Social cognition in schizophrenia: a review of face processing

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Background: People with schizophrenia have difficulties in interpreting social information. Much social information is gathered from faces, and face processing represents a well-characterized model to study the basis of social deficits in schizophrenia.

Sources of data: A narrative review of selected literature.

Areas of agreement: Individuals with schizophrenia have impairments in recognizing basic emotions and making social judgements from facial stimuli.

Areas of controversy: The neural basis of these abnormalities is still being determined. However, initial evidence implicates dysfunction of frontal and temporal lobe brain regions. Hyper-activation of the amygdala, a brain region involved in fear, to facial stimuli may be an important underlying neural abnormality.

Growing points: The present article highlights the difficulties that people with schizophrenia have in interpreting social cues from faces.

Areas timely for developing research: Research is required to understand more about both the basis of social deficits in schizophrenia and their potential remediation.

Keywords: Emotion/identity/functional outcome/fMRI/social judgements/amygdala

Introduction

The extent of our social abilities is unique to humans, and the enjoyment of social activities and relationships lends meaning and importance to life. Recent work has investigated the cognitive processes and neural structures that are required for social interaction, abnormalities of which may underlie social dysfunction in psychiatric illness, opening up new avenues for intervention and understanding. This overview will explore the types of abnormalities seen in one key component of social interaction, face processing, in one major neuropsychiatric disorder, schizophrenia.

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Schizophrenia is a common, chronic, heterogeneous neuropsychiatric condition associated with substantial morbidity and mortality. Symptoms include the presence of abnormal mental features (‘positive symptoms’) such as hallucinations and delusions, often with social content, and the absence of normal mental features (‘negative symptoms’) including deficits in motivation, affect and social skills. The illness is in general associated with marked social and occupational dysfunction, and difficulties in interpreting the emotions and intentions of others have been hypothesized to contribute to the development of symptoms of psychosis.  

Given the social dysfunction seen in schizophrenia, studying social cognition is important because it allows abnormalities to be defined at the level of cognitive dysfunction and neural abnormalities in addition to the more distal outputs of symptoms and behaviour. Identifying particular cognitive deficits may help in diagnosis, in delineating sub-types of schizophrenia, in predicting treatment response and in prognosis. Cognitive mechanisms are also potential therapeutic targets for cognitive therapies. Understanding the neural basis offers insight into pathogenesis and hence possibly better treatment, or even prevention. All these endeavours are supported by the fact that social cognition has been shown to be functionally important in schizophrenia in determining social behaviour and outcome with regard to a range of tasks and measures, and there is considerable overlap between brain regions identified as important in social cognition in health and as abnormal in schizophrenia.

However, trying to deconstruct the complexities of social interaction into individual processes is difficult, and social cognition is a broad concept including many overlapping processes. It can be most simply defined as thinking about other people, including processes of perception, reasoning and interpretation of social stimuli, and the generation of appropriate social output. One of the most important sources of social information are faces: humans are predominantly visual animals, and we spend a great deal of time looking at and analysing faces. Face processing has thus received a great deal of investigation in health and in schizophrenia, and is comparatively well understood in comparison with newer fields. This overview will therefore focus on the social cognition of face processing as an exemplar that illustrates issues common to many other domains of social cognition.

This overview will first briefly summarize face processing in health. Secondly, the types of abnormality seen in schizophrenia are described and related to abnormalities in brain regions where known. Finally, the functional importance of face processing in schizophrenia is summarized.
Facial processing in health: an introduction

Facial processing is a complex process, contributed to by many brain regions, acting in parallel and in concert. Haxby et al. proposed the following hierarchical model: following basic analysis in the visual cortex, perception of facial features is mediated by neurones in the inferior occipital gyri. Perception of the face as having a unique identity, based on invariant facial features, is mediated by the lateral fusiform gyrus. Perception of variable facial features required for social communication, such as mouth shape or eye gaze, is mediated by the superior temporal sulcus. They suggest that this core information is then interpreted by extended neural systems, including the amygdala and prefrontal cortex, to allow perception of meanings such as emotion and attentional focus.

The variant/invariant distinction of Haxby et al. reflects the emotion/identity distinction of Bruce and Young whose influential framework argued that there are separate brain processes underlying perception of identity, emotion and familiarity. However, Haxby et al. suggest that the fundamental difference is in the type of analysis completed by brain regions, not the function of that analysis, as the two functions of identification and emotion recognition can be made more accurate by sharing information from different analysis types, for example, on characteristic expressions. More recently, on the basis of a thorough review of available evidence and the use of principal components analysis, Calder and Young have further questioned the extent of a simple dichotomy in analysis of identity versus emotion and have suggested that these differences may be more relative than absolute. However, reflecting the overall evidence that different components of face processing are at least partly separable at the neural level, work in schizophrenia has focussed on tasks distinguished by their type of functional output, with separate tests of facial identity and facial emotion, and more recently, tests of more complex social meaning. Facial processing in schizophrenia is therefore considered under these categories in the following sections.

Facial identity processing in schizophrenia

A deficit in facial identification may lead to social difficulties in itself, and can also negatively impact on the perception of other more complex information derived from faces. Facial identity recognition tasks can be divided into two main types: discriminating between or matching individuals, and recognizing a face as familiar (requiring
memory). This section considers the patterns of performance of these tasks seen in schizophrenia, and their neural basis.

**Behavioural studies**

Deficits in facial recognition have been demonstrated in many, but not all, studies of patients with schizophrenia. Impairment has been shown in both identity matching/discrimination tasks\(^7\text{–}10\) and familiarity recognition tasks.\(^11\text{,}12\) However, there is considerable heterogeneity in the results that may reflect the different types of neuropsychological tasks used. Notably, deficits in facial identity recognition are reduced in simpler tasks in which other mnemonic and attentional demands are minimized, such as matching identically posed faces\(^13\) or recognizing famous faces.\(^12\) In contrast, tasks requiring memory are particularly likely to show impairment.\(^11\text{,}12\) Therefore, there is overall evidence of a deficit in face identity recognition in schizophrenia but non-specific cognitive factors may contribute to the deficits seen in tasks with high mnemonic and attentional demands.

No clear relationship of facial recognition deficits to symptom status has emerged. Some studies have found the impairment to be worse in association with negative symptoms,\(^9\) some with symptom severity\(^10\text{,}11\) but the majority for there to be no association with symptom class\(^7\) or severity.\(^8\) However, the small sample sizes in most studies gives them little power to identify such associations and currently no strong conclusions can be drawn on the relationship with symptoms.

An issue that remains to be addressed is whether any impairment in facial recognition is due to abnormalities in more basic visual processing, leading to a bottom up contribution, and/or whether the impairment is specific to faces or occurs in the processing of all complex images. Although several studies have demonstrated abnormalities in early visual processing in schizophrenia, no studies to our knowledge have attempted to control for the impact of these abnormalities on face recognition impairment. Concerning the specificity of the deficit, one study compared the scores of those with schizophrenia for 3D manipulation with scores for the Benton test of facial recognition, a test which presents faces from multiple angles, and found that they correlated.\(^12\) The limited number of studies means that further work is needed to clarify both these issues.

**Neuroimaging studies**

The neural basis of the impairment in face identification in schizophrenia has received some attention. In health, the fusiform gyrus has been
implicated strongly as crucial to facial identity recognition by fMRI, electrophysiological and lesion studies (for review see Haxby et al.4).

In schizophrenia, the fusiform gyrus has been demonstrated to have a number of structural and functional abnormalities. Structurally, MRI studies in schizophrenia have shown the fusiform gyrus to be bilaterally reduced in volume in first episode schizophrenia14 and in chronic schizophrenia15 where volume reduction is proportional to impairment at remembering face identities.15 A meta-analysis of voxel-based whole-brain sMRI showed reduction in volume of the left fusiform gyrus in around a quarter of studies included, although only ~5% showed reduction in the right fusiform gyrus.16 Functionally, reduced activation relative to controls of the right fusiform gyrus while matching facial identity and emotion has been found by fMRI.17 Structural and functional abnormalities in the fusiform gyrus thus seem likely to underlie at least some of the impairment in face identification seen in schizophrenia.

In summary, face recognition in schizophrenia is impaired, especially in tasks with higher cognitive demands. The relationship with symptoms is unclear. The impact of basic visual processing abnormalities or the specificity of the impairment to faces has received little attention. The deficits in facial recognition in schizophrenia are likely to relate to abnormalities in the fusiform gyrus, although other brain areas may additionally be involved.

**Facial emotion processing in schizophrenia**

The ability to recognize the emotions displayed by others is crucial to appropriate social interaction. Misinterpretation of facial affect in schizophrenia has been suggested as a potential mechanism leading to symptoms such as delusions of persecution, or of social withdrawal following repeated dysfunctional interactions.1 Facial emotion recognition is also a skill that can be improved by training.18,19 For these reasons, a great deal of work has been performed to characterize the nature and extent of the deficit, its relationship with symptoms and its neural basis. However, studies seeking to address these questions have been varied in design and participants, for example, the nature of stimuli, diagnostic procedures and duration of illness. As a consequence, there is some heterogeneity in the results of different studies considered below, although an impairment in emotion recognition in schizophrenia has repeatedly been demonstrated.20 First, however, we will briefly consider the types of tasks used in investigating facial emotion recognition (Box 1).
Box 1  Types of emotion recognition task

Six emotions expressed in the face have been termed primary and found to occur across cultures—happiness, surprise and the negative emotions of sadness, fear, anger and disgust—and standardized images of these emotions have been developed and widely used in tests of facial emotion recognition, although some studies have used less well-characterized stimuli. Emotion recognition tests can explore two main abilities: the identification (labelling) of a particular emotion or the discrimination of emotions shown on two faces as being the same or different. The difficulty of the tests can be varied by blending emotions, by varying the intensity of emotion, by degrading the images or by inverting them.

These faces are examples from the collection created by Ekman et al., subsequently widely used in emotion research.

With thanks to Paul Ekman, PhD, and publishers Pearson for kind permission to reprint images.

Behavioural studies

Overall, individuals with schizophrenia show an impairment in facial affect recognition relative to non-psychiatric controls (reviewed in). This has been shown in emotion identification tasks and discrimination tasks, although a minority of studies have not found impairment, perhaps reflecting heterogeneity in the characteristics of the patients included in the sample or differences in task methodology. Despite the overall consensus as to an impairment, findings conflict over a number of issues.

Firstly, the extent to which the deficit is valence specific is unclear. Some studies have found an impairment in negative emotions in general, some find the impairment to be greatest for particular negative emotions, for example, fear, and some find no valence effect on impairment. The probable greater impairment for negative emotions may in part reflect the difficulty of identifying those emotions: happiness is the easiest emotion to identify and fear the most difficult, even for control subjects.
Secondly, the relationship of the impairment to symptoms is unclear. Some studies report deficits to be unrelated to symptoms, others that impairment is greatest in association with positive symptoms, others in association with negative symptoms and others in association with overall severity of symptoms. These results suggest that the severity of both positive and negative symptoms may influence facial emotion recognition, although the exact relationship between symptoms and facial emotion recognition remains to be resolved.

There is in addition some evidence to suggest that facial emotion recognition is a trait deficit, a marker of the disease seen throughout the illness and in attenuated form in unaffected genetic relatives, as opposed to a state deficit (one related to symptoms). In support of this, impairment in facial emotion recognition has been found in first episode schizophrenia and to a lesser degree in the unaffected biological relatives of those with schizophrenia. Longitudinal studies have shown deficits to be stable, albeit only over periods of 3 months and 12 months. However, the absence of a deficit in those ‘at-risk’ for psychosis in a prodromal state lessens the likelihood of facial emotion recognition being a trait marker. Also, cross-sectional studies show the impairment to be worse in chronic schizophrenia rather than recent onset, suggesting the deficit is not stable across the disease, but worsens with illness progression. Therefore, it remains unclear whether facial emotion recognition deficits are trait markers for schizophrenia.

The existence of abnormalities in at risk groups also has relevance for another major issue in schizophrenia research: the role of medication. The vast majority of subjects with schizophrenia will differ from controls not only in psychiatric diagnosis but also in exposure to antipsychotics, and the potential influence of these neuro-active medications on face processing is difficult to account for. One study has demonstrated an antipsychotic effect on some aspects of face processing: one-off administration of an atypical antipsychotic to healthy volunteers was associated 2 h later with a selective deficit for anger recognition in faces, but no influence on recognition of other emotions or facial identity recognition. This suggests antipsychotics may not be able to account for the full range of deficits in face processing seen in schizophrenia, a view that is also supported by the impairments seen in first episode cases and drug naïve unaffected relatives.

Finally, as with facial identity recognition, the specificity of the impairment has been questioned—is it a ‘differential deficit’ specific to facial emotion processing, or does it reflect general cognitive impairment and/or the problems in face identity processing described in the previous section? It seems most likely that there is facial processing
impairment specific to affect, as studies that control statistically for facial identity recognition impairment are still able to demonstrate a facial affect recognition deficit,\textsuperscript{8,10} and some studies have demonstrated an impairment in facial affect recognition despite intact facial identity recognition.\textsuperscript{25}

**Neuroimaging studies**

In health, a range of brain regions have been associated with emotion processing, with some specificity of particular brain regions for particular emotions. In particular, the basal ganglia and insula cortex appear to be consistently implicated in disgust recognition\textsuperscript{33} and the amygdala in fear, along with other emotions.\textsuperscript{33} Abnormalities in these regions in schizophrenia are considered below.

There is evidence of a reduction in insula volume in Schizophrenia\textsuperscript{16} and its extent of volume reduction has been found to be proportional to severity of psychotic symptoms.\textsuperscript{34} Structural abnormalities in the basal ganglia as a whole have been found in schizophrenia in two-thirds of studies identified in a large meta-analysis\textsuperscript{35} and particular regions of the basal ganglia have been found to be smaller in first episode patients and expanded in chronic illness, perhaps mediated by the action of anti-psychotics.\textsuperscript{36} Portions of both the insula and the basal ganglia have shown a lesser increase in blood flow while viewing facial expressions of disgust in those with schizophrenia relative to controls.\textsuperscript{37}

The amygdala has been shown to be structurally and functionally abnormal in schizophrenia. Structurally, the amygdala is on average reduced by about 6% in volume bilaterally in those with schizophrenia relative to controls.\textsuperscript{38} Functionally, amygdala responses to facial affect appear to be abnormal in schizophrenia, but the nature of the abnormality is subject to debate. Several studies have reported hypoactivation of the amygdala when comparing blood flow changes observed by fMRI elicited by fearful faces versus neutral faces.\textsuperscript{39} However, more recent work has suggested that there may be hyperactivation of the medial temporal lobe, especially the amygdala, to neutral faces in schizophrenia\textsuperscript{29,40,41} accounting for the apparent deficit in blood flow when comparing fearful faces with neutral faces (see Fig. 1). The equivalent response of the amygdala to neutral and fearful faces may impede their distinction and could potentially lead to either over or under-attribution of fear, depending on context. As the amygdala is a central component of the neural system mediating fear responses, the finding that it is abnormally activated in schizophrenia resonates with the enhanced fear and arousal characteristic of patients experiencing psychotic symptoms.
In summary, the results of a large number of studies using a wide range of methodologies suggest that facial affect recognition in schizophrenia is impaired. The majority of studies suggest that impairment is greatest for recognition of negative affect. Deficits in lower levels of face processing are unlikely to account for all of the impairments seen. The time-course of the impairment and its relationship with symptoms and medication use remains to be clearly established. Neuroimaging studies suggest that this impairment may relate to abnormalities in amygdala structure and function, including inappropriate activation of the amygdala to neutral faces in schizophrenia.

Fig. 1 (A) Viewing neutral faces: statistical parametric map (SPM) of brain showing areas of significantly greater blood flow in those with schizophrenia compared with controls while viewing neutral faces, contrasted with baseline. Significantly greater activation of the left amygdala to neutral faces is seen in patients than controls (marked by the cross-hairs). SPM thresholded at $P < 0.001$ uncorrected. (B) Amygdala blood flow: Bar charts show magnitude of amygdala blood flow comparing patients with controls in three contrasts: neutral versus baseline (N versus B); fear versus baseline (F versus B) and fear versus neutral (F versus N). It can be seen that those with schizophrenia show greater blood flow to the amygdala than controls when viewing neutral faces versus baseline. Error bars show standard error of the mean. With thanks for permission to reprint figure images. 

In summary, the results of a large number of studies using a wide range of methodologies suggest that facial affect recognition in schizophrenia is impaired. The majority of studies suggest that impairment is greatest for recognition of negative affect. Deficits in lower levels of face processing are unlikely to account for all of the impairments seen. The time-course of the impairment and its relationship with symptoms and medication use remains to be clearly established. Neuroimaging studies suggest that this impairment may relate to abnormalities in amygdala structure and function, including inappropriate activation of the amygdala to neutral faces in schizophrenia.
Making complex social judgements from faces in schizophrenia

Identity and emotion are not the only types of information gained from faces. Combining information on the structural and emotive elements of the face with information retrieved from memory and generated by reason allows increasingly complex judgements to be made of attributes such as attractiveness, distinctiveness, trustworthiness and approachability. These complex yet everyday tasks overlap with social cognition skills such as theory of mind (most simply defined as the ability to know another’s mental state). Inaccuracy in the perception of others’ mental states, erroneously judging their intentions, emotions or beliefs, has been theorized to underlie some of the symptoms of psychosis.1 For example, falsely perceiving someone’s expression as reflecting a threatening mental state could lead to delusions of persecution. Recent work has thus begun to investigate the performance of those with schizophrenia in making such complex social judgements.

Behavioural studies

Four recent studies have investigated the performance of patients with schizophrenia in tests of making more complex social judgement from faces. Three of these studies examined the rating of trustworthiness of faces on a Likert scale. One found patients were more likely to over-identify trustworthiness42 whereas two found little difference with controls,43,44 although a sub-group of patients experiencing paranoia were more likely to under-rate trustworthiness.43 The fourth study looked at the performance of patients with schizophrenia when making a wide range of dichotomous social judgements from faces and found that across a range of such judgements patients with schizophrenia differed significantly from controls in their rating of faces.25 This effect was particularly pronounced for judgements of approachability, intelligence and distinctiveness. Notably, these findings could not be explained by deficits in emotion recognition alone, as deficits were seen even in a group of patients with relatively intact emotion recognition, suggesting a specific deficit at the level of more complex social judgements over and above that seen for emotion recognition.25

Neuroimaging studies

Complex social judgements are mediated by the interaction of many brain regions. In health, a number of brain regions are known to be
involved in such higher order social cognition. Core areas that are particularly strongly associated with social cognition include the superior temporal sulcus, amygdala and medial prefrontal cortex although other areas are also involved to varying extents dependant on task and nature of stimuli. For example, fMRI studies in health of judgements of facial characteristics such as attractiveness show blood flow increase in regions of the brain connected with motivation/reward, the ventral striatum and orbitofrontal cortex, and viewing of untrustworthy faces additionally increases blood flow to the insula.

These core social regions are among those most consistently identified as structurally abnormal in schizophrenia. For example, the superior temporal sulcus, amygdala and prefrontal cortex have all been found to have reduced grey matter volumes in schizophrenia in the majority of MRI studies that have quantified these brain regions. The white matter tracts linking the frontal lobes with the temporal and parietal lobes have also been found to be structurally abnormal by MRI, suggesting that there may be a functional disconnection of frontal and temporal lobe regions. There is also initial evidence of abnormalities of brain function in schizophrenia during social judgement of faces, with abnormalities in blood flow while assessing trustworthiness identified in the amygdala, ventrolateral prefrontal cortex and medial orbitofrontal cortex. These changes may be clinically relevant: increased blood flow to the medial prefrontal cortex during a social cognition task is associated with clinical improvement in schizophrenia following an acute psychotic episode.

In summary, early findings suggest abnormalities in the ability to make complex social judgements about faces in schizophrenia. There is substantial overlap between regions that are structurally abnormal in schizophrenia and those involved in making social judgements from faces, suggesting that the deficits seen in patients with schizophrenia may derive from impairments in function of several components of the neural systems involved in social cognition. In particular, dysregulation of the functional interaction of frontal and temporal lobe regions during social judgement could influence many of the observed impairments in social judgement.

**Functional importance of face processing in schizophrenia**

A number of studies have addressed the functional importance of deficits in face processing in schizophrenia in relation to ‘real-world’ social outcomes and these are considered below.

Only a few studies have investigated the relationship between facial recognition and functional outcome. Two studies have found no
relationship with social behaviour on the ward\textsuperscript{13,51} whereas one study found scores were related to social mixing and personal hygiene.\textsuperscript{52} Many more studies, however, have investigated the relationship between facial emotion identification and functional outcome in a range of settings and with a range of instruments. Association between facial emotion recognition performance and functional outcome has been shown in in-patients\textsuperscript{13,51,52} and outpatients\textsuperscript{30} with a number of measures of outcome including scores for tests of social skills,\textsuperscript{52} ward behaviour rating scales completed by staff members\textsuperscript{51,52} and broader measures of community functioning/quality of life.\textsuperscript{23,30} However, associations are often found for some tests of outcome only or sub-scales within tests only and there seems to be little consistency in which particular outcomes are associated. Only one study has found no relationship between facial emotion recognition and any of the outcome measures chosen (relating to social behaviour on the ward).\textsuperscript{27}

However, as ever, the specificity of the relationship is debated. Social cognition and standard cognition measure scores often correlate and may overlap in the processes they test, and various aspects of ‘non-social’ cognition have also been shown to be related to functional outcome.\textsuperscript{53} The relationship between facial emotion recognition and other cognitive measures in the prediction of functional outcome remains open: some studies have found that facial emotion recognition does not have predictive value over and above other cognitive measures,\textsuperscript{51} whereas others studies have found that it does.\textsuperscript{23,30}

Considering interventions, there is evidence that facial emotion identification ability may predict response to treatment. A study combining measures of facial affect perception with measures of vocal affect and multi-modal affect perception found that the total score on these measures at baseline predicted improvement in functional outcome after 12 months of rehabilitation.\textsuperscript{54} Furthermore, facial emotion identification itself has been shown to be amenable to intervention.\textsuperscript{19} These improvements may be functionally important; a study of a cognitive intervention in schizophrenia showed that scores for emotion perception (in combination with other social cognitive measures) increased along with improvement in self-reported social relationships and fewer aggressive incidents compared with a control group of patients.\textsuperscript{18}

In summary, facial emotion recognition, but probably not facial identity recognition, is functionally important for some social outcomes in schizophrenia and may contribute impact beyond standard cognitive abilities. Enhancement of facial emotion identification is a potential therapeutic strategy for cognitive therapies, and improvement following such therapy may be associated with improved functional outcome.
Further work

Several areas of face processing in schizophrenia could benefit from further exploration. In particular, the degree of influence of ‘bottom up’ contributions from basic visual processing and abnormal eye movements on higher order deficits could be better characterized and controlled for. The role of medication could also be usefully clarified, particularly by focussing on individuals at risk for schizophrenia (such as prodromal states) and prospective studies assessing subjects before and after medication initiation. Work in at risk individuals could continue to be aided by neuroimaging that could potentially provide more sensitive evidence of early brain abnormalities than behavioural testing.

Conclusion

This review has discussed social cognition in schizophrenia using face processing as a well-characterized exemplar. We have reviewed evidence that schizophrenia is associated with functionally important abnormalities in face processing in the domains of emotion recognition and complex social judgements.

A range of neural abnormalities in schizophrenia in regions associated with face processing have been described. The strongest evidence is for structural abnormalities in the fusiform gyrus, superior temporal sulcus, amygdala and prefrontal cortex. New evidence has been highlighted showing that medial temporal lobe structures, especially the amygdala, may be hyperactivated by viewing faces in subjects with schizophrenia, providing a potential link to the development of psychotic symptoms such as paranoia. This hyperactivation of the amygdala may contribute to the general impairment in the ability of individuals with schizophrenia to judge the intentions of others, which has been hypothesized to contribute to the development of symptoms of psychosis.

In conclusion, studying social cognitive processes such as face processing in schizophrenia allows a greater understanding of the key cognitive mechanisms and neural abnormalities leading to the distressing symptoms and disabling outcome of the illness, potentially enabling better pathophysiological understanding, prognosis and treatment.

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