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Indian Psychiatric Society Publication

Social Psychiatry

Principles & Clinical Perspectives



Editors

Rakesh K Chadda • Vinay Kumar • Siddharth Sarkar

2nd Edition

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CHAPTER 7

Concept, Relevance and Management of Social Cognition Impairments in Psychiatry: A Biopsychosocial Approach

Urvakhsh Meherwan Mehta, BN Gangadhar

SUMMARY

Most psychiatric disorders have clinical presentations within a social or interpersonal framework. Apart from subjective distress, the reasons for seeking help include apparent problems in social behavior and the resulting socio-occupational dysfunction. Over the last few decades, scientists from across diverse fields including mental health professionals, anthropologists, evolutionary biologists, neuroscientists and developmental biologists have identified the critical contribution of social cognition to such social behavioral problems. We have now begun to identify a connection between social brain networks, social cognition, and social behaviors in these psychiatric disorders. This chapter focuses on understanding the concepts, dimensions, clinical relevance, assessment and measurements, and management of social cognition impairments in a clinical setting. These sections are guided by a bio-psychosocial framework of understanding the connections from the social brain to social behavior through social cognition. Management strategies including cognitive therapies, neuromodulation therapies, novel pharmacotherapies, and traditional life-style practices such as yoga are reviewed and recommendations for clinical practice and future research are discussed.

BACKGROUND: SOCIAL BRAIN TO SOCIAL BEHAVIOR THROUGH SOCIAL COGNITION

Behavioral problems in the social or interpersonal context are common manifestations of a broad range of neuropsychiatric disorders, ranging from developmental (e.g. autism) to degenerative (e.g. frontotemporal dementia) in their origins. The disturbances can broadly be understood as positive phenomena, where patients exhibit undesirable behaviors or negative phenomena, characterized by a lack of desired behavior. Such social behavioral disturbances are related to an underlying deficit or deviation in how the brain processes social information. This ability is also referred to as social cognition, and social neuroscience experiments have demonstrated distinct brain networks underlying these unique higher order cognitive processes.

While a substantial proportion of our social cognition is hard-wired, guided by genetic factors that shape our neuronal networks, the role of social environment (parents, society,

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and culture) in nurturing its development and expression cannot be underplayed. A common feature across neuropsychiatric disorders is the huge quantum of disability in the domains of personal, family, occupational, and social domains of real world functioning of an individual (Vos et al., 2012). Social cognition impairments and the related social behavioral problems are strong predictors of this commonly observed real world functional disability (Fett et al., 2011). Here, it is important to note that functional disability is a multifactorial construct and there could be other determinants independent of social cognition. Nevertheless, this social chain **(Fig. 1)** starting from the social brain shaping social cognition, social behavior, and real-world functioning is often deficient or deviant in individuals with severe psychiatric disorders; a classical example being that of schizophrenia. Through this chapter, we provide a succinct overview of social cognition domains, its neural underpinnings, clinical relevance, and emerging novel management strategies.

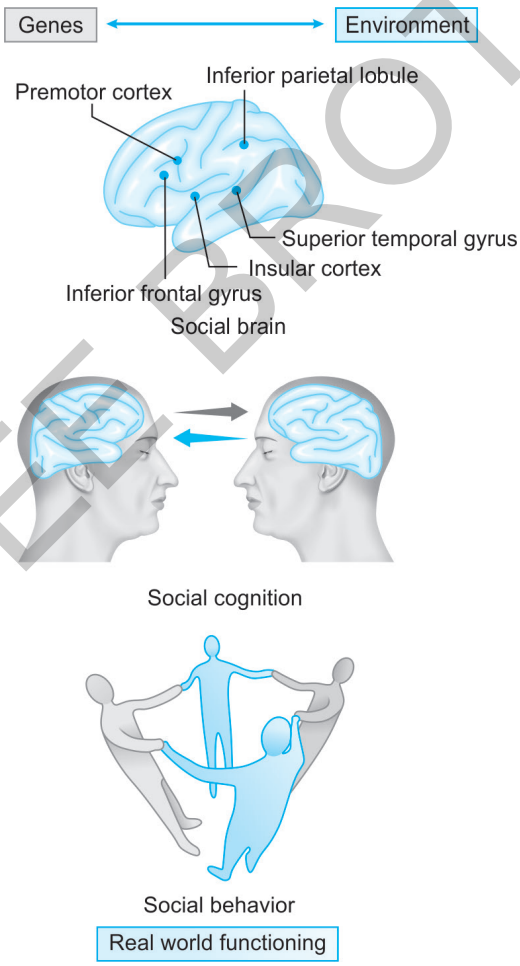

Fig. 1: The social chain leading from social brain networks through social cognition to social behavior and functional outcomes.

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CONCEPT AND DOMAINS OF SOCIAL COGNITION

Many of our day-to-day experiences are centered on social cognition like our ability to process socially relevant information within an interpersonal context. These include, but are not restricted to empathy, perspective taking, trust, cooperation, altruism, deception detection, pretense, metaphors, irony, and social blunders or faux pas. Social cognition experiments began in the late 1960s as psychological investigations into the social mind (Heider, 1967), and extended to examining developmental milestones and ontogeny of these processes (Chandler, 1976). At the same time, evolutionary psychology approaches were used to understand the phylogeny of these abilities by identifying comparative processes in social animals like non-human primates (Premack & Woodruff, 1978). The next decade saw its implementation in clinical populations by examining social cognition impairments in children with mental retardation (Blacher, 1982) and adults with traumatic brain injuries (Wais, 1982). However, it was much later that social cognition was seriously examined in disorders such as autism (Dawson & Fernald, 1987) and schizophrenia (Corrigan & Green, 1993), which today are considered as social brain disorders (Burns, 2006a).

Earlier meetings of the National Institute of Mental Health (NIMH)—Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) initiative (Green et al., 2005, 2008) recommended a set of sub-domains of social cognition to be examined in future studies. Over the last decade, a series of neuroscience-driven experiments have now started to give concerted information regarding neural networks underlying social cognition (Green et al., 2015). We have identified the following four domains of social cognition, based on expert consensus of earlier NIMH meetings and recent evidence from social neuroscience experiments in humans:

1. **Social cue perception:** Social cues are sensory stimuli appearing within a social framework. The perception of these social cues is vital for recognition of identities and emotions in faces and voice, as well as in interpreting gestures and body language (biological motion of head, hands, other body parts, eye contact, lip-reading, etc). This involves initial processing of social information, which later culminates in the precise appraisal of beliefs and intentions in other individuals. The superior temporal sulcus and nearing fusiform face area and fusiform body area are important cortical structures that enable these functions (Allison et al., 2000). The amygdala, an important part of the limbic cortex has a specific role in identifying aversive stimuli that evoke disgust. Factor analytical studies (Mehta et al., 2014b) on social cognition in schizophrenia and healthy individuals also identify a distinct socioemotional processing component that has loadings from scores on tests of social cue detection and facial emotion recognition.
2. **Theory of mind (ToM):** This is a unique cognitive ability present only in higher nonhuman primates and humans. This concept was introduced by primatologists and psychologists Premack and Woodruff in their 1978 article "Does the chimpanzee have a theory of mind?" as a capacity to impute mental states to oneself and to others. This ability was viewed as a "theory", first, because such states are not overtly observable, and second, because it can be used to make predictions, specifically about the behavior of other individuals (Premack & Woodruff, 1978). Several terminologies have been coined that refer to ToM or to certain aspects of the mental state inferential process. Mental state attributions, mentalizing

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ability, meta-representations, representational or Machiavellian intelligence, reflexive awareness, folk psychology, taking the intentional stance are few such terminologies (Burns, 2006b, Harrington et al., 2005). The literature identifies the possession of a ToM as manifest in imitation, self-recognition, social relationships, role taking, deception and perspective taking (Heyes, 1998). ToM is defined as the ability to infer intentions, dispositions, and beliefs of others (Green et al., 2008). There are two neural networks that enable our processing of ToM: (a) *the mentalizing network,* which subserves a controlled pretense or simulation-based reflective processing of others' thoughts and is comprised of the medial prefrontal cortex, posterior cingulate cortex, precuneus, and the temporoparietal junctions, and (b) *the mirroring or experience sharing network* comprising of the premotor, inferior frontal, inferior parietal and insular cortices, which helps in a more automatic or a spontaneous embodied simulation driven understanding of others' thoughts. This was also identified as an independent cognitive construct in schizophrenia and healthy subjects using factor analyses (Mehta et al., 2014b). Moreover, experiments in untreated schizophrenia subjects have revealed a significant association between poor ToM performance and reduced mirror neuron system activity (Mehta et al., 2014a).

3. **Emotion regulation:** Our ability to achieve optimal control over our emotions is a dynamic interplay between the generation of emotions and the cognitive appraisal we employ to understand the emotion and respond appropriately (Green et al., 2015). This involves strategies that permit us to generate differing interpretations of emotional stimuli and then regulate how and when we experience and choose to express or respond to these emotions. These complex processes are a function of *emotion generating and experiencing centers* like the amygdala, insula, ventral striatum, thalamus and anterior cingulate, and the *emotion regulation centers* like the dorsolateral prefrontal cortex (maintenance of emotion interpretations in working memory), the ventrolateral prefrontal cortex (selection and inhibition of interpretations) and the dorsomedial prefrontal cortex (self-reflective processes for elaborating the meaning of emotional stimuli).
4. **Attributional styles:** This refers to explanations people generate about possible causes of good and not-so-good events in their lives. It is understood as the ubiquitous tendency to explain the cause of social actions in terms of oneself or others, or the context of the event (Bentall et al., 2009). While individuals experiencing depression tend to have a strong self-blaming bias (attributing negative events to oneself), those with paranoid delusions tend to have a strong self-serving bias (attributing negative events to other people). Transdiagnostic neuroimaging experiments reveal the contribution of brain regions involved in *automatic interpretations* of intentions of others that include the mirroring network (premotor cortex, inferior frontal gyrus and inferior parietal lobule) and those involved in *motivated behaviors* (dorsal striatum, cerebellum and anterior cingulate), which are thought to underlie our attributional biases (Blackwood et al., 2003; Hao et al., 2015; Park et al., 2009; Rose et al., 2014).

CLINICAL RELEVANCE OF SOCIAL COGNITION DEFICITS IN PSYCHIATRY

The last decade has witnessed an exponential increase **(Fig. 2)** in the number of published studies on social cognition, especially from clinical and neuroscience perspectives. This is

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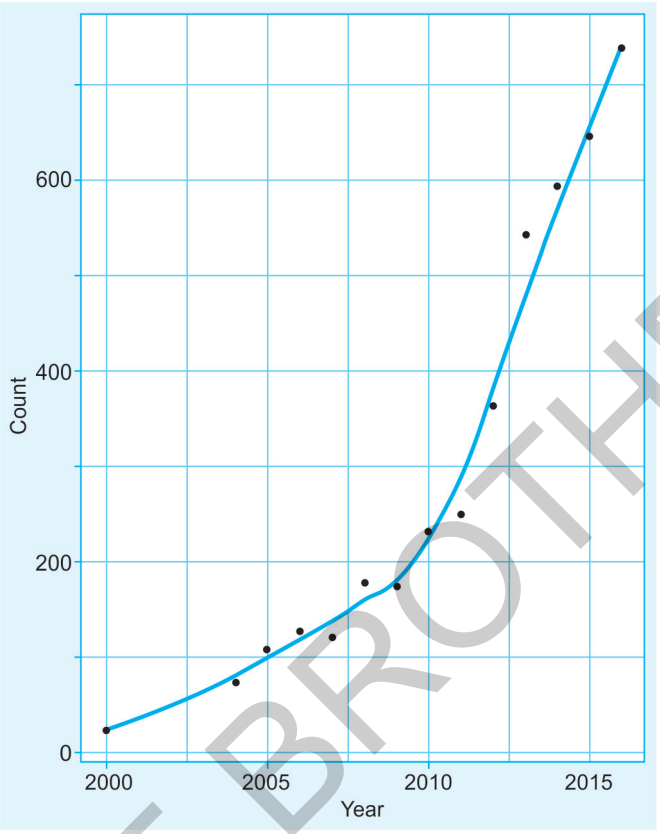

Fig. 2: Number of publications with "social cognition" in the title or abstract in PubMed increasing over the years.

perhaps because of two fairly consistent observations—(a) deficits in social cognition are related to real-world functional disability in patients with severe psychiatric disorders like schizophrenia (Fett et al., 2011) and bipolar disorder (Green, 2006) and (b) these deficits are putative biomarkers for psychiatric disorders (Derntl & Habel, 2011). We will discuss in brief evidence to support these observations, taking schizophrenia as an example.

1. **Relation to functional outcomes:** Schizophrenia, a mental illness caused due to genetic and environmental influences, generally affects individuals during their adolescence or young adulthood. Persons suffering from schizophrenia develop delusions (holding false beliefs, like there is danger to them from others, etc.), hallucinations (e.g. hearing voices when there are none) and disorganized behavior (e.g. wandering aimlessly, talking irrelevantly, etc.). Often, they neglect their self-care, lose aim and motivation in life goals (negative symptoms) and lead compromised lives. It is one of the leading causes of disability among young people in the world.

   There is a consistent relationship between social cognition deficits and real-world functional ability in patients with schizophrenia. The more the deficits in social cognition, the poorer a person's ability to have a satisfactory day-to-day functioning like personal

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care, and in meeting family, social, occupational and academic roles. In addition, social cognition deficits may be causally linked to symptoms of schizophrenia (social behaviors). While deficits in social cognition are related to negative symptoms (Mehta et al., 2014c), such as being withdrawn, aloof and apathetic, deviant social cognition (hypermentalizing and externalizing attributional bias) is related to positive symptoms like delusions and poor self-monitoring or insight (Bentall et al., 2009). A host of other factors play a role in determining such complex functional roles in humans. However, social cognition deficits seem to play a significant role in predicting functional abilities directly, as well as indirectly, by mediating the influence of general cognitive dysfunction. A recent meta-analysis on this subject revealed that together general and social cognition influence about 25% of variance in functional outcomes in schizophrenia (Fett et al., 2011). Substantial proportion of this influence was mediated by social cognition deficits rather than neurocognitive deficits, even though both these cognitive functions are strongly related. In our own research, we have used structural equation modeling to study how social cognition could influence functional outcome. We have found that the impact of social cognition on functional outcome is in turn mediated by symptoms such as lack of insight, lack of motivation, and other symptoms of schizophrenia (Bhagyavathi et al., 2015; Mehta et al., 2014c). These observations have resulted in attempts by mental health professionals to train patients using innovative cognitive and social exercises (Eack et al., 2007; Keshavan et al., 2014; Kurtz & Richardson, 2012). However, these strategies show only modest benefits that are difficult to maintain over longer periods of time.

2. **Endophenotype status:** Endophenotypes are subtle manifestations of disorders that lie in between on the path from genes to the disease manifestation, are heritable and are present even in the absence of manifest illness in vulnerable and remitted individuals (Gottesman & Gould, 2003). There is increasing evidence to suggest that social cognition deficits are endophenotype markers for psychiatric disorders, especially schizophrenia. Deficits in all domains of social cognition have been observed even before onset of schizophrenia (Lee et al., 2015). Similar deficits have been demonstrated in even among family members of patients (Eack et al., 2010b), suggesting that these deficits have possible genetic origin. In fact, a recent meta-analysis has demonstrated deficits in facial and voice emotion recognition, and ToM in individuals who are at high-risk for developing schizophrenia (van Donkersgoed et al., 2015). In our investigations in patients who are no longer experiencing hallucinations and delusions, we found extensive deficits across all sub-domains (Mehta et al., 2013). It is important to note that these deficits are, however, not specific to schizophrenia. Nevertheless, they are present in a large magnitude in schizophrenia when compared to other disorders (Yalcin-Siedentopf et al., 2014). These observations suggest that social cognition deficits are putative endophenotypes of schizophrenia and other psychiatric disorders. They possibly have genetic influences and identifying their neural substrates may help us better understand these deficits and treat them.

MANAGEMENT OF SOCIAL COGNITION DEFICITS

From the previous sections, it is apparent that social cognition deficits are clinically relevant predominantly because of their relationship with social behaviors like delusions and negative symptoms, and real world functional outcomes. Hence, treatment strategies are required to focus on first assessing and defining the type of social cognition impairment (deficiency

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versus deviance) and then using novel biopsychosocial approaches in correcting the deviant processing or training the deficient ability.

1. **Measuring social cognition:** Assessments of social cognition have most often been performed within a research setting to understand its relationship to behaviors and neural underpinnings. There is a broad range of assessment tools to examine social cognition with varying degrees of psychometric properties. Measures of emotion processing vary broadly and include rating of emotions that are displayed in faces (facial emotion recognition) or voices (affective prosody) or rating from brief vignettes of how individuals manage, regulate, or facilitate emotion. The "gold standard" test of comprehending other persons' minds is the ability to understand that others can hold false beliefs that are different from one's own (correct) knowledge (Dennett, 1978). Emotion recognition tests explore two main abilities: the identification (labeling) of an emotion or matching of emotions shown on two faces as being the same or different. The difficulty of the tests can be varied by blending emotions, varying the intensity of emotion, degrading the images or by inverting them (Thatcher effect). Tests of social perception use video-depicted social scenarios. The participants view them and answer questions, which test their ability to observe social cues (Corrigan and Green, 1993). Attributional styles are assessed using hypothetical positive and negative events for which the participants are asked to draw causal inferences and label them as internal personal, external personal or situational (Kinderman & Bentall, 1996).

   Social cognition is under the influence of the sociocultural systems in our immediate milieu. Given the fact that culture has a moderating effect on the contextual expression of social behaviors, and development and processing of social information, it is important to have culture specific tools to examine this complex cognitive ability (Mehta et al., 2011). Within the Indian sociocultural context, ToM, attributional styles and social perception can be measured using the Social Cognition Rating Tools in Indian Setting [SOCRATIS (Mehta & Thirthalli, 2014; Mehta et al., 2011)] and facial emotion recognition can be measured using the Tool for Recognition of Emotions in Neuropsychiatric Disorders [TRENDS (Behere et al., 2008)]. Both these tools have undergone validation in the Indian sociocultural setting and have satisfactory psychometric properties.
2. **Treatment of social cognition impairments:** There is no gold-standard treatment for addressing social cognition impairments in psychiatric disorders yet. Despite initial evidence that atypical antipsychotic drugs improved social cognition (Harvey et al., 2006), subsequent studies have not shown a substantial difference with antipsychotic medications (Kucharska-Pietura & Mortimer, 2013; Sergi et al., 2007). Cognitive processes are certainly more complex than the rather simplistic mechanistic understanding that we have been able to achieve over the last several decades of cognitive neuroscience research. Nevertheless, there have been several attempts over the last two decades to develop novel treatment strategies that can specifically address social cognition impairments. This is certainly a cutting-edge research area with newer strategies being investigated by the day. The following section is a brief overview of the possible treatment avenues:
   a. **Cognitive training:** This is an intervention that uses specifically designed and behaviorally constrained cognitive or socioaffective learning events, delivered in a scalable and reproducible manner, to potentially improve neural system operations

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(Keshavan et al., 2014). The ultimate target is to correct known neural mechanisms of behavioral impairment to result in clinical change. Social cognitive training is typically delivered in several hour-long sessions every week over a period of few months to a year, in conjunction with general cognitive training and social skills training. It is combined with computer-based cognitive exercises and group-based interactive and communicative strategizing and feedback. Efforts are made to scale up the exercises, maintain motivation to learn, and provide bridging exercises to generalize the gains to real world scenarios. There is evidence that such cognitive training exercises change brain structure (Eack et al., 2010a). Facial emotion recognition and ToM abilities are the most likely to change with modest effect sizes according to a recent review (Kurtz et al., 2016). The effect on negative symptoms is also significant, but not so pronounced on delusions and similar positive symptoms.

b. **Cognitive behavioral therapy for psychosis:** This is a structured, time-bound therapy that establishes links between thoughts, emotions and behaviors, and challenges dysfunctional thoughts. It is mainly used in the treatment of resistant delusions, by correcting the deviant social cognitive processes. The administration of such therapies, as in cognitive training requires skilled manpower and is delivered over several weeks or months. The focus here is to identify key targets in a collaborative manner, and facilitate insight by means of cognitive challenging, logical questioning and behavioral experiments. Principles of formation of delusions based on social cognitive theories of a deviant ToM (hyper-mentalizing) and self-serving or externalizing attributional bias (Abu-Akel, 1999) are also used to understand and then correct the dysfunctional thought pattern. Empirical studies leading to meta-analysis, however, demonstrate minor gains on primary outcomes, such as delusions, but better gains on secondary outcomes like depression and general psychopathology (Hazell et al., 2016, Jauhar et al., 2014). It is therefore important to refine the therapy methods and perhaps examine alternative therapeutic options. This brings us to alternative strategies, which as of today are promising, but have limited evidence. These include non-invasive brain stimulation techniques like transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS), intranasal oxytocin therapy and traditional life-style practices like yoga.

c. **Intranasal oxytocin:** Emerging evidence on the neurochemical basis of social cognition comes from the study of evolutionarily conserved neuropeptides—oxytocin and vasopressin. These nonapeptides interact with dopaminergic neurons in the nucleus accumbens and ventral tegmental areas to regulate complex social information processing and responses (Young & Wang, 2004). Specifically, oxytocin plays a crucial role in social recognition, social motivation and response to social threat, while vasopressin facilitates social responsiveness and social memory (Skuse & Gallagher, 2009). A recent study demonstrated reduced plasma vasopressin levels in patients with schizophrenia compared to healthy subjects, and lack of association between plasma oxytocin levels and social cognition performance in patients and their relatives unlike the positive correlation observed in healthy subjects (Rubin et al., 2014). Another study also found lack of association between plasma oxytocin levels and social cognition

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in schizophrenia patients without delusions (Walss-Bass et al., 2013). This suggests a disruption in the ability of oxytocin to modulate social cognition in patients with schizophrenia. Moreover, intranasal oxytocin has been shown to enhance mirror neuron activity (Perry et al., 2010) in healthy subjects and social cognition ability in patients with schizophrenia (Davis et al., 2014; De Berardis et al., 2013; Gibson et al., 2014). Thus, a dysregulated oxytocin-vasopressin system (Rubin et al., 2014) may mediate social cognition deficits in schizophrenia. In this context, intranasal oxytocin has been examined either as a stand-alone treatment or in combination with social cognition or social skills training in individuals with schizophrenia and autism. While single doses do improve recognition of basic facial emotions in healthy individuals, there are either weak or no definitive improvements on ToM and emotion processing tasks in clinical populations according to recent meta-analyses (Leppanen et al., 2017; Ooi et al., 2017). However, there might be merit in combining intranasal oxytocin with conventional cognitive training sessions to leverage an additive effect of both treatments as has been examined in more recent but limited number of studies (Cacciotti-Saija et al., 2015; Gibson et al., 2014), whose results are promising but not conclusive.

d. **Non-invasive brain stimulation:** Both TMS and tDCS are noninvasive neuromodulatory techniques that utilize magnetic or weak electrical stimulation of focal brain regions to either suppress or activate the underlying cortical structures and their trans-synaptic connections (Hoy & Fitzgerald, 2010). This provides an avenue to modulate brain function by application of the magnetic or weak electrical stimulation based on our current understanding of neural network dysfunction in specific psychiatric disorders. For example, anodal tDCS (facilitating activation) of the right orbitofrontal (ventromedial) prefrontal cortex, when compared to sham stimulation resulted in better facial emotion recognition in a group of healthy individuals (Willis et al., 2015). Similarly, another study demonstrated anodal tDCS to the right dorsolateral prefrontal cortex (DLPFC) and cathodal tDCS to the left DLPFC resulting in faster facial emotion recognition in male healthy volunteers when compared to sham stimulation (Conson et al., 2015). In patients with schizophrenia, a single session of bilateral DLPFC anodal tDCS improved facial emotion processing (Rassovsky et al., 2015). Though these are promising findings, no definitive conclusions can be drawn as the evidence is limited to single session treatments with poor data on durability of effects and their generalizability to social functioning. In the only randomized controlled trial of high frequency TMS (facilitating activation) delivered to the left DLPFC, schizophrenia patients receiving true TMS (10 sessions) showed significant improvements in facial emotion recognition abilities when compared to sham TMS. However, there was no generalizability of these changes on social behavior and symptom outcomes (Wolwer et al., 2014). TMS is now also being used in clinical trials of autism. A clinical trial using deep TMS (2 weeks) delivered to the bilateral medial prefrontal cortices reported significant gains on social behaviors in adults with autism spectrum disorders when compared to sham TMS (Enticott et al., 2014). A potential site for delivering TMS or tDCS that activates the underlying cortex is the anterior mirror neuron system comprised of the ventral premotor cortex and the inferior frontal gyrus. In a randomized controlled trial

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of single session, high frequency TMS delivered to the left inferior frontal gyrus, we demonstrated greater mirror neuron system activity in the true group when compared to the sham group of healthy individuals (Mehta et al., 2015). This also replicated our earlier findings of a similar TMS protocol improving mirror neuron system activity in a healthy subject (Mehta et al., 2013). Future studies can examine if repeated sessions of TMS to these regions can improve social cognition in clinical populations. In summary, both TMS and tDCS have been used as novel therapeutic strategies to improve social cognition. The evidence from both these techniques are only preliminary and further refinements are the need of the hour.

e. **Yoga therapy:** Yoga is a multicomponent practice that involves physical postures and exercise, breath regulation techniques, deep relaxation practice and meditation/mindfulness techniques involving attention control (Khalsa, 2013; Rao et al., 2013). While earlier studies have demonstrated the utility of yoga therapy in improving social connectedness (Kinser et al., 2013), depression (Cramer et al., 2013) and negative symptoms of schizophrenia (Duraiswamy et al., 2007; Manjunath et al., 2013), recent studies have examined its effect on specific social cognitive processes. It is likely that yoga therapy improves facial emotion recognition and ToM deficits in schizophrenia. Two studies from the National Institute of Mental Health and Neuro Sciences (NIMHANS), Bengaluru, have demonstrated improvements in emotion recognition ability of patients who received yoga with effect sizes ranging from 0.6 (Behere et al., 2011) to 0.8 (Jayaram et al., 2013). Both these studies employed a randomized controlled design and compared the yoga intervention to physical exercise and/or a treatment-as-usual in a waitlist group. Effects on social cognition in both these studies were observed as early as 1–2 months of yoga therapy. These findings were recently replicated in yet another study that examined other domains of social cognition like social cue detection and ToM. In this study, yoga not only improved all domains of social cognition over a period of 20-sessions delivered over 4–6 weeks, but also improved social behavior (negative symptoms) and real world functional outcomes (Shalini, 2017). Given the effectiveness and feasibility of yoga therapy, this is the most clinically feasible and effective treatment of social cognition deficits in our opinion. Yoga therapy for social cognition deficits is now in the process of being put to regular clinical use at NIMHANS in a graded manner. This is not just for individuals with schizophrenia but also for patients with other neuropsychiatric disorders—adults with autism, social deficits following traumatic brain injury and borderline personality disorder. Possible neurobiological mechanisms through which yoga improves social cognition are discussed in a recent review article (Mehta et al., 2016). In summary, learning and performing coordinated physical postures (asanas) with a teacher facilitates imitation and the process of being imitated. This two-way process can improve social cognition and empathy through reinforcement of the premotor and parietal mirror neuron system (Radhakrishna et al., 2010). Oxytocin may play a role in mediating these processes that further lead to better social connectedness and social outcomes (Aoki & Yamasue, 2015; Jayaram et al., 2013).

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CONCLUSION

Most neuropsychiatric disorders have a common impairment in social cognition, which results in abnormal social behaviors. In the more severe disorders, these impairments (deficient or deviant) also predict real world functional outcomes. Social cognition has specific domains based on the underlying neural processes, and a broad range of deficit patterns are expressed across individual neuropsychiatric disorders. Correcting or retraining these impairments requires us to employ a biopsychosocial approach. The management begins with performing detailed assessments within a sociocultural framework using culturally valid rating instruments. It also requires a thorough understanding of the putative neural networks underlying the individual's clinical condition presenting with social cognition impairments. A thorough assessment of social behavior and social functioning is also necessary. Based on the above assessments—cognitive, behavioral, and neurobiological, a comprehensive treatment plan can be planned. Treatment options range from cognitive therapies to brain stimulation and novel pharmacotherapy using oxytocin and traditional yoga therapy. Among the various therapies, the best evidence so far is for yoga therapy and this is now gradually being introduced in routine clinical practice. Given the costs of brain stimulation and oxytocin and the manpower intensive requirements of cognitive therapies, yoga therapy also seems to be a more feasible therapeutic option. A concerted biopsychosocial understanding of how yoga therapy works includes the behavioral processes of imitating others and being imitated by others, which through the possible effects of oxytocin enhances the mirroring networks of the performer's brain resulting in better social cognition. Future investigations will build on these preliminary leads, and continue to explore within a biopsychosocial framework better therapeutic avenues for patients with severe neuropsychiatric disorders. These include refining the current treatment options, innovating to develop more advanced therapies (e.g., better pharmacotherapy) and integrating the above treatment options to leverage additive benefits in a rational and scientifically guided manner.

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Social Psychiatry

Principles & Clinical Perspectives

This book focuses on principles and clinical perspectives of social psychiatry. The social psychiatric aspects of various disorders find due space, and so do the various issues related to social psychiatry. It is enthralling to notice that several issues which are particularly relevant to the Indian cultural context (like rural mental health and farmer suicides) are covered. This book is divided into 6 sections, which are further divided into 50 chapters. Section 1 gives a brief introduction to the discipline of social psychiatry going into its historical development across the world and in India. Section 2 includes conceptual issues on the relationship between mental health and society. It begins with a chapter on the concept of social psychiatry. It then highlights the family and social context in relation to the mental health of the individual. Section 3 covers the social dimensions of psychiatric disorders. Section 4 expands upon the social interventions that are relevant to the field of social psychiatry. Social support and networking are presented first which highlights the dynamic social networks of the present times. Thereafter, couple therapies, family therapies, and group therapies are discussed. Section 5, dwells upon the social issues and mental health. The last section discusses the contemporaneous issues in relation to social psychiatry like relationship issues, effect of COVID-19, behavioral addictions, and loneliness. Social psychiatry has a wide range and multiple interfaces. This book provides information and perspective to the readers and contributes to the cause of advancement of social psychiatry.

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