Eur Arch Psychiatry Clin Neurosci (2011) 261 (Suppl 2):S145–S149
DOI 10.1007/s00406-011-0244-0

REVIEW

Deficits in social cognition: a marker for psychiatric disorders?

Birgit Derntl · Ute Habel

Received: 8 July 2011 / Accepted: 29 July 2011 / Published online: 24 August 2011

Abstract Research on social cognition focuses on several human abilities with a huge diversity in the approaches to tap the different functions. Empathy, for instance, is a rather elaborated human ability, and several recent studies point to significant impairments in patients suffering from psychiatric disorders, such as schizophrenia or autism. Neuroimaging data from these patients commonly indicate neural dysfunctions accompanying the behavioral deficits. Studying the neural correlates of social cognition is of particular importance, because deficits in these domains may explain the major dysfunctions in psychiatric disorders that prevent effective (re) integration into work and social life. It has also become clearer that social cognition deficits, similar to emotion dysfunctions, may represent trait markers and endophenotypes of the diseases. However, there are several challenges for future studies on social cognitive dysfunctions: on the one hand, the complexity of the constructs and thus the variety of definitions which make it hard to develop adequate tasks. On the other hand, results are needed that particularly address the disorder specificity of these impairments, as well as their potential as endophenotypes via analyzing people at high-risk and their relatives.

Keywords Social cognition · Schizophrenia · Autism · Emotion · fMRI

B. Derntl (✉)
Institute for Clinical, Biological and Differential Psychology, Faculty of Psychology, University of Vienna, Liebiggasse 5, 1010 Vienna, Austria
e-mail: birgit.derntl@univie.ac.at

B. Derntl · U. Habel
Department of Psychiatry, Psychotherapy, and Psychosomatics, RWTH Aachen University, Pauwelsstrasse 30, 52074 Aachen, Germany

U. Habel
JARA-BRAIN Jülich Aachen Research Alliance, Translational Brain Medicine, Juelich, Germany

Social cognition

The term "social cognition" comprises a huge number of psychological constructs that vary in complexity from more elaborated ones—such as empathy, theory of mind, and self-representation—to more elementary ones such as social perception, action-monitoring, and more generally the processing of social stimuli [cf. 1–3]. Considering its origin in the synthesis of social and cognitive psychology, scientists postulate that social cognition captures how social information is encoded, how it is stored and retrieved from memory, how social knowledge is structured and represented, and what processes are involved when individuals form judgments and make decisions [4]. Lee et al. [5] even state that social cognition has developed in order to solve social, adaptive problems and particularly refer to theory of mind and empathy. Taken together, all definitions of social cognition commonly refer to this ability as the basis for successful social interaction. Patients suffering from psychiatric disorders such as schizophrenia or autism show severe impairments in social interaction, and many studies indicate a dramatic deficit in various dimensions of social cognition.

Different abilities within the broad category of social cognition may require relatively different weighting of subcomponents of the underlying neural network [6]. The (medial) prefrontal cortex (mPFC), the amygdala, and the

inferior parietal lobe are especially implicated in such functions [1]. Most likely, disturbances in social cognition in schizophrenia and autism may represent an abnormal interaction between the frontal lobe and its functionally connected cortical and subcortical areas. In the integration of emotion and cognition within the domain of social cognition, the orbitofrontal region may play a substantial role, as it is supposed to code for more stable values and relationships [7]. However, the nature and extent of dysfunctional interactions between cognitive and emotional deficits in schizophrenia and autism remain to be elucidated.

Based on Ochsner's proposal [8], two processes have to be analyzed: bottom-up processes covering recognition of social and emotional cues and top-down processes to draw mental state inferences. Among the bottom-up processes, regions implicated in social-affective value processing and recognition are the amygdala, the striatum, and ventromedial as well as orbitofrontal cortex (first processing stream). The amygdala and insula are the bases for recognition of and response to social-emotional stimuli, and parts of the superior temporal sulcus are especially involved in the integrative processing of nonverbal cues (second processing stream).

A successful interaction in a constantly changing complex social environment requires understanding the actions of others. Only through this process, we are able to understand others' affective states and intentions and, thus, anticipate their future behavior. In analogy to imitating simple observed actions, the same mechanisms have been proposed for understanding more complex behavior. During perception of complex social actions, such as facial emotion expression, the same neural systems are activated as during its generation. Just as understanding others, behavioral adaptation occurs in many cases without conscious reasoning: internal simulation might change our own internal affective state and thereby our intention and disposition for a certain behavior. An extended "mirror neuron system" (MNS) has been proposed as the fundamental neurophysiological mechanism underlying these processes [9]. Later, a revised view on the MNS has been proposed which stresses its distributed nature in a hierarchy of predictive codes [10] that, via stratified priors, may dynamically integrate a priori knowledge, sensory cues, and internal states into behaviorally relevant outputs. Its dysfunction may hence be a major contributor to the abnormal social functioning in patients with schizophrenia and autism.

In the subsequent sections, we discuss how neuropsychological and brain imaging studies in schizophrenia and autism have shed light upon the mechanisms underlying the apparent deficits in these patients.

Schizophrenia

In schizophrenia, social interaction deficits are described as a core feature of the disorder and typically add to the deviant social behavior [11]. Schizophrenia patients show substantial deficits in several aspects of social cognition, including emotion recognition [12–14], theory of mind [ToM, 15, 16], cognitive empathy [17], and attributional style [18]. Recently, we investigated the three core components of empathy (emotion recognition, emotional perspective taking, and affective responsiveness, according to [19]) and demonstrated that schizophrenia patients show a significant deficit in every single domain, thus indicating a much broader emotional deficit as previously assumed [20].

Regarding the neural correlates, most previous neuroimaging studies addressing empathic deficits in schizophrenia patients focused on only one of the single components. For emotion recognition, studies mainly report hypoactivation of regions known to be involved in facial emotion processing, e.g., fusiform gyrus (FG), insula, amygdala [12, 21, 22], a pattern that has also been observed in subjects at risk of psychosis [23] and juvenile patients [24]. Interestingly, during the processing of neutral faces, hyperactivation in these regions in patients occurred [e.g., 12, 24–26].

Functional imaging studies investigating ToM mostly report hypoactivation of prefrontal areas in patients [e.g., 27–29], albeit with inconsistent results.

Recently, two neuroimaging studies investigated the neural correlates of empathy in schizophrenia patients by applying almost identical versions of a cartoon task measuring cognitive as well as affective empathy [30, 31]. Both studies report stronger response of the superior temporal gyrus (STG) to the cognitive empathy cartoons in controls, while results for affective empathy are inconsistent: Benedetti et al. [30] observed greater response of the right STG to affective empathy cartoons in patients, while Lee et al. [31] reported stronger response of the left insula to the affective empathy cartoons in patients. Despite their heterogeneous findings, both studies suggest dissociable neural networks for the single empathy components.

Rarely, studies report an association of neural activation with clinical parameters, thus, the impact of psychopathology on the different emotional functions is far from being elucidated and needs further research. Therefore, a more detailed understanding of the exact nature of these impairments is mandatory as a core psychopathological dimension and thus provides a target for the assessment and monitoring of patients during treatment.

Autism

Most recently, evidence has accumulated for similarities in social cognition deficits between schizophrenia patients (particularly subjects with negative symptoms) and patients with autism spectrum disorders (ASD, [32]). On the neural level, the so-called mirror neuron hypothesis of autism [33–35] has been put forward during the last years and stimulated many neuroimaging studies in ASD. These studies revealed diminished activation in several components of the human MNS during motor tasks. Moreover, ASD individuals have repeatedly been shown to be impaired in motor cognitive abilities, like imitation or the understanding of goal-directed action, that have been suggested to rely on the human MNS [36]. Most importantly, Dapretto et al. [37] showed that children with ASD exhibited less activation in the inferior frontal gyrus (IFG) compared with controls during observing and imitating emotional expressions. Notably, IFG activation in ASD children was negatively related to social impairments. An alternative explanation for impaired social interaction in ASD has been formulated by Schultz [38]. In his heuristic stage model of face processing, he proposes that typically developing infants have an inborn bias for faces which, together with learning processes during infancy, led to enhanced salience of facial stimuli. As a consequence, children spend much time processing faces, develop high experience in reading faces, and thus gain increasing perceptual skills in the face domain. These processes are thought to be predominantly mediated by the amygdala and the FG. Schultz suggests that these basal processes provide the "scaffolding" for the development of more sophisticated social skills influenced by multiple brain regions. The model posits that in ASD, early dysfunctions in face-processing areas (i.e., the amygdala and the FG, as evidenced by [39, 40]) lead to cascading deficits, including impairments in social interaction, which may also involve deficits in empathy. Schultz′s model is supported by studies in blind children who show social impairments similar to those observed in autistic subjects [41], indicating that visual input and experience are crucial for the development of social skills [38]. Interestingly, Pierce et al. [42] found that ASD children exhibited less FG activity to faces of adult strangers compared with controls, while no activation differences occurred in response to their mother's face or to other children's faces. This finding indicates that FG hypoactivation in ASD is no "all-or-nothing" phenomenon but can be modulated by certain factors, such as the familiarity of faces. Moreover, Dziobek et al. [43] observed a specific local increase in cortical thickness of the FG which was not only associated with impairments in face processing in ASD patients but also with amygdala volume. Thus, their data provide anatomical evidence of an abnormal amygdala-FG system and its behavioral relevance to face-processing deficits in ASD.

Several studies have reported that ASD subjects exhibit less activation in the neural network for ToM, including the mPFC, the temporal pole, and the STG [e.g., 44, 45]. Importantly, recent data suggest that ASD subjects activate these ToM areas in a comparable way as controls (or even to a greater extent) if an explicit instruction to attend to social cues is provided [46, 47]. Regarding the ability to share emotions, Bird et al. [48] revealed that the strength of activation in the anterior insula during empathy for pain was associated with the degree of alexithymia in autistic and control subjects. Thus, these results indicate that empathic deficits observed in ASD are strongly linked to comorbid alexithymia rather than representing a necessary feature of the social impairments in autism.

Conclusion

The aim of the present review was to give a brief overview on the existing literature on social cognitive dysfunctions in schizophrenia and autism. In schizophrenia research, there is a general consensus that social cognition is distinct from, though related to, basic neurocognition and other clinical features [3, 49]. Moreover, social cognition shows unique relationships to functional outcome, above and beyond basic cognition [2] and thus exhibits an even better target for intervention than basic cognition. And in the last years, several intervention studies impressively demonstrated that social cognitive deficits are modifiable through experimental manipulations or specific training programs [e.g., 50, 51].

Studying the neural correlates of social cognition is of particular importance, because deficits in these domains may explain the major dysfunctions in psychiatric disorders that prevent effective (re-) integration into work and social life [52].

Major aims for future research in this domain are the establishment of the association of social cognitive deficits with certain clinical symptoms and syndromes, the assessment of the stability and specificity of these dysfunctions, and the relationship to clinical outcome variables. This not only will help to better characterize certain patient groups, people at high-risk and relatives, but also provides input for psychotherapeutic treatment. And in the long run bearing this specificity analysis in mind, a particular pattern of deficits in social cognition might have the potential to be a valid marker for certain psychiatric disorders, such as schizophrenia and autism and thereby help to establish efficient interventions to tap these deficits and ameliorate these devastating diseases.

Acknowledgments B. Derntl and U. Habel were supported by the German Research Foundation (DFG IRTG 1328; Ha3202/7-1, KFO 112) and the Austrian Science Fund (FWF P23533).

Conflict of interest The authors declare that they have no conflict of interest.

This supplement was not sponsored by outside commercial interests. It was funded by the German Association for Psychiatry and Psychotherapy (DGPPN).

References

1. Brunet-Gouet E, Decety J (2006) Social brain dysfunctions in schizophrenia: a review of neuroimaging studies. Psychiatry Res 148:75–92
2. Couture SM, Penn DL, Roberts DL (2006) The functional significance of social cognition in schizophrenia: a review. Schizophr Bull 31:S44–S63
3. Green MF, Olivier B, Crawley JN et al (2005) Social cognition in schizophrenia: recommendations from the measurement and treatment research to improve cognition in schizophrenia new approaches conference. Schizophr Bull 31:882–887
4. Bless H, Fiedler K, Strack F (2003) Social cognition: how individuals construct social reality. Taylor & Francis, London
5. Lee KH, Farrow TFD, Spence SA et al (2004) Social cognition, brain networks and schizophrenia. Psychol Med 34:391–400
6. Pinkham AE, Hopfinger JB, Pelphrey KA et al (2008) Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders. Schizophr Res 99:164–175
7. Shur S, Shamay-Tsoory SG, Levkovitz Y (2008) Integration of emotional and cognitive aspects of theory of mind in schizophrenia and its relation to prefrontal neurocognitive performance. Cogn Neuropsychiatry 13:472–490
8. Ochsner KN (2008) The social-emotional processing stream: five core constructs and their translational potential for schizophrenia and beyond. Biol Psychiatry 64:48–61
9. Gallese V (2003) The roots of empathy: the shared manifold hypothesis and the neural basis of intersubjectivity. Psychopathology 36:171–180
10. Kilner JM, Friston KJ, Frith CD (2007) Predictive coding: an account of the mirror neuron system. Cogn Process 8:159–166
11. Brüne M (2005) "Theory of mind" in schizophrenia: a review of the literature. Schizophr Bull 31:21–42
12. Habel U, Chechko N, Pauly K et al (2010) Neural correlates of emotion recognition in schizophrenia. Schizophr Res 122:113–123
13. Schneider F, Gur RC, Koch K et al (2006) Impairment in the specificity of emotion processing in schizophrenia. Am J Psychiatry 163:442–447
14. Van't Wout M, van Dijke A, Aleman A et al (2007) Fearful faces in schizophrenia: the relationship between patient characteristics and facial affect recognition. J Nerv Ment Dis 195:758–764
15. Shamay-Tsoory SG, Aharon-Peretz J, Levkovitz Y (2007) The neuroanatomical basis of affective mentalizing in schizophrenia: comparison of patients with schizophrenia and patients with localized prefrontal lesions. Schizophr Res 90:274–283
16. Shamay-Tsoory SG, Shur S, Harari H et al (2007) Neurocognitive basis of impaired empathy in schizophrenia. Neuropsychology 21:431–438
17. Langdon R, Ward P (2009) Taking the perspective of the other contributes to awareness of illness in schizophrenia. Schizophr Bull 35:1003–1011
18. Moore R, Blackwood N, Corcoran R et al (2006) Misunderstanding the intentions of others: an exploratory study of the cognitive etiology of persecutory delusions in very late-onset schizophrenia-like psychosis. Am J Geriatr Psychiatry 14:410–418
19. Decety J, Jackson PL (2004) The functional architecture of human empathy. Behav Cogn Neurosci Rev 3:71–100
20. Derntl B, Finkelmeyer A, Toygar TK et al (2009) Generalized deficit in all core components of empathy in schizophrenia. Schizophr Res 108:197–206
21. Fakra E, Salgado-Pineda P, Delaveau P et al (2008) Neural bases of different cognitive strategies for facial affect processing in schizophrenia. Schizophr Res 100:191–205
22. Gur RE, Loughead J, Kohler CG et al (2007) Limbic activation associated with misidentification of fearful faces and flat affect in schizophrenia. Arch Gen Psychiatry 64:1356–1366
23. Seiferth NY, Pauly K, Habel U et al (2008) Increased neural response related to neutral faces in individuals at risk for psychosis. Neuroimage 40:289–297
24. Seiferth NY, Pauly K, Kellermann T et al (2009) Neuronal correlates of facial emotion discrimination in early onset schizophrenia. Neuropsychopharmacology 34:477–487
25. Holt DJ, Kunkel L, Weiss AP et al (2006) Increased medial temporal lobe activation during the passive viewing of emotional and neutral facial expressions in schizophrenia. Schizophr Res 82:153–162
26. Mier D, Sauer C, Lis S et al (2010) Neuronal correlates of affective theory of mind in schizophrenia out-patients: evidence for a baseline deficit. Psychol Med 40:1607–1617
27. Brunet E, Sarfati Y, Hardy-Baylé M-C et al (2003) Abnormalities of brain function during a nonverbal theory of mind task in schizophrenia. Neuropsychologia 41:1574–1582
28. Lee KH, Brown WH, Egleston PN et al (2006) A functional magnetic resonance imaging study of social cognition in schizophrenia during an acute episode and after recovery. Am J Psychiatry 163:1926–1933
29. Russell TA, Rubia K, Bullmore ET et al (2000) Exploring the social brain in schizophrenia: left prefrontal underactivation during mental state attribution. Am J Psychiatry 157:2040–2042
30. Benedetti F, Bernasconi A, Bosia M et al (2009) Functional and structural brain correlates of theory of mind and empathy deficits in schizophrenia. Schizophr Res 114:154–160
31. Lee SJ, Kang DH, Kim C-W et al (2010) Multi-level comparison of empathy in schizophrenia: an fMRI study of a cartoon task. Psychiatry Res 181:121–129
32. Couture SM, Penn DL, Losh M et al (2010) Comparison of social cognitive functioning in schizophrenia and high functioning autism: more convergence than divergence. Psychol Med 40:569–579
33. Iacoboni M, Dapretto M (2006) The mirror neuron system and the consequences of its dysfunction. Nat Rev Neurosci 7:942–951
34. Williams JH, Whiten A, Suddendorf T et al (2001) Imitation, mirror neurons and autism. Neurosci Biobehav Rev 25:287–295
35. Williams JHG (2008) Self-other relations in social development and autism: multiple roles for mirror neurons and other brain bases. Autism Res 1:73–90
36. Gallese V, Rochat M, Cossu G et al (2009) Motor cognition and its role in the phylogeny and ontogeny of action understanding. Dev Psychol 45:103–113
37. Dapretto M, Davies MS, Pfeifer JH et al (2006) Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. Nat Neurosci 9:28–30
38. Schultz RT (2005) Developmental deficits in social perception in autism: the role of the amygdala and fusiform face area. Int J Dev Neurosci 23:125–141
39. Critchley HD, Daly EM, Bullmore ET et al (2000) The functional neuroanatomy of social behaviour: changes in cerebral blood flow

when people with autistic disorder process facial expressions. Brain 123:2203–2212
40. Hadjikhani N, Joseph RM, Snyder J et al (2007) Abnormal activation of the social brain during face perception in autism. Hum Brain Mapp 28:441–449
41. Hobson RP, Bishop M (2003) The pathogenesis of autism: insights from congenital blindness. Phil Trans R Soc Lond B Biol Sci 358:335–344
42. Pierce K, Redcay E (2008) Fusiform function in children with an autism spectrum disorder is a matter of "who". Biol Psychiatry 64:552–560
43. Dziobek I, Bahnemann M, Convit A et al (2010) The role of the fusiform-amygdala system in the pathophysiology of autism. Arch Gen Psychiatry 67:397–405
44. Castelli F, Frith C, Happé F et al (2002) Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes. Brain 125:1839–1849
45. Pelphrey K, Adolphs R, Morris JP (2004) Neuroanatomical substrates of social cognition dysfunction in autism. Ment Retard Dev Disabil Res Rev 10:259–271
46. Wang AT, Lee SS, Sigman M et al (2006) Neural basis of irony comprehension in children with autism: the role of prosody and context. Brain 129:932–943
47. Wang AT, Lee SS, Sigman M et al (2007) Reading affect in the face and voice: neural correlates of interpreting communicative intent in children and adolescents with autism spectrum disorders. Arch Gen Psychiatry 64:698–708
48. Bird G, Silani G, Brindley R et al (2010) Empathic brain responses in insula are modulated by levels of alexithymia but not autism. Brain 133:1515–1525
49. Sergi MJ, Rassovsky Y, Widmark C et al (2007) Social cognition in schizophrenia: relationships with neurocognition and negative symptoms. Schizophr Res 90:316–324
50. Habel U, Koch K, Kellermann T et al (2010) Training of affect recognition in schizophrenia: neurobiological correlates. Soc Neurosci 5:92–104
51. Horan WP, Kern RS, Tripp C et al (2011) Efficacy and specificity of social cognitive skills training for outpatients with psychotic disorders. J Psychiatr Res doi:10.1016/j.jpsychires.2011.01.015
52. Brekke JS, Hoe M, Long J et al (2007) How neurocognition and social cognition influence functional change during community-based psychosocial rehabilitation for individuals with schizophrenia. Schizophr Bull 33:1247–1256