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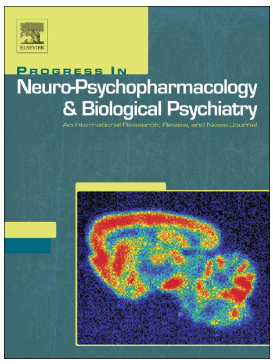

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Social Cognition and Self-other Distinctions in Neuropsychiatry: Insights from Schizophrenia and Tourette Syndrome

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Abstract

Impairments in social cognition may reflect dysfunction of disorder specific or disorder general mechanisms. Although cross-disorder comparison may prove insightful, few studies have compared social cognition in different neuropsychiatric disorders. Parallel investigation of schizophrenia and Tourette syndrome (TS) is encouraged by similarities including the presence of problematic social behaviour, echophenomena, emotional dysregulation and dopamine dysfunction. Focusing on tests of social cognition administered in both disorders, this review aims to summarize behavioural, neurophysiological and neuroimaging findings, before exploring how these may contribute to clinical symptoms. Studies investigating social cognition (imitation, emotion recognition, and understanding of beliefs or intentions) in patients with schizophrenia or TS were identified from Web of Science and PubMed searches. Although findings indicate that socio-cognitive deficits are more apparent in schizophrenia, adults with TS can exhibit similar task performance to patients with paranoia. In both disorders, behavioral and neuroimaging findings raise the possibility of increased internal simulation of others' actions and emotions, in combination with a relative under-application of mentalizing. More specifically, dysfunction in neurobiological substrates such as temporo-parietal junction and inferior frontal gyrus may underlie problems with self-other distinctions in both schizophrenia and TS. These difficulties in distinguishing between actions and mental states linked to the self and other may contribute to a range of psychiatric symptoms, including emotional dysregulation, paranoia, social anhedonia and socially disruptive urges. Comparing different patient populations could therefore reveal common neuro-cognitive risk factors for the development of problematic social behaviors, in addition to markers of resilience, coping strategies and potential neuro-compensation mechanisms.

Keywords: social cognition; mirror neuron system; mentalizing; schizophrenia; Tourette syndrome.

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1. Introduction: Social cognition, schizophrenia and Tourette syndrome

1.1. The importance of social cognition in neuropsychiatry

Social cognition refers to the collection of skills needed for successful interaction with others, and that can allow one to take advantage of their social group (Frith, 2008). These skills include the capacity to empathise and the ability to understand mental states such as beliefs and intentions (i.e. mentalizing, or theory of mind). Social interactions between patients and their families, colleagues, and health care professionals are frequently indicators of mental health needs, and social exchanges between patient and clinician influence assessment and diagnosis. Indeed, problems with social cognition can be crucial determinants of the presence of neuropsychiatric disorders (e.g. autistic spectrum disorder, anti-social personality disorder). The importance of social cognition is such that studies have now been conducted across many neurological and psychiatric disorders, including movement disorders, the dementias, epilepsy, mood and anxiety disorders and more. This research offers critical insight into the phenomenological experience of living with these conditions, and the neuropsychological underpinnings of behavioral symptoms and functional impairment.

Within the domain of social cognition, an important distinction has been highlighted between mirroring and mentalizing processes (Grafton, 2009; Van Overwalle and Baetens, 2009). The Mirror Neuron System (MNS: Rizzolatti et al., 2009; Iacoboni, 1995; Iacoboni et al., 1999) underpins motor simulation of observed actions, providing a basis for imitation and recognition of action goals, facial emotions and non-verbal communication based on visual motor cues. However, mental states such as beliefs are less directly observable, and may therefore be determined through mentalizing based on semantic reasoning and autobiographical memory. Spunt and Lieberman (2012a) propose that a social cognitive task first activates the MNS (including inferior parietal cortex and inferior frontal gyrus: IFG), followed by mentalizing areas (ventromedial prefrontal cortex, precuneus, temporo-parietal junction: TPJ, posterior superior temporal sulcus: STS, and anterior temporal lobe). While the MNS contains some areas that respond more to action perception, the mentalizing network appears to support abstract, modality independent representations of mental states (Spunt and Lieberman, 2012b). While the MNS is automatically engaged by pertinent social stimuli, mentalizing processes may involve more conscious deliberation about mental states (Van Overwalle et al., 2009). This is supported by evidence that the MNS operates more efficiently than the mentalizing system, as its activation is less affected by concurrent cognitive demands (Spunt and Lieberman, 2013). Neurodevelopmental findings could support the idea that the MNS develops before mentalizing ability, as children can imitate from early infancy (Meltzoff, 1988) and the understanding of mental states seems to progress from emotions and intentions (linked to action

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goals), to more abstract desires, and finally beliefs (Wellman et al., 2004). More recently, studies have emphasized the importance of self-other distinctions in social cognition i.e. the ability to distinguish between the mental states of oneself and other people (e.g. see de Guzman et al., 2016) and have explored the neurodevelopmental underpinnings of this ability (Steinbeis, 2016). A failure in self-other differentiation when mirroring or mentalizing could lead to confusion, distress, and inaccurate mental state attribution.

Other important concepts now becoming more widely applied within the social cognitive domain include those of hyper- and hypo-mentalizing. Hyper-mentalising, or 'hyper-theory of mind' (Abu-Akel, 1999) was perhaps first described in relation to schizophrenia, and is thought to involve unconstrained hypothesis generation relating to the attribution of knowledge to the self and/or others (Abu-Akel & Bailey, 2000). Crespi and Badcock (2008) further suggest that psychosis and autism may fall at opposite ends of the cognitive spectrum, given that social cognition can appear underdeveloped in autism (i.e. this disorder may most frequently feature hypo-mentalizing), but hyper-developed to a dysfunctional degree in those with psychosis. These concepts may prove useful in drawing cross-disorder comparisons. However, it is important to recognise the distinction between abnormalities of social cognition that reflect application deficits versus conceptual deficits (Abu-Akel & Bailey, 2000).

Many psychiatric conditions could be construed as disorders of social interaction, although it is unclear whether patients' social cognitive deficits reflect dysfunction of disorder specific or disorder general mechanisms (Schilbach, 2016). Cross-disorder comparison may prove insightful in this regard. One previous study (Gallagher and Varga, 2015) did consider the application of major theories of social cognition (theory theory; simulation theory; interaction theory) in relation to explaining the social cognitive difficulties of individuals with schizophrenia and autistic spectrum disorder. The authors concluded that some disorders may involve more generalised impairments in social cognition, whereas others may feature more circumscribed deficits. Another recent study (Bora et al., 2016) compared cognitive abilities including theory of mind in schizophrenia and bipolar disorder, suggesting that social cognition was more impaired in schizophrenia, and that such an approach may enhance diagnostic classification and treatment. Overall however, very few existing studies have compared social cognition in different neuropsychiatric disorders. This review will explore how abnormalities in social cognition and underlying brain function could help to explain the behavioral symptoms of both schizophrenia, and Tourette Syndrome (TS). Schizophrenia is widely accepted to involve disturbances in social cognition (e.g. Green et al., 2015), but only more recently have these skills been implicated in TS (Eddy and Cavanna, 2013b). Investigation of these two disorders in parallel is encouraged by the presence of

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problematic social behaviors in both conditions, in addition to other notable similarities, such as dopaminergic dysfunction and temporal lobe abnormalities. Exploring social cognition in both schizophrenia and TS could offer insight into how similar underlying problems with social cognition may manifest as alternative behavioral problems based on individual neurological differences, conditioned behavioral responses or various coping and compensation mechanisms.

1.2. Schizophrenia

The prevalence of schizophrenia has been estimated at between 0.5 (Saha et al., 2005) and 1% (e.g. Keith et al., 1991; DSM 2000 4th Ed as cited by Shultz et al., 2007) across all cultures. Genetic liability is high (about .81), with a concordance rate of 50% between identical twins (MacDonald and Schulz, 2009). Although childhood onset can occur (see Bartlett, 2014), most cases of schizophrenia are diagnosed between age 20-25 (Walker et al., 2004). There is frequently an earlier prodromal phase, which involves gradual development of symptoms, social withdrawal, loss of interest in activities and self-care, and emotional problems (Shultz et al., 2007), including emotional dysregulation (see Aleman & Kahn, 2005; Livingstone et al., 2009) and difficulties in interpreting and describing one's own emotions (i.e. alexithymia; see Fogley et al. 2014; O'Driscoll et al., 2014). Schizophrenia is thought to be a neurodevelopmental disorder (Weinberger, 1987; Rapoport et al., 2012), involving early insult and a latent period through much of neurodevelopment, until the emergence of psychosis in late adolescence or early adulthood, perhaps at a point when compensatory mechanisms become inadequate (Insel, 2010). Those who go on to develop the condition often demonstrate motor, cognitive and social impairments during childhood (Goghari et al., 2010; Larson et al., 2010), including a high frequency of negative facial expressions and reduced responsivity in social situations (e.g. Dworkin et al., 1993; Walker et al., 1994; Walker et al.,1999).

Early descriptions of schizophrenia (e.g. Bleuler, 1911) noted disturbances of association, affect and reality, including delusions and hallucinations, movement disturbance (e.g. catatonia), somatic symptoms, manic and melancholic states. Schneider's (1959) first-rank symptoms distinguish between behavioral signs with an excess of ideas or sensory experience (positive symptoms) versus those which involve a decrease in behavior (negative symptoms). Negative symptoms include lack of motivation, anhedonia, flattened affect and social withdrawal; whilst common positive signs include delusions and hallucinations, such as command hallucinations, whereby patients perceive voices telling them to commit harmful or dangerous acts (Schultz et al., 2007). Based on previous factor analyses, three symptom domains (positive, negative and disorganized) are now widely accepted (Grube et al., 1998), with the disorganization dimension including symptoms such as

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formal thought disorder, loose associations, bizarre behavior and inappropriate affect. The positive, negative and disorganized symptoms domains may be associated with distinct underlying neural correlates (Goghari et al., 2010).

A continuum model of schizophrenia is now widely accepted, such that schizophrenia falls within a larger spectrum of disorders also including Schizotypal Personality Disorder, schizoaffective disorder and numerous other psychotic conditions, and experimental work now frequently includes 'at risk' populations. Symptoms are highly heterogenous, with severity of positive, negative and disorganized symptoms varying across time within and across individuals (MacDonald and Schulz, 2009). In addition to a distorted perception of reality, key characteristics of schizophrenia include excessive threat anticipation (Freeman, 2007), loss of volition (Goghari et al., 2010) and disturbed social function (Larson et al., 2010). Deterioration of social relations may be considered a "hallmark" of schizophrenia (Hooley, 2010), and social symptoms include echophenomena (i.e. mirroring of other people's speech or actions; Peralta et al., 2010), and social anhedonia (e.g. Blanchard et al., 1998), which refers to a lack of pleasure from social interaction. A diagnosis of schizophrenia according to DSM-V (American Psychiatric Association, 2013) criteria requires the presence of delusions, hallucinations or disorganized speech; plus at least one other symptom in the form of delusions, hallucinations, disorganized speech, disorganized/catatonic behavior, and negative symptoms; present for over 6 months, and leading to impairment in one or more areas of functioning (social, occupational etc.). Presentation is typically chronic with an episodic exacerbation in symptoms, and while dopamine blocking agents are therapeutically beneficial, they have limited impact on negative symptoms, and a proportion of cases remain refractory (Van Sant and Buckley, 2011).

1.3. Tourette syndrome

Approximately 1% of school age children are thought to have TS, with a median age of onset between 4 and 6 years of age (Leckman et al., 2006). The cardinal motor signs of TS are tics: repetitive, rhythmic and stereotyped movements and vocalisations (Faridi and Suchowersky, 2003). They can be simple (e.g. blinking, grimacing, kicking) or complex (bending over, shouting certain words), and can occur in sequences. Tics characteristically wax and wane over time in severity and frequency (Coffey et al., 1994). Most people with tics experience a sensory-psychological urge before the tic is performed which can make the tic feel semi-voluntary, or 'unvoluntary' (Jankovic, 1997). There is usually a short period of relief after the tic, although most people with tics can suppress them for at least a short period of time. A diagnosis of TS indicates the presence of both motor and vocal tics across 12 months (though not necessarily concurrently), with onset before age 18 (DSM-V:

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APA, 2013). Tic severity tends to peak around puberty and reduce during early adulthood. It is currently unclear why tics affect more males than females, with a ratio of approximately 3-4:1 (Robertson, 2015).

Some common symptoms of TS are linked to sensory stimuli. These include compulsions to make things look or feel 'just right' (e.g. Neal and Cavanna, 2013) e.g. to visually align objects. Such symptoms are suggestive of an overlap with obsessive-compulsive disorder (OCD; for a review see Eddy and Cavanna, 2014). Other complex social symptoms initially described by Gilles de la Tourette (1885) include echophenomena and coprophenomena (obscene language or gestures). Between 25-50% of patients with TS may also exhibit urges to perform socially inappropriate behaviors, such as to insult people (Kurlan et al., 1996; Eddy and Cavanna, 2013a). Emotions can influence tics, and emotion dysregulation, including difficulties with anger control, are quite common (e.g. Budman et al., 2003; Cavanna et al., 2015). Intrusive thoughts compelling acts of harm to the self or others can also feature in TS (Eddy and Cavanna, 2013; George et al., 1993; Robertson et al., 1989).

TS is suggested to be heriditary (Comings, 1990; Pauls and Leckman, 1986; Deng et al., 2012), but a clear genetic marker is yet to be confirmed. The basal ganglia are implicated in the motor aspects of TS (e.g. Mink, 2001;2006). However, the neural mechanisms underlying complex context-related tics are poorly understood. Tics respond well to dopamine blocking medication (Eddy et al., 2011b), and elevated levels of dopamine in TS may help to explain many symptoms, including impulse control problems (Wright et al., 2012), jumping to conclusions (Eddy and Cavanna, 2014), and hyper-mentalizing (Eddy and Cavanna, 2015).

While diagnosis of frank psychosis is rare in TS, some studies suggest that such symptoms are more common in these patients than the general population. Early studies suggested that the manifestations of TS can include hallucinations, paranoid ideation, schizotypal thinking, and psychosis (Comings, 1990; Comings and Comings, 1987, Comings and Comings, 1991; Kerbeshian and Burd, 1985, 1988; Sverd et al., 1993; Takeuchi et al., 1986). For example, Comings et al. (1987) reported that auditory hallucinations of voices were reported by 14.6% of TS patients versus 2.1% of healthy controls (HC). A later study confirmed a higher prevalence of schizotypal traits in adults with TS than in the general population (Cavanna et al., 2007). In addition, a few studies have reported case series who exhibited tics in childhood and later developed schizophrenia, albeit generally with persisting tics (Müller et al., 2002; Takeuchi et al., 1986). Echophenomena (e.g. Peralta et al., 2010), obsessive compulsive behaviors and catatonic like motor behaviors (Müller et al., 2002) are associated with both TS and schizophrenia.

1.4. Review aims

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Multiple intriguing parallels in schizophrenia and TS include emotional dysregulation, socially dysfunctional behaviors, obsessive-compulsive features, and the efficacy of anti-dopaminergic drugs. Furthermore, overlapping characteristics include symptoms rarely reported in other psychiatric disorders, such as echophenomena and sensory distortions. Perhaps most importantly, both of these clinical populations can feature socially inappropriate or harmful urges. While such symptoms appear less severe in TS, patients' self-injurious and socially inappropriate urges may appear reminiscent of the command hallucinations seen in schizophrenia. It is possible that there is some shared etiological or developmental relationship between TS and schizophrenia. Whatever the case, studying the association between these symptoms and social cognition across disorders may offer invaluable insight into the neuropsychological factors that contribute to dangerous and socially disruptive behaviors.

This review first provides a background on the social cognition literature in schizophrenia and TS, summarising behavioral findings, followed by evidence of related abnormalities in brain function. Rather than an exhaustive systematic review of all studies, there is a focus on more commonly administered tasks within core areas of social cognition (e.g. imitation, emotion recognition, understanding of beliefs) that have been investigated in both schizophrenia and TS, allowing cross-disorder comparison. The section after this explores how self-other distinctions may be compromised in these disorders, and how excessive mirroring and hypo-mentalizing may contribute to symptoms such as emotional dysregulation, paranoia, social anhedonia (SA) and socially disruptive urges. Clinical implications, limitations of current knowledge and directions for future research are summarized in the final section.

2. Findings on tests of social cognition

2.1. Behavioral findings

This section outlines behavioral performance of patients with schizophrenia and TS on a range of tasks within the domain of social cognition. Papers were obtained through searches of Web of Science and PubMed between 06/2016 and 12/2016. Search terms were *emotion*; *social cognition*; *theory of mind*; *mentalizing*; or *mirror neuron*; plus at least one of *Tourette*; *schizophrenia*; *psychosis*. Abstracts were manually searched for relevance and studies are reported within themes where at least one study was available for each disorder. Firstly, studies likely to involve the MNS are considered, followed by related tasks involving recognition of overt emotional expressions, and empathy measures. Finally, mentalizing tasks involving inference about covert mental states such as beliefs and intentions are discussed. These themes broadly align to the domains

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'experience sharing', 'social cue perception', and 'mentalizing' (respectively), within in the model of empathy proposed by Green et al. (2015). Key findings are summarized in Table 1.

2.1.1. Imitation and motor resonance. The MNS allows an observer to (overtly or covertly) simulate perceived actions in order to achieve both motor and emotional resonance with the observed actor (e.g. Brunet-Gouet et al., 2006), and may contribute to recognition of actions and emotions in addition to imitation. A few studies have investigated imitation in schizophrenia, and have noted deficits when patients are asked to copy e.g. observed mouth movements or hand gestures, although it is unclear whether deficits reflect attention or motor problems (Park et al., 2008; Falkenberg et al., 2008). For example, reduced imitation of others' head or body movement (Kupper et al., 2015) or laughing (Haker and Rossler, 2009) is often associated with negative symptoms. Nakagawa and Hoshiyama (2015) monitored the grip strength of patients with schizophrenia and HC when watching actors grip at different strengths. Patients' grip strength reduced when watching someone else grip at a lower strength (patients with more positive symptoms showed the greatest effect), whereas HC showed no fluctuation in grip force. This may imply greater interference from observation of others' actions in schizophrenia. Relatively few studies have explored imitation in TS. One study reported that in addition to echophenomena, these patients exhibit a tendency to mirror the movements of others when observed under experimental conditions (Finis et al., 2012). The findings of another study in TS show some similarity to those reported for patients with schizophrenia (especially positive symptoms) above. Jonas et al. (2010) compared patients with TS and HC when making finger movements while observing compatible (same) and incompatible (different) finger movements made by an actor. Patients performed similarly to HC for compatible movement observation, but showed greater interference than HC when viewing incompatible finger movements.

2.1.2. Empathy and emotion recognition. The MNS may assist in the simulation of (and therefore perception of) embodied emotion (Niedenthal, 2007). Numerous studies have examined the ability of people with schizophrenia to recognise basic emotions (happiness, sadness, anger, fear, surprise and disgust) in facial expressions. Deficits may be most apparent for sadness, fear (Edwards et al., 2001; Kohler et al., 2003), or disgust (e.g. Kohler et al., 2003). Interestingly, patients sometimes over-attribute negative emotions in response to neutral expressions (e.g. Kohler et al., 2003; Tsoi et al., 2008; Behere et al., 2011). Other studies suggest that paranoid patients exhibit better emotion recognition than patients with other forms of schizophrenia (Phillips et al., 1999). In relation to TS, a small study of five adult patients (Devinsky et al., 1993) reported intact or enhanced ability when exploring recognition of emotion from voice, face or body. Two other studies (Baron-Cohen et al., 1997; Sprengelmeyer et al., 1997) found no facial emotion recognition deficit in adults with TS.

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However, the latter study found that those patients showing evidence of OCD (with or without TS) exhibited poor recognition of disgust and anger. A more recent study (Drury et al., 2012) also found little evidence for any impairment in basic emotion recognition from facial expressions in children and adults with TS.

The Reading the Mind in the Eyes Test (RMET: Baron-Cohen et al., 2001) is a task of facial expression recognition requiring the attribution of complex neutral (e.g. interested), positive (e.g. playful) and negative (e.g. upset) mental states. Studies have used this task in schizophrenia and reported deficits (Lam et al., 2014; Lugnegård et al., 2013) or no impairment in comparison to HC (e.g. Wischniewski and Brüne 2011; Scherzer et al., 2012). Performance has been linked to symptom severity (e.g. Rominger et al., 2016) and constricted affect (Irani et al., 2006). Deficits can include errors for neutral expressions (Prevost et al., 2015), as can be seen in young people at high risk for psychosis (Zhang et al., 2016). In TS, Eddy et al. (2011a) reported a mild deficit on the RMET when comparing adult patients to HC, but other studies reported no significant behavioral deficit (Baron-Cohen et al., 1997; Eddy et al., 2017).

The Interpersonal Reactivity Index (Davis, 1980) assesses self-reported empathy and contains four subscales, assessing fantasy (imagination and self-projection), empathic concern (appreciating others' emotional state), personal distress (PD: experiencing negative emotion when observing other people's distress) and perspective taking (PT: tendencies to put oneself in someone else's 'shoes'). Therefore PD may involve mirroring processes than enable emotion contagion, whereas PT may require mentalizing. Patients with schizophrenia exhibit significantly higher scores than HC on the PD subscale (e.g. Abramowitz et al., 2014; Wojakiewicz et al., 2013; Martins et al., 2011; Sparks et al., 2010) and sometimes lower PT scores (Montag et al., 2007). Those with first-episode psychosis (Kosaka et al., 2002) exhibit lower PT versus HC, and a trend for higher PD, and high social anxiety. Lehmann et al. (2014) found that high PD was linked to patients reporting more contagion of negative emotions. In HC, high empathy was associated with good emotion control. However, high empathy in patients was not related to emotion regulation capacity. Using the Questionnaire of Cognitive and Affective Empathy (Reniers et al., 2011), which contains some items adapted from the IRI, Holt et al. (2015) found decreased cognitive empathy (e.g. PT) alongside increased affective empathy (e.g. emotion contagion) in schizophrenia. In relation to the TS literature, one study using the IRI found that adults with TS reported increased PD and decreased PT when compared to HC (Eddy et al., 2015). This is a very similar pattern to that seen in schizophrenia.

A number of studies have reported impaired recognition of emotion as expressed through vocal prosody in schizophrenia (e.g. Hoekert et al., 2007), with poor recognition of negative emotions and sarcasm

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(Sparks et al., 2010; Cassetta and Goghari, 2014). However, deficits in auditory sarcasm recognition can correlate with pitch perception deficits implying impaired auditory processing (Kantrowitz et al., 2014). In relation to TS, Drury et al. (2012) investigated recognition of happy, sad, angry or frightened vocal tone and found that adults with TS alone were only impaired at recognising angry tone when the semantic content of the vocalisation was conflicting (i.e. contained pleasant content). Further research in this area is warranted.

2.1.3. Understanding beliefs and intentions. The faux pas task (Stone et al., 1998; Gregory et al., 2002), contains stories featuring remarks which may cause unintended offence to a character, and control stories with no faux pas. Participants are required to recognise whether faux pas is present, the potential negative impact, and its unintentional nature. Studies have reported deficits in recognising and understanding faux pas in paranoid schizophrenia (Lam et al., 2014; Scherzer et al., 2015), and sometimes despite intact performance on control questions (Ho et al., 2015). Shur et al. (2008) found a specific deficit whereby faux pas remarks were interpreted as intentional. In TS, Eddy et al (2010a; 2011a) and Channon et al. (2012) reported errors on the faux pas task including indications of faux pas where this was not present and under-recognition of socially inappropriate remarks. Sometimes patients concluded faux pas remarks were intentional, as reported by Shur et al. (2008) in schizophrenia.

Understanding humour frequently requires mentalizing. Corcoran et al. (1997) compared the ability of patients with schizophrenia and HC to understand humorous cartoons involving mentalizing or physical humour ('slap-stick'). Overall, patients with schizophrenia were more impaired at understanding mentalizing jokes, with paranoid patients and patients with passivity exhibiting the mildest deficit. In a later study involving cartoon jokes (Langdon et al., 2010), patients with schizophrenia again showed a deficit in understanding jokes involving theory of mind, but not slap-stick style humour. However, a few studies have reported a more generalised deficit (e.g. Marjoram et al., 2005). In one study that explored the understanding of humorous cartoons in TS (Eddy et al., 2011a), patients and HC exhibited no difference for slap-stick jokes, but adults with TS were more likely to misunderstand ironic and sarcastic jokes and to adopt a literal interpretation. This could reflect problems understanding the communicative intentions of the cartoonist.

A number of studies have assessed the understanding of beliefs in schizophrenia, on tasks including false-belief tasks (which require participants to understand that they know something that the story character does not), and the Strange Stories task (Happé, 1994) which requires understanding of beliefs and deception. Pickup and Frith (2001) explored responses to first-order (he thinks) and second-order (he thinks that she thinks) tests of mentalizing. Most patients with schizophrenia (particularly those with negative behavioral signs such as

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e.g. blunt affect, social withdrawal, poverty of speech) exhibited a deficit on only the second-order task. However, deficits may be seen on both first and second-order belief tasks (Mazza et al., 2008), on pragmatic language tasks (Langdon et al., 2002; Mazza et al., 2008) including the Hinting Task (devised by Corcoran et al., 1995; see Scherzer et al., 2015), and the Strange Stories task (Langdon and Ward, 2010); although impairments may be seen on questions assessing the understanding of physical states in addition to mental states (Scherzer et al., 2015). One study investigating the understanding of written non-literal language in adults with TS reported unconventional interpretations of sarcastic and metaphorical remarks, including literal interpretations (Eddy et al., 2010b). However, the same study reported no deficit on the Hinting Task. An early study in TS (Channon et al., 2004) reported no impairment on the Strange Stories. Subsequent studies assessing the understanding of false-belief (Eddy et al., 2010a; Eddy et al., 2016) also reported no significant deficit in adults with TS (Eddy et al., 2016).

The Frith-Happé Animations Task (AT: Abell et al., 2000; Castelli et al., 2000) requires viewers to watch short video-clips and describe the movements of two triangles, which can be either random (e.g. drifting), or depict simple (e.g. dancing) or complex (e.g. coaxing) interactions. Schizophrenia can be associated with a reduced tendency to attribute intentions when social interactions are depicted (Horan et al., 2009; Lugnegård et al., 2013; Ventura et al., 2015). Russell et al. (2006) reported that patients with paranoia and behavioral signs exhibited worse performance than HC on the AT, and groups with passivity delusion or remitted symptoms. This included a trend for longer explanations on random animations, perhaps linked to over-attributing mental states. Schizotypal individuals can also show an increased tendency to attribute mental states during the random movement condition (Fyfe et al., 2008). More recently, Eddy and Cavanna (2015) revealed that adults with TS were more likely than HC to describe social interactions and use mental state references when viewing random movement during the AT. These findings appear similar to those reported by Russel et al., in patients with paranoia, and could be indicative of hyper-mentalizing.

Tasks involving gaze cues include the Yoni cartoon task (Shamay-Tsoory and Aharon-Peretz, 2007), which requires the use of gaze direction to determine what the central character is thinking about or likes. Ho et al. (2015) found intact performance on this task in first episode schizophrenia on first-order trials, but significant deficits on second-order mentalizing questions. Another study (Zhang et al., 2016) found that the performance of patients with schizophrenia on this task was related to clinical insight (i.e. awareness of needing treatment). Eddy et al. (2011a) assessed the performance of adults with TS on a similar task (Shamay-Tsoory et al., 2007) that involved understanding of socially competitive emotions. The task was to use simple facial cues (eye gaze

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direction and mouth expression) to select a target that the central character was either envious of (target happy, character sad), or gloating about (target sad, character happy). Control participants scored at ceiling on the task, and adults with TS made significantly more errors, perhaps suggesting difficulties in dealing with two perceived conflicting emotions because of mirroring related interference.

Finally, given that fewer studies are available in TS than schizophrenia, it is worth mentioning a couple of other studies that did include social measures although they did not specifically aim to study social cognition. These studies (McGuire et al., 2013; Güler et al., 2015) found that the parents of young people with tic disorders reported increased evidence of social problems on a scale that can indicate the presence of behaviours associated with autistic spectrum disorder (e.g. aspects of social interaction, signs of obsessionality and inflexibility). However, both studies seemed to suggest that it was those individuals with tics plus obsessive compulsive or attention deficit features who showed the greatest differences to HC on the scale.

2.1.4. Summary of behavioral findings. Social cognitive deficits are usually more obvious in schizophrenia than in TS. For example, some patients with psychosis can demonstrate difficulty with recognising basic facial expressions whereas people with TS do not. However, social cognition has been much less explored in TS. Importantly, the specific symptom profile of schizophrenia is linked to task performance, such that patients with more negative symptoms demonstrate more robust evidence of deficits in social cognition. Patients with more positive symptoms show more subtle differences to HC such as seeing negative emotions under neutral conditions. Based on the data currently available, adults with TS (including those without comorbid obsessive compulsive or attention deficit disorders) exhibit only selective abnormalities on social cognitive tasks, and therefore appear more similar to patients with paranoia (or those with more positive symptoms of schizophrenia), rather than other presentations. Difficulties comprise understanding intentionality in the context of faux pas and sarcasm, and traits that may reflect greater mirroring than HC. In most cases it seems that people with TS and schizophrenia can experience motor interference when observing others' actions, and it is possible that studies reporting reduced imitation or mentalizing deficits have not controlled for factors such as general cognitive impairment, inattention or social anxiety. The following section explores neural abnormalities during social cognitive tasks in schizophrenia and TS.

2.2. Neurophysiological and neuroimaging findings during tests of social cognition

Quite a few studies have explored the neural correlates of tasks involving biological motion and imitation in schizophrenia, but the neural correlates of imitation have yet to be investigated in TS. Previous research has employed brain imaging methods during tests of facial emotion processing in schizophrenia and

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TS. A few other studies have looked at brain activity while reasoning about intentions and false-belief, covered last in this section. Additional papers to those presented in the previous section were identified through searches of Web of Science and PubMed (06/2016 – 12/2016) using the search terms *emotion*; *social cognition*; *theory of mind*; *mentalizing*; or *mirror neuron*; plus at least one of *Tourette*; *schizophrenia*; *psychosis*; plus *neuroimaging* or *MRI*. Given that there is a focus on social cognitive tasks, most studies discussed here report fMRI findings. Key findings are summarized in Table 1.

2.2.1. Imitation and motor resonance. A number of studies have reported reduced activation in motor areas that may form part of the MNS (e.g. premotor area) during action observation, when comparing patients with schizophrenia to HC (e.g. Enticott et al., 2008; Mehta et al., 2014a). A review of MNS function in schizophrenia (Mehta et al., 2014b) concluded that most studies interpreted their results as indicative of reduced MNS activity, in association with greater negative symptoms and deficits on social cognitive tasks. However, some studies suggest increased MNS activity. These include studies that have used EEG to investigate mu wave suppression. Essentially, higher mu wave suppression over MNS areas would be indicative of greater activation. This occurs in HC to a certain degree during action observation, and most strongly during action imitation. McCormick et al. (2012) investigated mu suppression in actively psychotic patients and those with residual symptoms versus HC. All participants observed hand movements made by others, and this condition was compared to self-hand movement. More mu suppression (i.e. approx. 20% more MNS activity) was observed over left sensorimotor cortex in actively psychotic patients watching hand movements in comparison to the other two groups.

Thakkar et al., (2014) showed patients with schizophrenia and HC videos of a moving hand pressing a button, a static photo, or a symbolic representation, and they were instructed to observe or execute the depicted button press. When comparing imitative (after viewing the moving hand) to non-imitative action (only static/symbolic representations), HC showed more activation in the MNS (inferior parietal lobe, posterior superior temporal sulcus and IFG). Group comparisons indicated less activity in patients than HC in the posterior STS for imitative actions, but greater activation in the right inferior parietal cortex and posterior STS for non-imitative action. The authors conclude that there is a less differentiated response for imitative action versus non-imitative action in schizophrenia. This could imply that patients are less able to distinguish between actions linked to others and those generated by oneself.

Explanations for reduced MNS activity include reduced attention, reduced neural integrity, or factors related to baseline comparisons. Kato et al. (2011) used magnetoencephalography to compare unmedicated

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patients (mainly paranoid) to HC, while observing jaw movements. Right inferior parietal cortex activity was less specialized in patients, showing greater activity during the control condition. Reduced activation of the MNS system in response to task cues may therefore reflect higher activation compared to HC at baseline. Studies exploring the neural correlates of imitation in TS are lacking. However, a few studies discussed later in this section (Eddy et al., 2016; Eddy, Cavanna and Hansen, 2017) have suggested their findings implicate abnormal neural activity within brain areas associated with the MNS (e.g. inferior parietal area/TPJ; inferior frontal gyrus), when adults with TS complete tests of social cognition.

2.2.2. Empathy and emotion recognition. There is mixed evidence for amygdala hyper- and hypo-activation in schizophrenia during emotion recognition. For example, there can be a reduced amygdala response in patients with paranoia, but an increased response to fearful expressions in patients with flat affect (e.g. Gur et al., 2007). Rasetti et al. (2009) found a deficit in amygdala reactivity to negative faces in schizophrenia, and an alteration in functional connectivity between the amygdala and the subgenual cingulate. However, many studies report elevated amygdala activity in these patients in response to neutral or positive expressions. (e.g. Holt et al., 2006; Kosaka et al., 2002; Surguladze et al., 2006; Mier et al., 2010). The amygdala has also been highlighted in TS studies. Neuner et al., (2010) revealed increased amygdala response in TS to fearful, angry and neutral expressions during judgment of gender. More recently, Eddy et al. (2017) compared the brain activity of adults with TS and HC during mental state judgment versus when making age judgments about the same RMET stimuli. Areas including right amygdala were more active in TS. When patients with schizophrenia undertook a modified version of the RMET, they exhibited greater activity than HC in superior frontal gyrus, superior parietal lobe and left medial prefrontal cortex (de Achával et al., 2012).

Other brain areas exhibiting differential activity in schizophrenia during emotion expression processing are the right TPJ and IFG. Quintana et al. (2001) report right IFG hypo-function during facial emotion matching, and Smith et al. (2015) found both right-side TPJ and IFG hypo-function in schizophrenia during an empathy task which required selection of a basic emotional expression for a character based on a visual scenario. De Achaval et al. (2012) reported reduced activity in the right IFG and TPJ in schizophrenia during face processing, although this was in the context of greater overall brain activity than HC in areas including left superior temporal and middle temporal gyrus, left parietal lobe, left medial frontal gyrus and right insula. Such findings could imply increased engagement of MNS areas, coupled with decreased activity in right TPJ and right IFG. These latter two areas may underpin self-other distinctions (see Eddy, 2016) and control reactions to emotional stimuli (Brown et al., 2012; Mitchell et al., 2008), respectively. In TS, one recent study (Eddy et al., 2017) using

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the RMET (mental state versus age judgments) reported abnormal right-side TPJ and IFG activity. For HC, right IFG activity was greater for mental state judgment than age judgment, but in TS, the inverse pattern was found. This could suggest that emotion recognition is associated with right IFG hypo-function in TS.

The aforementioned findings are also interesting to consider in relation to the observed opposing relationships between right IFG activity and empathy measures reported in schizophrenia. Horan et al. (2014) used IRI scores as a covariate when scanning patients with schizophrenia and HC using fMRI during imitation, execution and observation of both finger movements and facial expressions. Patients exhibited lower PT and empathic concern scores, and a trend towards higher PD. Higher total IRI scores were associated with greater right IFG activity in HC during the observation of emotional facial expressions. However, higher IRI scores were related to lower right IFG activation in patients during this condition. A later study (Horan et al., 2014) supported this finding of contrasting relationships between empathy scores and IFG activity in schizophrenia patients versus HC, although in this study a composite score for the IRI was used.

Overall these findings suggest that in patients with schizophrenia, the amygdala responds to neutral and perhaps positive emotional expressions as if they have negative emotional connotations. There is also evidence for amygdala dysfunction during emotion recognition in TS. In addition, both TS and schizophrenia exhibit atypical patterns of right IFG activity during facial emotion recognition tasks.

2.2.3. Understanding beliefs and intentions. A couple of studies have shown greater activity in the superior temporal gyrus (STG) in patients with schizophrenia versus HC when inferring a story character's intentions from cartoon stimuli (Benedetti et al., 2009; Brüne et al., 2008), and that higher neural activations can be inversely correlated with behavioral performance. When using the AT and comparing social interaction versus random movement, Pedersen et al. (2012) found stronger activity in areas including right IFG and left STS, despite lower behavioral ratings for intentionality in patients than HC. Das et al. (2012) reported less activity in bilateral IFG and right TPJ in patients versus HC for mentalizing AT videoclips minus random movement. This task is yet to be combined with neuroimaging in TS.

Other studies have reported hypo-activation of the TPJ in schizophrenia or TS during mentalizing tasks. Walter et al. (2009) reported this in schizophrenia during a task when there was a need to differentiate between the intentions of characters. However, increased medial prefrontal activity in paranoid subjects during the control condition involving natural physical causality suggested less of a neural distinction in patients than HC when processing causality related to natural processes (e.g. gravity) versus human actions. Notably, a recent study in TS (Eddy et al., 2016) revealed hypo-activation in brain regions including right TPJ during a

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mentalizing task, reflecting less activation difference in patients when reasoning about natural versus human causality. Studies exploring the neural correlates of false-belief have also revealed cortical hypo-function in these patient groups. Dodell-Feder et al. (2015) showed that medial prefrontal activity was reduced during false-belief reasoning in schizophrenia patients with high social anhedonia. Another study in schizophrenia (Lee et al., 2011) used fMRI during a false-belief story task (Saxe and Kanwisher, 2003), which allows brain activity during the processing of mental (false-belief) and physical (false-photo) states to be compared. This contrast showed fewer differences in patients with schizophrenia than HC (i.e. not including medial prefrontal cortex or TPJ). The same task was used by a recent study in TS (Eddy et al., 2016). Contrasting conditions revealed similar findings: less activity difference for false-belief in brain regions including right TPJ.

2.2.4. Summary of neurophysiological and neuroimaging findings. Social cognitive tasks may activate both mirroring and mentalizing networks, leading to complementary information (Spunt and Lieberman, 2012a). However, these systems may be competitive, given that there is anti-correlated activity in these networks in healthy individuals at rest (Fox et al., 2005). Individuals with deficits in social cognition could be hypothesised to have difficulties in engaging one of these systems, or problems in switching between them. One general pattern which may be starting to emerge in schizophrenia and TS is excessive brain activity in areas linked to mirroring, and contrasting hypo-activation in regions thought to underpin mentalizing in response to social cognitive tasks. For example, the findings of McCormick et al. and Thakkar et al. could imply greater motor and/or emotional resonance with others in schizophrenia (especially in association with positive symptoms), and studies reporting hypo-function may have failed to account for greater activation within MNS areas during baseline or control conditions. There are also hints of increased activity in limbic and MNS regions in response to facial expression tasks in TS. In particular, amygdala activity can be elevated during facial expression tasks in both patient groups. Other regions of interest associated with abnormal function in schizophrenia and TS are the right IFG and right TPJ. The possible relationships between these findings and patients' behavioral symptoms are explored next.

3. The potential role of social cognition in patients' symptoms

Patients with schizophrenia or TS can exhibit abnormal performance on some social cognitive tasks which in some cases may be indicative of increased internal simulation of others' actions or emotions, or poor control over MNS activity. On other occasions errors may reflect an over-application or an under-application of mentalizing. This section will discuss how problems with self-other distinction in schizophrenia and TS may

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contribute to symptoms involving emotional dysregulation, paranoia, SA and harmful or socially disruptive behaviors (Table 2).

3.1. Self-other distinction and emotional dysregulation

At times, effective social cognition requires one to mirror, or simulate, the physical or mental state of another person. For example, empathy may be achieved when the observer activates similar neural circuitry to the feeler, gaining a sense of the emotion. However, this mirroring process must be controlled to ensure that a distinction between the self and other is maintained, especially when conflicting mental states must be appreciated at the same time (e.g. during false-belief tasks). Problems with self-other distinctions could lead one to be confused about the origin of a simulated action or emotion. Previous studies suggest that in schizophrenia, and perhaps to a lesser extent in TS, brain areas involved in simulation of others' actions and emotions are readily activated, and there may be poorer control over mirroring processes than in typically developing individuals. Confusing the actions of self and others could contribute to symptoms such as echophenomena, while difficulties distinguishing the self from other people experiencing or expressing emotions could contribute to emotional dysregulation.

In social settings, patients with schizophrenia can be overly sensitive and more reactive to the feelings of other people when compared to HC (Michaels et al., 2014). Dysfunctional emotion regulation (Van der Velde et al., 2015), excessive awareness of others' feelings (Masillo et al., 2012) and a hyper-empathising profile (Brosnan et al., 2010) can be associated with psychosis. Enhanced empathy could also help to explain why some patients with schizophrenia attribute mental states better to others than to themselves (Langdon et al., 1997), or find it easier to reason about their own mental states when taking the perspective of another (Gambini et al., 2004). In relation to TS, while one study reported no difference between patients with TS and HC in terms of interpersonal reactivity (Eddy et al., 2015), preliminary evidence suggests that individuals with TS who exhibit echophenomena demonstrate increased empathy (and grey matter differences within the MNS) when compared to patients who don't report echophenomena (Harrison et al., 2007).

Increased tendencies towards mirroring others could explain high ratings of PD on the IRI in schizophrenia and TS (Table 1). That is, mirroring another person's negative emotion could induce negative emotion in oneself, and a faulty internal attribution could heighten this experience. Longer term consequences could include distress and reduced sense of control over one's own emotions. Emotional dysregulation and associated negative feelings could become intrinsically bound to social cognitive processes within the individual, leading to confusion and unpleasant experiences during social interaction. The right TPJ is part of

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the mentalizing network and has been specifically suggested to underpin self-other distinctions (Sowden and Shah, 2014). Hypo-activation of this region could therefore explain reports of reduced PT in schizophrenia (e.g. Montag et al., 2007) and TS (e.g. Eddy et al., 2015), and problems with self-other distinctions reflected in high PD. However, control over mirroring processes or simulated actions and emotions could also involve the right IFG, an area associated with control of action and related emotional responses (Brown et al., 2012; Kringelbach and Rolls, 2003). For example, in healthy subjects completing an emotion labelling task, increased activity in right IFG is coupled with reduced amygdala activity, implying prefrontal control over emotional responses (Hariri et al., 2000). Previous studies indicate both schizophrenia and TS have can be linked to excessive amygdala activity while viewing of emotional facial expressions. Poorly controlled amygdala activations could lead to conditioned aversive response, in addition to abnormal performance on social cognitive tasks.

The phenomenological experience of schizophrenia highlights a role for negative affect in association with reduced self-other distinction. Henriksen et al. (2012) describes self-disorders in schizophrenia; a sense of diminished identity and first-person perspective. One illustrative case felt upset by people as they would come to close to her; claimed to have no idea who she was; felt like others could see her thoughts; and suffered with social anxiety and SA. Poor self-other boundaries have also been reported in children with a genetic high risk to schizophrenia (Brent et al., 2014). While such severe self-disorders have not been described in TS, these patients do exhibit evidence of increased sensitivity to the actions and emotions of others. In addition to echophenomena, TS can feature ironic or egodystonic socially inappropriate urges, whereby the patient easily identifies remarks or actions that would upset others, but denies intent to hurt or offend, finding such thoughts and urges distressing (e.g. Eddy & Cavanna, 2013). Perhaps acting out socially inappropriate behaviors emphasize conflict between the self and the other, which may counteract the uncomfortable emotional or psychological effects of over-blending (Eddy, 2016).

3.2. Mirroring, mentalizing and paranoia

Paranoid patients feel that other people's intentions are negative, reflecting a tendency towards attribution of intention, and a negative emotional bias. Potential routes towards attributing intention include mirroring processes (i.e. MNS) or cognitive reasoning (mentalizing). Studies in monkeys have shown that they can understand what the end result of an action will be when this is obscured from view (Ulmità et al., 2001), suggesting that the mirroring mechanism allows for predictions about action effects and therefore a form of intention attribution. However, because intentions are derived from the observed action or its closely related consequence, intention and action must match, thus backward inference implies outcomes are intended.

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Alternative means of attributing intention could involve mentalizing about a person's beliefs or desires. Such reasoning can allow for greater flexibility, and the incorporation of knowledge e.g. about a person's traits or past behaviors, allowing for an actor's intention to be different to the consequence of their action. Interestingly, very young children (who may have an operational MNS but not fully developed mentalizing abilities) tend to make attributions about intentions based predominantly on the consequences of actions (e.g. Zelazo et al., 1996). When adults successfully integrate information about beliefs into their moral judgments about people's actions, activity is seen in metalizing areas such as TPJ and precuneus (Young and Saxe, 2008).

Greater reliance on mirroring than mentalizing during social situations could lead one to assume that actions and their consequences are synonymous with the actor's intentions, such that negative consequences imply negative intentions. That is, these patients could show an inflated 'intentionality bias' (Rosset, 2008), and paranoid interpretations of the motives underlying observed behavior. Although such effects have only been explored directly in schizophrenia (Peyroux et al., 2014), both patients with TS and schizophrenia can show a greater tendency than typically developing HC to assume intention based on consequence, as demonstrated by responses to the faux pas task, where frequent identification of an offensive remark is frequently followed by the assumption that offence was intended (Table 1). Such tendencies may have additional implications in relation to judgments of blame and morality. Future research should investigate relationships between the valence of attributed intentions and observed action consequences in these patient populations.

A tendency to attribute intentions, internal negative affect, and poor self-other distinctions could underlie delusions of persecution. Low self-esteem and negative self-schemas are common in patients with persecutory delusions (Kesting et al., 2013; Freeman et al., 2014) and self-blame predicts long term paranoia (Westermann et al., 2013). If self-other boundaries are unclear, we may mistakenly attribute the origin of our negative emotion and project onto others, feeling their emotions (and intentions) towards us are negative. Emotional dysregulation, and sensing a lack of control over our emotions may fuel a sense of being manipulated or controlled by others. In sum, impaired self-other distinctions could lead to not only taking on another's emotion, but also misattributing our own mental state to another person. Behavioral features associated with this latter problem could also include thought insertion, and egocentric errors on social cognitive tasks, helping to explain inconsistent social cognitive performance across studies. Future research could explore whether possible markers of excessive mirroring (e.g. echophenomena) are associated with emotional dysregulation and paranoia.

Feelings of harm in association with relating to other people's mental states could be coupled with a conditioned neural response. For example, excessive amygdala or orbitofrontal activity seen in patients with

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schizophrenia and TS during social cognitive tasks (Table 1) could reflect negative reinforcement given the involvement of these brain regions in emotion related learning (e.g. O'Doherty et al., 2001). Green et al. (2004) report that patients with schizophrenia exhibit a greater automatic pre-attentive response to threatening stimuli, but may then apply attention avoiding strategies (e.g. fewer fixations on angry facial stimuli), reducing the chance for reappraisal. Patients' attempts to rationalize the unpleasant emotional experiences they have in response to social interaction with others may lead them to conclude that the cause is external: others intend to do them harm. In summary, the reported association between unstable self-concept and paranoia (Tiernan et al., 2014), probably reflects a combination of increased intention attribution, negative affect and impaired self-other distinctions.

3.3. Self-other distinction, social anhedonia and hypo-mentalizing

Numerous studies have reported high levels of social anhedonia (SA) in patients with schizophrenia. For example, Blanchard et al. (1998) found that SA in these patients was associated with increased social anxiety. In healthy populations, high SA can be associated with schizotypal personality traits (Rey et al., 2009), paranoia during every-day social interactions (Hooker et al., 2014), and poorer inhibitory control over emotionally salient stimuli (Martin et al., 2012). Therefore increased PD and distressing experiences linked to impaired self-other distinctions, could reduce reward from social interaction. Interestingly, patients with schizophrenia may show blunted affect at the same time as reporting normal intensity of emotional experience. For example, in one study using International Affective Picture System images, 40% of patients with schizophrenia patients found the negative stimuli more negative and arousing than the control group, despite experiencing higher levels of SA (Strauss and Herbener, 2011). Social sensitivity could therefore contribute to negative symptoms such as SA which appear paradoxically like social disinterest or insensitivity.

People with SA may perceive the stressors they face as particularly uncontrollable or overwhelming (Horan et al. 2007). Indeed, excessive mirroring and related emotion contagion could lead social situations to become aversive. The emotional overarousal experienced by patients with schizophrenia when in the company of others may lead to extreme regulatory strategies (Harder and Lysaker, 2013) in addition to avoidant coping, anxiety, depression and social withdrawal (Masillo et al., 2012). Signs of social withdrawal could therefore reflect negative symptoms, or coping strategies linked to positive symptoms. This may explain why ratings on empathy measures do not always correlate with social behaviors as expected. Haker and Rössler (2009) found a positive association between higher PD scores and greater evidence of negative symptoms. Alexithymia in schizophrenia (e.g. Fogley et al., 2014) could be related to a similar kind of emotional 'burnout'. In sum, some

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aspects of social dysfunction in schizophrenia could result from excessive negative affect in response to social stimuli rather than a lack of social cognitive ability *per se*.

Interestingly, SA may be associated with abnormalities in brain areas underlying mirroring and mentalizing. For example, studies have revealed correlations between SA score and cortical thickness in areas including the IFG, postcentral gyrus, supramarginal gyrus and inferior parietal cortex (Wang et al., 2014). Yin et al. (2015) showed that when compared to schizophrenia patients with lower SA, those with high SA exhibited reduced functional connectivity between ventrolateral prefrontal cortex (VLPFC) and the motor cortex, inferior parietal cortex and posterior temporal area when viewing socially positive (versus neutral) videos of emotional expressions. However reduced connectivity could underpin increased SA or some kind of adaptive response. Another study (Germine et al., 2011) showed that patients with schizophrenia and high SA exhibited reduced activity in face processing areas (right fusiform gyrus, medial prefrontal cortex and right superior temporal gyrus) when discriminating between emotional facial expressions. Again, it is not clear whether this hypoactivation reflected SA, or other factors such as attention avoidance.

While positive symptoms can be linked to hyper-mentalizing, negative symptoms can be linked to hypo-mentalizing (Montag et al., 2011). Future studies therefore need to consider where errors may reflect coping strategies or conditioned avoidance, in addition to neurodegeneration or executive deficits. Some evidence of hypo-mentalizing could reflect avoidance of social cognition; a good example being the dual report of high PD but low PT on the IRI, as seen in both schizophrenia and TS (Table 1). Furthermore, when processing facial expressions, patients with TS exhibit hypo-activation within brain mentalizing regions when conscious reasoning about mental states is prompted (Eddy et al., 2016), but greater activity than HC in similar brain regions when mentalizing is not required by the task (Eddy et al., 2017). Poor control over some aspects of social cognition (i.e. automatic mirroring), may lead to conscious or subconscious attempts to reduce or control other aspects of social cognition (i.e. mentalizing).

3.4. Sense of agency and impulsive socially disruptive behaviors

One possible consequence of impaired self-other distinction may be problems determining agency, due to a weakened sense of internal control over one's own thoughts, actions and their consequences. It has been shown that an external locus of control in childhood predicts later paranoia (e.g. Thompson et al., 2011) and altered sense of agency may be a risk factor for the development of psychosis (Hauser et al., 2011). Moritz et al. (2007) compared attribution style in schizophrenia, anxiety, depression and HC. When judging whether negative events were due to people or the situation, paranoid patients were less likely than HC to rate themselves as the

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cause. This could imply decreased internal locus of control and the potential for delusions of control. For a more in depth discussion of sense of agency in schizophrenia, the reader is referred to Nelson et al. (2014). The possibility of atypical sense of agency may in TS is yet to be explored.

Altered sense of agency may be associated with an increased propensity towards impulsive behavior, given that it may be more difficult to learn associations between one's own willed actions and their consequences. A range of impulsive and risky or dysfunctional behaviors are common to both TS (Kurlan et al., 1996; Eddy and Cavanna, 2013a; Wright et al., 2012; Frank et al., 2011; Mathews et al., 2004) and schizophrenia (Hirjak et al., 2013; Desai and Potenza, 2009; Pluck et al., 2013; Hoptman, 2015; Tracy et al., 1996), including self-harm, explosive disorder, antisocial behavior and impulse control disorders (e.g. gambling, hypersexuality etc.). More specific effects may involve sensory symptoms and related motor behavior. For example, tics are viewed as semi-voluntary or involuntary by patients. However, it is worth further considering the potential role of MNS dysfunction in patients' symptoms. Action representations are thought to be subconsciously activated by observation of another's action (Jeannerod, 1999). In TS, the experience of frequent mirroring of others may lead the motor system to feel less under internal control, resulting in reduced sense of agency. A conditioned loss of control over the motor system could contribute to involuntary movements (which have also been observed in drug-naïve schizophrenia e.g. Peralta et al., 2010). Alternatively, if the motor system is readily 'hi-jacked' by external stimuli, having tics could help bring the system under internal control (allowing self-other distinction), and this could develop into a conditioned response.

Perhaps both TS and schizophrenia feature certain compulsive behavioral problems that serve to emphasize self-other differences and enhance sense of agency. One good example of this may be the phenomenon of impulsive non-conformity, as described in schizophrenia. Chapman et al. (1984) constructed the *Impulsive Nonconformity Scale* to measure 'impulsive antisocial behavior' which may indicate a risk factor for psychosis. Patients with schizophrenia or schizoaffective disorder scored higher on the scale than HC, and controls with high scores reported more psychotic or psychotic-like experiences. In another study involving a normative population (Aldebot Sacks et al., 2012), increased self-certainty was associated with greater impulsive nonconformity. Therefore impulsive nonconformity could in some way be protective to the self-concept and sense of agency, perhaps by highlighting self-other boundaries more clearly.

Impulsive nonconformity may lead to signs of inflexibility, challenging or anti-social behavior, or ironic acts that resemble the socially inappropriate urges seen in TS. They could also include self-injurious behaviors. When self-other boundaries are confused, impulsive non-conformity could occur against the will of

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the self in addition to the will of others. This would help to explain egodystonic intrusive thoughts of harming the self or others. In schizophrenia, dystonic command hallucinations can include voices telling the patient to e.g. drink bleach; smash things in the house; shout or swear at other people; jump in front of cars; be violent towards others (Birchwood et al., 2014). Sometimes the instructions are insignificant (e.g. make a facial grimace) but sometimes they are serious violent actions (Hersh and Borum, 1998). While descriptions of tics have referred to 'strict internal commands' (Hollenbeck, 2001), only very rarely have auditory hallucinations been reported in TS (e.g. Kerbeshian and Burd, 1985). Rather than hearing voices, patients report sensory or psychological urges to carry out similar harmful actions, including self-injurious or dangerous behaviors (e.g. touching a hot iron: Leckman, 2003; Cheung et al., 2007) and socially inappropriate actions (e.g. to insult or physically harm others: Kurlan et al., 1996; Eddy and Cavanna, 2013a). Socially inappropriate urges in TS create direct conflict based on the intentions and desires of the self and the other. The ironic nature of these urges is emphasized by their association with an awareness of other people's mental states, and a sensitivity to others' negative emotions. Based on the action-goal implications of the MNS, acting in a certain way implies a matching intention to perform that act. Carrying out the prohibited action therefore yields attribution of a mental state directly at odds with the mental state of the victim, emphasizing self-other distinction.

In summary, socially disruptive and inappropriate symptoms in TS and schizophrenia may reflect manifestations of the patient's struggle to achieve self-other distinction, and perhaps even help to maintain sense of self and self-agency. While they may be partly adaptive for the self-concept, they are clearly destructive within society and impair the patient's social integration.

4. Conclusions, limitations and directions for future research

This narrative review has discussed the evidence for deficits in social cognition in schizophrenia and TS, and shown that both patient groups can exhibit complex patterns of impairment across social cognitive tasks. There is mounting evidence for differences in the way that these patient groups respond to tasks that involve mirroring processes, in a way that may imply a greater tendency to engage the MNS than HC. There is also a tendency for some patients in these populations to over-apply mental states, particularly intentions, when stimuli are ambiguous or neutral; but to also exhibit evidence of hypo-mentalizing, particularly during tasks involving prompted mentalizing. Attributed intentions are also frequently associated with a negative affective interpretation. Overall, there are intriguing similarities in relation to how both patient groups respond to tasks requiring mirroring versus mentalizing. The ability to distinguish mental states relating to the self and other may be impaired in both TS and schizophrenia, contributing to a range of symptoms, including impulsive and

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compulsive socially disruptive behaviors. However, there are complexities in interpreting task performance and the basis for patients' behavioral deficits. The limitations of reviewed studies will be discussed first, before more specific directions for future research to help answer these queries are covered. Finally, the overall implications of the review findings are considered.

4.1. Limitations

Deficits on social cognitive tasks could reflect a range of incidental confounds. These include medication effects such as fatigue, and distractions due to primary symptoms associated with the disorder (i.e. tics or hallucinations). Symptoms such as paranoia and suspiciousness may pose additional challenges to assessment. Some schizophrenia samples exhibit deficits in social cognition that are no more significant than those found in patients with depression (e.g. Kettle et al., 2008), raising the limitation that few studies have included a clinical comparison group to help control for mood disorder or anxiety. In addition, while there is scant data in relation to the influence of dopaminergic or serotonergic agents on social cognitive tasks in these patient populations, these agents could influence task performance (e.g. Savina and Beninger, 2007). Other mediating factors include general cognitive or neurological impairment (e.g. Wang et al., 2015; Kantrowitz et al., 2014). The inclusion of matched control tasks and better clinical characterisation of samples would be helpful.

Problems reflecting upon and expressing emotion have been frequently highlighted in schizophrenia (e.g. O'Driscoll et al., 2014) and alexithymia could contribute to deficits in social cognitive tasks. The relationship between social cognitive performance and alexithymia requires much more thorough exploration in schizophrenia, TS and other disorders. However, it should be considered that (in a similar way to that proposed for SA) alexithymia could reflect emotional confusion as a result of poor control over mirroring and emotional contagion; or be a reactive mechanism to counteract emotional dysregulation, whereby the patient conditions themselves against focusing on emotional stimuli to counteract problems with feeling overwhelmed. Selective attention to social cues has been suggested to result in evidence of hypo-mentalizing (e.g. Combs et al., 2008). This raises the point that more generally, some of the problematic abnormal behaviors seen in psychiatric conditions could actually reflect coping mechanisms rather than the primary source of dysfunction. Comparing coherent subgroups with heterogenous patient samples (e.g. based on predominant symptoms or symptom severity) could be beneficial.

The complex pattern of deficits across tasks could partly reflect inconsistencies in the level of error analysis undertaken. Those studies that report a breakdown or errors on control questions, or scores across

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subscales of a measure tend to offer most insight. In addition, some discrepancies across studies could reflect differences in the task instructions linked to explicit versus implicit emotion processing. For example, Li et al., (2010) reported abnormal fusiform gyrus activity in schizophrenia only during explicit recognition of emotion and not for tasks involving implicit emotion processing (e.g. during age judgments). In TS, the same stimuli can elicit hypo- or hyper-activation in brain mentalizing areas depending on task instructions (e.g. Eddy et al., 2017). In schizophrenia, MNS activations may also be greater during implicit processing (e.g. Quintana et al., 2001).

The findings of some studies on facial emotion recognition in schizophrenia add a very interesting caveat to this debate. Davis et al. (2000) compared paranoid patients to non-paranoid (disorganized) patients and HC on a task involving recognition of genuine and posed emotional facial expressions. They found that paranoid patients showed enhanced recognition of genuine expressions, and only performed more poorly than HC when expressions were posed. This study was motivated by the findings of La Russo (1973), who reported that some aspects of emotion recognition in paranoid patients appear to be enhanced, such that they show a greater sensitivity to non-verbal cues when these are genuine. It is likely that the majority of studies considered in this review used actors posing expressions, raising issues around the validity of laboratory tests of social cognitive ability.

One fundamental problem raised earlier in this review relates to control tasks and baselines. Sometimes patients with TS or schizophrenia appear to exhibit hypo-activation in contrasts when compared to HC. However, this could reflect abnormally high activity in the baseline condition or towards neutral stimuli used as control tasks. For example, Modinos et al. (2015) compared patients with first episode schizophrenia and ultra high-risk individuals to HC, and discovered increased brain activity in areas including the insula, amygdala and IFG for neutral stimuli, which likely contributed to the result of hypo-activation versus HC for emotional stimuli. Another problem relates to amygdala activations. This structure has been shown to activate abnormally in TS and schizophrenia in some studies, but not in others. As the amygdala rapidly habituates to stimuli (e.g. Phillips et al., 2001) it is possible that some studies may miss this effect. There is also the possibility that earlier and later amygdala activations may depend on different (i.e. cortical and subcortical) routes. This is worthy of exploration but has, as yet, received little attention.

It is of course imperative for studies of social cognition to consider the possible influence of comorbid autistic spectrum disorder within patient populations including schizophrenia and TS (e.g. Sverd et al.,1991; Lugnegård et al., 2015). While some characteristics (e.g. hyper-mentalizing) may appear less likely to be

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attributable to autism, in other cases the situation is less clear. Determination of the primary disorder responsible for social cognitive problems could be complicated by factors such as the varying severity of symotoms across the autistic spectrum, and the possibility that some behavioral difficulties may arguably constitute an integral part of multiple diagnostic entities. These difficulties further emphasise the potential benefits of research directly comparing the responses of individuals with autistic spectrum disorders to those of patients presenting with social cognitive problems but diagnosed with other psychiatric disorders.

It has been suggested that while one of the most popular concepts referred to in neuroscience, the mirror neuron theory is not without limitations (see Kilner and Lemon, 2013), including the tendency for studies to rely on fMRI data rather than direct evidence from single cell recordings from within the numerous brain areas now thought to form part of the MNS. Furthermore, it could be argued that motor predictive coding may allow for similar functions (Kilner et al., 2007). While it is currently unclear how self-other distinctions may relate to the MNS, one possibility is that activity within brain areas containing mirror neurons may vary in order to signal internally or externally generated actions, hence contributing to self-other differentiation. While it is currently unclear how self-other distinctions may relate to the MNS, one possibility is that activity within brain areas containing mirror neurons may vary in order to signal internally or externally generated actions, hence contributing to self-other differentiation. Alternatively, self-other distinctions could be underpinned by brain regions which may not necessarily contain mirror neurons (e.g. right TPJ), but which form part of a system involving other brain regions that do. In such a system, dysfunction within brain regions that actually perform the differentiation function rather than the mirroring function would likely provide a plausible explanation for the difficulties of patients with schizophrenia or TS, especially when these patients do not show deficits with abilities such as imitation, but do exhibit evidence of difficulties in disentangling mental and physical states relating to the self and other. In other words, social cognitive difficulties in some disorders may not involve dysfunction of mirror neurons per se, but rather abnormalities within brain areas that allow one to interpret or control the output of mirror neurons.

The challenges involved in this narrative review included the breadth of literature available and the scope of this topic. One specific problem was the current lack of studies involving children with TS, and research in this area will make a valuable contribution given the neurodevelopmental nature of TS and the possibility that adult patients may comprise a more severe subpopulation. It is also the case that the majority of existing research on social cognition in TS comes from the same lab, which could influence the variance across studies and emphasises the importance of further research of this nature. Manual search was used in order to

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maintain focus within specific themes, and it is possible that a more refined or systematic approach may have reduced the potential for bias. However, the review findings inform the development of novel hypotheses based on existing literature. Therefore many of the suggestions raised here remain to be directly tested through empirical study or meta-analyses.

4.2. Unanswered questions for future research

Schizophrenia and TS fall across a fairly wide spectrum of severity, and it has been suggested that different subtypes of schizophrenia could be related to different types of social cognitive impairment i.e. paranoia is associated with hypermentalizing whereas patients with more negative symptoms demonstrate hypomentalizing (Frith, 2004). In addition, a recent study indicated that disorganized symptoms may be more closely linked to social cognition than other symptom clusters, including negative symptoms (Minor and Lysaker, 2014). Studies involving larger samples of patients more clearly characterised in terms of e.g. subtype, predominant symptom cluster, treatment and symptom status, will enable better exploration of relationships between social cognitive performance and specific symptoms. Moreover, although schizophrenia and TS can be considered neurodevelopmental disorders, relatively few within-participant longitudinal studies have been conducted. In order to understand the mechanisms driving patients' social cognitive performance, and identify related clinical risk factors and prognostic markers, it is essential to study the progression of impairments within individuals, especially in populations with such heterogeneous symptom expression. Longitudinal studies will be particularly useful in determining whether social cognitive deficits are due to different underlying problems at different stages. For example, the findings of this review may suggest that earlier stage deficits indicate excessive mirroring and emotional dysregulation, while later impairments reflect conditioned aversive or avoidant coping responses, or in schizophrenia, the result of neurodegenerative processes. Such studies will also be useful in determining the causal relationship between behavioral problems, neural changes and social cognitive impairments. To the authors' knowledge there are not yet any published studies exploring theory of mind in children with TS. Longitudinal studies involving children at risk of tics or schizophrenia (e.g. with symptomatic relatives) could reveal that social cognition is a risk factor for the development of these disorders.

Differences in application of the mirroring and mentalizing systems could explain related symptoms in other disorders such as OCD. Many of the negative intrusive thoughts in OCD contain similar content to themes apparent in command hallucinations and socially inappropriate urges. While there are many similarities in the symptoms of TS, schizophrenia and OCD which could reflect right TPJ dysfunction (Eddy, 2016), thought action fusion (TAF) may be of particular interest. This describes the tendency to believe thinking about

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something can be causal to it happening (i.e. a thought is like an action: Salkovskis, 1985), and is thought to be a primary source of symptoms in OCD (see Berle and Starcevic, 2005). TAF therefore appears to involve perceiving an abnormally strong association between one's internal thoughts and external actions or events. Perhaps people who demonstrate TAF show a tendency towards over-use of mirroring mechanisms to explain the reasons for actions and events, assuming the action follows an intention. Moral TAF has been described, which links negative thoughts (e.g. about harm occurring to someone) with negative self-attributions about morality. These attributions would be based on a backward inference about the imagined negative action, which can only be assumed to arise from negative intention. Patients with TAF may carry out compulsive actions (e.g. arranging objects) to reduce the likelihood that the negative event will occur. Perhaps these goal-directed action compulsions allow the patient to use their tendency towards TAF to construct an opposing backward inference model (i.e. I did this positive action therefore my intention is positive, so now no harm will occur) that reassures them of their intention and also their physical sense of agency? Functional connectivity studies comparing activation in the MNS and mentalizing networks in individuals with TAF may prove to be informative.

Another interesting question arises when focusing on the differences between TS and schizophrenia. Despite many apparent similarities in terms of social cognition and symptom profiles, people with schizophrenia clearly exhibit much more evident psychosis. The reasons for this could lie in other observed differences, one of which is the lack of more obvious motor disorder in schizophrenia. It has been suggested that motor tics could be protective in terms of maintaining a sense of physical self and enhancing self-other distinction (Eddy, 2016). The motor system and basal ganglia in particular may have an important role to play in terms of behavioral and neural compensation processes. For example, the putamen is larger in boys with TS than HC (Roessner et al., 2011), and increased putamen size in schizophrenia has been linked to symptom reduction (Buchsbaum et al., 2003). Moreover, findings from prospective, longitudinal studies of birth cohorts have identified motor delays and impairments as key antecedents of schizophrenia (see Macmanus et al., 2014). The relationship between motor dysfunction and social cognition has been highlighted in multiple disorders including Huntington's disease (e.g. Eddy et al., 2014; Eddy and Rickards, 2015), and Parkinson's disease (e.g. Buxton et al., 2013; Marneweck et al., 2014). Future studies should include motor function assessments in order to explore the relationship between social cognition, psychosis and a range of motor signs including tics.

Dopamine dysfunction is an additional factor that may be linked to atypical social cognition in both TS and schizophrenia. This neurotransmitter is implicated in motor functions and reward related behaviors. It could therefore interact with emotion related learning and perhaps increase the tendency towards activation of brain

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motor areas. In addition to considering the impact of medications that alter dopamine, studies should consider the relationship between dopamine and the specific type of social cognitive problems exhibited by patients. It is possible that elevated dopamine may contribute to hyper-mentalizing, (see e.g. Abu-Akel and Shamay Tsoory; Brunet Gouet and Decety, 2006), perhaps through increasing the salience of social stimuli (Rosenfeld et al., 2011). While high dopamine has been suggested to be linked to hyper-mentalizing in schizophrenia (Mier and Kirsch, 2017), TS (Eddy and Cavanna, 2015) and other disorders (e.g. Huntington's disease: Eddy and Rickards, 2015), perhaps reduced states of dopamine (as in e.g. Parkinson's disease) are more likely to be linked to hypo-mentalizing. In TS, it is known that tic severity fluctuates markedly over time, and this could be related to changes in tonic or phasic dopamine levels. Pharmacological challenge studies involving brain motor regions that may contribute to the MNS may offer insight, as would studies involving the role of dopamine in and reward related learning in association with social stimuli in schizophrenia and TS.

More broadly, this review has raised questions pertaining to the role of structures such as the TPJ and IFG in relation to the mirroring and mentalizing networks. The right IFG region has been implicated in the control of actions and emotions (Aron et al., 2004; Brown et al., 2012), reversal learning (Kringelbach and Rolls, 2003) and speech production (Rizzolatti et al., 2009; Molnar-szakacs et al., 2005) in addition to the MNS. Indeed, a meta-analysis demonstrated that in typically developing individuals, subregions of the IFG may be specialised for empathy, action control and language functions (Liakakis et al., 2011). A range of problems with social cognition, behavioral control and language functions (perhaps including inner speech) could therefore be related to IFG dysfunction. However, the TPJ (see Eddy, 2016) may also be relevant, given its suggested role in self-other differentiation for actions and emotions (right side), and language comprehension (left side). Even in healthy high-functioning individuals, further work is needed to establish whether mirroring and mentalizing processes are necessarily independent or mutually inhibitory. Variation in behavioral symptoms linked to social cognition across psychiatric disorders could reflect subtle differences in structural and functional connectivity or the automaticity and control of the neural networks underlying mirroring and mentalizing.

4.3. Conclusions

A review of social cognition in schizophrenia and TS highlights a range of similarities in both performance on social cognitive tasks and in clinical symptoms. Exploration of study findings suggests that processes associated with mirroring and mentalizing could be disrupted in both disorders. More specifically, patients within both populations can exhibit tendencies towards greater or less controlled MNS activations, and perhaps related under-application of mentalizing processes, leading to impaired self-other distinction. Such

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problems could be linked to a range of behavioral symptoms, including emotional outbursts, motor signs such as echophenomena and perhaps even some of the more serious and socially disruptive and harmful acts that can present in these disorders. If this is the case, it may be therapeutically beneficial to attempt to correct problems with self-other distinctions in these patients via for example, cognitive behavioral therapy. Alternatively, neurostimulation techniques could beneficially modulate activity within the MNS or mentalizing systems reducing symptom severity, or even the development of more serious psychosis. Such techniques are now beginning to be applied with a view to enhancing social cognition in schizophrenia (e.g. Rassovsky et al., 2015).

In addition to highlighting how investigating social cognition can offer insight into the neuropsychological basis for patients' symptoms and therefore guide intervention, this review has shown that comparing different groups of patients could help us to identify cognitive styles or attribution biases that constitute risk factors for the development of problem behaviors across multiple psychiatric disorders. While the possible use of self-other distinction as a marker will require careful operationalisation of this construct, identification of such markers can inform the development of useful dimensional screening measures for use in healthy and clinical populations.

Examining the differences in symptom profiles across disorders with similar neuropsychological deficits is also likely to provide insight into markers of resilience of potential compensation mechanisms. Importantly, some impairments highlighted by socio-cognitive tasks and related behavioral symptoms could reflect coping strategies, conditioned responses or neural reorganisation, issues that have been frequently neglected in previous literature. Therefore in addition to refining our tasks and assessments, we must bear these possibilities in mind when interpreting identified impairments. Considering the mechanisms underlying any existing effective compensatory strategy used by patients could be informative in guiding therapeutic interventions that help maintain the stability of the self. Perhaps in time more psychiatric symptoms will be understood to reflect an over-compensating brain, and a somewhat ironic but inevitable product of adaption.

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None.

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Table 1.
Key findings on social cognitive tasks in schizophrenia and Tourette syndrome

Task (section)	Schizophrenia	Tourette syndrome
Imitation and Mirror Neuron System (MNS) tasks (2.1.1./2.2.1)	• Reduced imitation and emotion contagion during social interaction and when observing others' movements, especially if more negative symptoms (Kupper et al., 2015) • This is not linked to empathy (Haker & Rössler, 2009) • MNS activity less specific to imitation context and possibility of greater MNS activity during baseline and control tasks (e.g. Thakkar et al, 2014) • Greater MNS interference from observed movement if more positive symptoms (Nakagawa and Hoshiyama, 2015)	• More interference from observed biological motion when producing conflicting movements to those observed (Jonas et al., 2010) • Activity differences in brain areas associated with the MNS during social cognitive tasks (Eddy et al., 2016;2017)
Recognition of basic emotional facial expressions (2.1.2./2.2.2)	• Deficit worst on first generation antipsychotics (Kohler et al, 2010) • Tendency for paranoid to over-attribute negative emotions (Behere et al, 2011) • Deficit for neutral expressions (e.g. Kohler et al., 2003) • Mixture of findings for amygdala activity, including hyper-activation to neutral expressions (e.g. Holt et al, 2006)	• No deficits in basic emotion recognition in uncomplicated TS (Drury et al., 2012) • possible amygdala hyper-sensitivity to these stimuli (Neuner et al., 2010)
Reading the Mind in the Eyes Test (2.1.2./2.2.2.)	• Deficits associated with social anxiety, constricted affect and lack of close friends (Irani et al., 2006) • More impaired if more severe auditory hallucinations (Rominger et al, 2016). • Errors for neutral mental states (e.g. Prevost et al, 2015).	• Behavioural differences are mild (Eddy et al., 2011) • More activity in lateral orbitofrontal cortex, posterior cingulate, right temporo-parietal junction and right amygdala during the task (Eddy et al., 2017)
Empathy/Interpersonal reactivity (2.1.2./2.2.2.)	• Lower PT related to poorer everyday function (Smith et al., 2012) • Lower PT and higher PD (e.g. Montag et al, 2007). • Higher total IRI scores were associated with greater right IFG activity in healthy controls during the observation of emotional facial expressions (Horan et al, 2014)	• Self-reported reduced cognitive perspective taking in everyday life, but increased distress when witnessing other people in distressing situations (Eddy et al., 2015)
Non-literal language/prosody (2.1.2-2.1.3./2.2.2.)	• Poor understanding of sarcasm, lies and paradoxical remarks from video-clips (e.g. Kosmidis et al., 2008). • Impaired perception and expression of emotional prosody (e.g. Hoekert et al, 2007). • Poor sarcasm perception linked to high PD (Sparks et al, 2010) • Impairments with prosody recognition correlate with deficits in	• Less accurate interpretation of written sarcastic and metaphorical marks than controls (Eddy et al., 2010b). • No deficits on spoken emotional prosody task (Drury et al., 2012).

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	pitch perception (Kantrowitz et al, 2014).	
Faux pas task (2.1.3./2.2.3.)	• Deficits in understanding faux pas but sometimes also errors on comprehension checks (e.g. Lam et al., 2015; de Achával et al., 2010). • Poor performance can be linked to conversion in at-risk population (Zhang et al., 2016). • Poorer performance associated with reduced grey matter in VMPFC versus controls (Hooker et al., 2011). • Increased tendency to interpret faux pas as intentional (Shur et al., 2008)	• More errors and false alarms than controls but good performance on comprehension questions (Eddy et al., 2010a; Eddy et al., 2011; Channon et al, 2012) • Increased tendency to interpret faux pas as intentional (Eddy et al., 2010a)
Humorous cartoons (2.1.3.)	• Selective deficits understanding jokes involving mentalizing but not slap stick style jokes in patients with paranoia or passivity (Corcoran et al., 1997). • Humour appreciation can be associated with severity of positive symptoms (Stratta et al., 2007)	• Selection of more literal responses when explaining why a cartoon is meant to be funny. Most errors for cartoons featuring sarcasm. Normal performance for slapstick cartoons (Eddy et al., 2011)
False-belief tasks (2.1.3./2.2.3.)	• Reduced medial prefrontal activity during false-belief reasoning in schizophrenia patients with high social anhedonia, and this correlated with everyday social functioning (Dodell-Feder et al, 2015) • Greater right TPJ activity during false-belief task linked to better emotion management (Dodell-Feder et al, 2015) • Errors on control questions (Scherzer et al., 2015) • Performance linked to positive and negative symptoms and IQ (Pickup & Frith, 2001)	• Intact understanding of false-belief (Eddy et al., 2010a; Eddy et al., 2016) and deception (Channon et al., 2004) • Hypo-activation of brain areas involved in mentalizing (e.g. right temporo-parietal junction, posterior cingulate) during false-belief task (Eddy et al., 2016)
Animations task (2.1.3./2.2.3.)	• Deficit on mentalizing and simple interaction animations, longer answers on random (Russell et al, 2006) • Lower intentionality and appropriateness than controls linked to negative symptoms and functional capacity (Ventura et al, 2015). • Dysfunction in inferior frontal gyrus and superior temporal sulcus during this task (e.g. Pedersen et al., 2012)	• Tendency to attribute intentions and mental states to animations showing random movement (Eddy & Cavanna, 2015)
Gaze direction tasks (2.1.3.)	• Deficit on using gaze to infer beliefs and desires task linked to poor insight into own symptoms (Zhang et al, 2016). • Greater evidence for impairment on second order affective tasks involving gaze (e.g. Ho et al, 2015).	• Difficulties making judgments about envy and gloating based on direction of eye gaze and facial expression (Eddy et al., 2011)

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Table 2.

Behavioural symptoms in schizophrenia and/or Tourette syndrome that may be related to impaired self-other distinctions

Related social cognitive impairment	Symptom/behavioural problem	Result of impairment or compensatory?
Poor differentiation between the mental states of self and other	Socially inappropriate symptoms*	Probably compensatory
	High personal distress	Result
	Low perspective taking/avoidance of mentalizing	Possibly compensatory
	Emotional dysregulation	Result
	Impulsive nonconformity	Probably compensatory
	Command hallucinations†	Result (egodystonic urges may appear like impulsive non-conformity)
	Social anhedonia†	Result or possibly compensatory
	Egocentricity†	Result
	Alexithymia†	Possibly compensatory
	Thought insertion†	Result
	Delusions of control†	Result
Poor differentiation between the mental states of self and other/hyper-mentalizing/intention attribution based on observed actions	Paranoia†	Result
Basic intention attribution based on observed actions/events	Thought action fusion	Result
Poor differentiation between the physical states/actions of self and other	Tics*	Probably compensatory
	Echophenomena*	Result

**Symptoms that appear most characteristic of TS; †symptoms that appear more characteristic of schizophrenia*

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Highlights

- Schizophrenia and Tourette syndrome present notable similarities in social cognition
- There is shared dysfunction in temporo-parietal junction and inferior frontal gyrus
- Both disorders feature increased mirroring of emotion and decreased mentalizing
- This pattern may underlie multiple symptoms including socially disruptive urges
- Comparing disorders reveals common neurocognitive risk factors for social dysfunction

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