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EDITED BY KHALIDA ISMAIL

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., Oyebode, F., *et al* (1996) Visual processing in patients with Fregoli syndrome. *Cognitive Neuropsychiatry*, **1**, 103–124.

Edelstyn, N. M., Drakeford, J., Oyebode, 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 Oyebode 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 Oyebode, 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 Oyebode 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 (Marshal & Lockwood, 2004).

https://doi.org/10.1192/bjp.186.3.262-a Published online by Cambridge University Press

The implications of the recent study of CBT for the prevention of psychosis (Morrison *et al*, 2004) need to be realistically interpreted with this background.

First, two people were excluded from the cognitive therapy arm after the trial had begun, which would have led to a non-significant result. This should have been acknowledged in the abstract, as an abstract has the most impact with service planners.

Second, after 6 months of cognitive therapy, there was a decrease in the development of psychosis compared with the control arm; however, there was similar distress for both groups. Cognitive therapy for psychosis has an aim of decreasing the distress of psychosis as well as the formulation of an explanatory model for that psychosis. It may be that a reframed and normalised explanatory language was taught to the individuals at high risk, and this led to the decreased identification of symptoms at 12 months and the masking of a psychotic episode. This would not ultimately lead to a decrease in distressing psychosis, but to a later identification of psychosis and a possible delay in pharmacological treatment.

The possible risk of harm or hazard was ignored, with a clear bias against the use of medication expressed by the authors in the discussion. Furthermore, the editorial comment alluded to the possibility of premature publication (Tyrer, 2004), but it is the implication of harm which needs to be explicitly stated.

Marshal, M. & Lockwood, A. (2004) Early intervention for psychosis (Cochrane review). Cochrane Library, Issue 3. Oxford: Update Software.

Morrison, A. P., French, P., Walford, L., *et al* (2004) Cognitive therapy for the prevention of psychosis in people at ultra-high risk: randomised controlled trial. *British Journal of Psychiatry*, **185**, 291–297.

Tyrer, P. (2004) From the Editor's desk. *British Journal of Psychiatry*, **185**, 360.

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Author's reply: We welcome Dr Marlowe's comments on our paper and would like to respond to the issues that he identified. The Cochrane review to which he refers examined more traditional approaches to early intervention (i.e. from first episode onwards) rather than a preventive approach in people at high risk, so we are unsure of the relevance of this. Within the manuscript we clearly acknowledge that there were several methodological limitations, including the exclusion of two participants, but we were unable to incorporate these in the abstract as he suggests because of limitations of abstract length imposed by the *Journal* (indeed, we were asked to further reduce the abstract at proof stage).

We agree that cognitive therapy for psychosis (and the prevention of psychosis) has an aim of decreasing the distress of psychotic experiences as well as the formulation of an explanatory model for a person's difficulties. We also agree that a reframed and normalised explanatory language may be developed by the service users; however, it is unlikely that this would lead to a masking of a psychotic episode. Rather, it is intended to reduce the potential for catastrophic appraisals of psychotic experiences, which are very clearly implicated in the experience of distress (Chadwick & Birchwood, 1994), and the development of normalising appraisals is at the heart of cognitive therapy for established psychosis (Morrison *et al*, 2003) and the prevention of psychosis alike (French & Morrison, 2004). Even if such a masking were to occur, the assumption that this could cause harm clearly demonstrates a bias against the use of psychosocial interventions, as it suggests that only pharmacological treatments can reduce the potential harm that may result from an untreated psychotic episode, when there is evidence that psychological treatment is also important in this respect (de Haan *et al*, 2003).

We are accused of being biased against using antipsychotic medication; we certainly are against medication in a population who are yet to develop a psychotic disorder, for the ethical reasons outlined within our paper and elsewhere (Bentall & Morrison, 2002). Finally, it is suggested that we avoid explicitly stating the possibility of harm arising from such an intervention; however, we clearly highlight the possibility of harm resulting from stigmatisation.

Bentall, R. P. & Morrison, A. P. (2002) More harm than good: the case against using antipsychotic drugs to prevent severe mental illness. *Journal of Mental Health*, **11**, 351–365.

Chadwick, P. & Birchwood, M. (1994) The omnipotence of voices: a cognitive approach to auditory hallucinations. *British Journal of Psychiatry*, **164**, 190–201.

de Haan, L., Linszen, D. H., Lenior, M. E., *et al* (2003) Duration of untreated psychosis and outcome of schizophrenia: delay in intensive psychosocial treatment versus delay in treatment with antipsychotic medication. *Schizophrenia Bulletin*, **29**, 341–348.

French, P. & Morrison, A. P. (2004) *Cognitive Therapy for People at High-Risk of Psychosis*. London: Wiley.

Morrison, A. P., Renton, J. C., Dunn, H., *et al* (2003) *Cognitive Therapy for Psychosis: A Formulation-Based Approach*. London: Psychology Press.

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Integration of psychiatric and physical health

In The Netherlands the *British Journal of Psychiatry* is distributed among Dutch psychiatrists by courtesy of the pharmaceutical industry. For the October issue of the Dutch edition I was asked to write the editorial comment, to be circulated with the *Journal* as an accompanying letter. My focus is integrated psychiatry in medicine.

Reading the October issue I was struck by the lack of an integrated perspective. Current epidemiological findings underscore how the organisation of our healthcare system is epidemiologically unfair and does not take into account the frequent co-occurence of psychiatric disturbances and physical illness (Kendell, 2001; Royal College of Physicians & Royal College of Psychiatrists, 2003). The fragmentation of care is seen as one of the major problems of current healthcare (Institute of Medicine, 2001); this applies with regard to treatment of physical disorders in mental healthcare and vice versa.

The editorial by Kingdon *et al* (2004) on the recommendations of the Council of Europe lacks such an integrated perspective. Among the recommendations the quality of physical care is not mentioned by the Council other than in relation to restraint, and this omission is not mentioned by Kingdon *et al*.

Similarly, the review by Thornicroft & Tansella (2004) opens with the fact that depression leads to more disability-adjusted life-years than cardiovascular disease and cancer, but it does not report their meaningful interrelation, for instance through compliance (DiMatteo *et al*, 2000). In the section 'Acute in-patient care' it is mentioned that patients with physical comorbidity should preferentially be seen in such facilities and not in community

https://doi.org/10.1192/bjp.186.3.262-a Published online by Cambridge University Press