

Correspondence

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Cognitive processing in schizophrenia

I read the short report by Hall *et al* (2004) with interest. The authors reported a marked impairment in the ability of people with schizophrenia to make social judgements from facial expressions. Their findings complement and extend earlier studies by us and others (Hellewell *et al*, 1994; Edelstyn *et al*, 1996, 2003) that have reported the presence of impairments in facial recognition memory. However, these abnormalities in facial and emotion recognition do not appear to lead to obvious difficulties in day-to-day life; for example, individuals do not appear to exhibit problems with the recognition of familiar people. This apparent inconsistency between experimental findings and real-life situations raises issues about the role played by these cognitive abnormalities in schizophrenia. It is likely that these impairments are stable abnormalities rather than being transient indicators of dysfunction. This would be consistent with structural or functional abnormalities in schizophrenia, which only become evident when the processing systems are placed under high levels of stress, for example, during the prodromal or psychotic phases of a functional illness. This line of reasoning is supported by Hall *et al*'s finding that individuals with positive symptoms are unable to identify even basic facial emotions. These inherent weaknesses within the processing system may remain hidden during quiescent periods, but may be artificially exposed in the laboratory by challenging the processing system with particularly difficult tasks. Such deficits in visual processing, when combined with other factors such as changes in mental state and impaired cognitive reasoning, operate in a complex interaction to produce psychotic episodes.

In an attempt to understand the basis of their findings, Hall *et al* draw attention to the roles of the frontal and temporal

cortices as well as the amygdala. In addition to these, we believe that abnormalities in the non-intentional, automatic acquisition of knowledge about the structural relations between objects or events may contribute to impairments in social cognition. Lewicki (1988) and others have suggested that intuitive knowledge can influence how people form impressions, draw inferences and react to situations and people. Interestingly, a number of recent studies have reported the presence of implicit learning abnormalities in people with schizophrenia (e.g. procedural learning, word-stem completion, lexical and semantic priming) (Schwartz *et al*, 2003). Future research might examine how those with schizophrenia acquire implicit knowledge of regularities in social contexts and how this knowledge relates to adaptive functioning in schizophrenia.

Edelstyn, N. M. J., Riddoch, M. J., Oyebo, F., et al (1996) Visual processing in patients with Fregoli syndrome. *Cognitive Neuropsychiatry*, **1**, 103–124.

Edelstyn, N. M., Drakeford, J., Oyebo, F., et al (2003) Investigation of conscious recollection, false recognition and delusional misidentification in patients with schizophrenia. *Psychopathology*, **36**, 312–319.

Hall, J., Harris, J. M., Sprengelmeyer, G., et al (2004) Social cognition and face processing in schizophrenia. *British Journal of Psychiatry*, **185**, 169–170.

Hellewell, J. S. E., Connell, J. & Deakin, J. F.W. (1994) Affect judgement and facial recognition memory in schizophrenia. *Psychopathology*, **27**, 255–261.

Lewicki, P. (1988) *Nonconscious Social Information Processing*. New York: Academic Press.

Schwartz, B. L., Howard, D.V., Howard, J. H., et al (2003) Implicit learning of visuospatial sequences in schizophrenia. *Neuropsychology*, **17**, 517–533.

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Authors' reply: Professor Oyebo draws attention to a number of interesting issues in response to our study of social cognition

and face processing in schizophrenia. A key question raised by our study is why deficits in emotion recognition were state-dependent, being limited to individuals experiencing positive symptoms, while impairments in social cognition were stable. One possibility, as discussed by Professor Oyebo, is that those who are free of positive symptoms are able to use alternative cognitive strategies to identify basic facial emotions. This view is supported by a functional magnetic resonance imaging study in which individuals with schizophrenia, none of whom was experiencing positive symptoms, were able to identify facial emotions correctly but nevertheless showed deficits in amygdala activation when processing facial affect (Gur *et al*, 2002). These findings suggest that other brain regions compensate for the normal functions of the amygdala in facial affect processing when individuals with schizophrenia are free of positive symptoms. More difficult tests, such as our social cognition task, may prevent such compensation and thus reveal an underlying stable deficit.

Professor Oyebo also points out the apparent discrepancy between the finding that people with schizophrenia have impairments in facial recognition memory on formal testing, but are able to recognise familiar people in day-to-day life. In our study we found no deficit in the ability of those with schizophrenia to recognise the identity of novel faces presented concurrently, suggesting that the deficits seen in previous studies resulted from the mnemonic and attentional demands of the tasks used, which may be lower for familiar people.

Gur, R. E., McGrath, C., Chan, R., et al (2002) An fMRI study of facial emotion processing in patients with schizophrenia. *American Journal of Psychiatry*, **159**, 1992–1999.

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Early interventions for psychosis

The last Cochrane systematic review of early intervention for those with psychosis included cognitive-behavioural therapy (CBT), family therapy and medication, and reported no significant decrease in the development of psychosis at 12-month follow-up (Marshall & Lockwood, 2004).