, and not separately within each group. With that said, we did find several significant associations within SZ participants alone: greater neural activity in MPFC was associated with less social impairment on the SAS, and greater neural activity in RTPJ was associated with better ability to manage emotions through the use of affective ToM skills on the MSCEIT-ME. The finding that some brain-behavior relationships were found in one group and not the other would suggest that there may be differences between the groups in how ToM-related neural activity may influence social behavior. However, reduced power may have prevented us from observing additional relationships between neural activity and social functioning within the SZ and HC group separately. Furthermore, recruitment of the ToM network should in theory be related to social functioning and ability regardless of diagnosis making it important to investigate these associations across groups where the relationship can be examined across the full range of neural and social functioning. Nonetheless, the relationships found here should be interpreted with caution, and examined in future work with larger sample sizes.

Together, these findings indicate that increased neural activity for ToM in MPFC and RTPJ is associated with greater perspective-taking in daily life, enhanced social cognitive ability, specifically the ability to use ToM skills to effectively manage emotions in different social situations, and social functioning. These findings are consistent with other

studies that have found increased MPFC activity during a ToM task to predict improvements in social functioning following recovery from a psychotic episode (Lee et al., 2006), and, in a separate study, following cognitive remediation (Subramaniam et al., 2012). Studies have also shown that VMPFC gray matter volume correlates with engagement in ToM to enhance interpersonal relationships among individuals with schizophrenia (Hooker et al., 2011), and MPFC/TPJ activity during ToM tasks correlates with increased perspective-taking in daily life (Falk et al., 2012, Hooker et al., 2008, Masten et al., 2013 and Moriguchi et al., 2006). These findings further highlight the functional significance of neural activity for ToM by relating it to several different aspects and measures of social behavior.

The relationship between MPFC and RTPJ activity and MSCEIT-ME performance is of particular significance. The MSCEIT-ME requires participants to use affective ToM (i.e., reason about the emotions of others) in order to effectively manage a story character's emotion and navigate various social situations. The correlations observed in the current study suggest that ToM-related neural circuitry may be important for social functioning by supporting affective ToM ability. These findings are of particular importance when considering that cognitive remediation programs, which include social cognition training, demonstrate intervention-related improvement on MSCEIT-ME performance (Eack et al., 2007, Eack et al., 2011, Hooker et al., 2012 and Sacks et al., 2013) and increased MPFC activity which tracks with improvements in social functioning (Subramaniam et al., 2012). Taken with our data, it suggests that the neural mechanisms supporting ToM are amenable to intervention and are likely to result in measurable changes in affective ToM skills and the use of those skills to improve social interaction. Furthermore, in line with the MATRICS initiative (Green et al., 2004 and Green et al., 2005), these data, taken with other neuroimaging findings (Wojtalik et al., 2013), demonstrate that performance on, at least, the social cognition subtest of the MATRICS tracks with important individual differences in neurobiology that are linked to social functioning, supporting the measure's validity and usefulness as a tool to assess intervention-related change.

Addressing our second aim regarding the role of ToM-related neural circuitry in the social anhedonia—social functioning relationship, mediation analysis replicated findings demonstrating a link between social anhedonia and social functioning (Blanchard et al., 1998, Blanchard et al., 2011 and Cohen et al., 2005), and provided novel evidence that 1) neural activity for ToM in MPFC is predicted by social anhedonia, such that individuals

reporting higher social anhedonia had less MPFC activity for ToM, and 2) neural activity in this region accounts for the relationship between social anhedonia and social functioning. Specifically, we found neural activity in MPFC to account for a significant portion of the variance between social anhedonia and our measures of social functioning, including the SAS (self-report) and GFS (clinician-rated). This finding suggests that social anhedonia impacts social functioning, at least partially, through its effect on ToM-related neural circuitry.

The extant literature on social anhedonia and social functioning has largely focused on the role of decreased reward and impaired memory for reward (Dowd and Barch, 2010, Gard et al., 2007, Horan et al., 2008, Kring and Elis, 2012 and Strauss and Gold, 2012). Here, we demonstrate a previously uninvestigated link between social anhedonia and social functioning through impaired ToM. Although the data here are cross-sectional and cannot speak to causality, nor do they fully account for social functioning impairments in schizophrenia, the results are consistent with several interpretations. Disruption to the neural mechanisms subserving ToM may make social interaction challenging, increase social stress, decrease social reward or increase disinterest in socializing, thereby contributing to anhedonia and social dysfunction. On the other hand, social anhedonia and isolation, both of which have been identified as risk factors for schizophrenia (Kwapil, 1998, Tarbox and Pogue-Geile, 2008 and van Os et al., 2000), may lead to deleterious changes in the neural mechanisms subserving ToM, thereby contributing to misperceptions of intentions and emotions, interpersonal conflict, and social impairment (Hoffman, 2007). Though both explanations are theoretically viable, additional work, such as prospectively measured reports of social anhedonia, ToM-related neural function, and social functioning, would be needed in order to better evaluate these possibilities. Nonetheless, the findings do demonstrate significant interrelationships between these different constructs. Furthermore, it suggests that training ToM, as opposed to pharmacologically augmenting response in reward-related neural circuitry (Juckel et al., 2006a, Juckel et al., 2006b, Schlagenhauf et al., 2008, Walter et al., 2009b and Waltz et al., 2007), may be a more accessible process for remediation that will most likely lead to improvements in social functioning.

It is important to note that functional outcome in schizophrenia is multiply determined. Neural function, while shown here to capture a significant portion of the variance in social functioning, still leaves much variance unexplained. Research has

demonstrated that cognitive and affective factors such as dysfunctional attitudes (Granholm et al., 2009, Grant and Beck, 2009, Green et al., 2012 and Horan et al., 2010), motivation (Gard et al., 2009), and metacognition (Lysaker et al., 2010a, Lysaker et al., 2010b and Lysaker et al., 2011), as well as external factors relating to social support (Brekke et al., 2005), disability policies and the availability of employment (Rinaldi et al., 2010 and Tandberg et al., 2013), also contribute to functioning. A comprehensive model of functional outcome would ideally incorporate all of these factors. Similarly, functioning may best be improved through a multipronged approach targeting neurocognitive functioning as well as these other factors.

Finally, the sample of schizophrenia participants tested here demonstrated less neurocognitive impairment (i.e., IQ, MATRICS) than what is typically observed in this population (e.g., Kern et al., 2011). Though this may be in part the result of recruiting an education-matched HC group, it warrants caution in generalizing our findings to lower-functioning individuals with schizophrenia. With that said, the neural data demonstrating reduced recruitment of MPFC for ToM in the schizophrenia group replicates several other studies with more neurocognitively impaired schizophrenia samples. This suggests that reduced MPFC activity for ToM is characteristic of individuals with schizophrenia high- or low-functioning.

In conclusion, the current study finds that individuals with schizophrenia exhibit reduced recruitment of MPFC for ToM, which is related to social functioning, and may be a contributing mechanism through which social anhedonia affects social functioning. These findings reveal proximal neurobiological determinants of social functioning, indicative of aberrant function of the social brain, and suggest neural targets for remediation.

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