

Correspondence

EDITED BY KIRIAKOS XENITIDIS and COLIN CAMPBELL

Contents ■ Explanatory models of schizophrenia ■ Restarting clozapine following leucopenia or neutropenia ■ Risk factors for coronary heart disease in people with severe mental illness

Explanatory models of schizophrenia

Das *et al* (2006) assessed the efficacy of interventions to change explanatory models of schizophrenia among relatives of people with schizophrenia in India. They claim that their educational intervention presented the biomedical model without dismissing non-biomedical models and that indigenous beliefs were not challenged. Depending on the way in which the intervention was delivered, one can argue that presenting biomedical models is in itself directly challenging to indigenous beliefs. Although the authors found that their educational programme significantly reduced the number of non-biomedical beliefs, this does not say anything about the quality or depth of these beliefs. Moreover, the description of participants' beliefs as 'persistent' and 'resistant' suggests that the authors consider holding alternative explanatory beliefs to be problematic. They further justified their aim by suggesting that holding indigenous beliefs contributes to a poor outcome, which they defined as not recognising a biomedical explanation of schizophrenia and not adhering to medication. This is circular logic, using a very limited construction of outcome.

Despite citing a paper by Angermeyer's German research team, Das *et al* miss their important and consistent finding that biomedical causal beliefs are significantly related to negative attitudes (e.g. Angermeyer & Matschinger, 2003). Such negative consequences of holding biomedical causal beliefs have been found in numerous countries among the public, relatives and patients with severe mental illness (Read & Haslam, 2004; Read *et al*, 2006).

How does exporting the beliefs of Western experts to low- and middle-income countries fit with the consistent finding that these countries have much better outcomes for 'schizophrenia' than Western countries (Harrison *et al*, 2001)?

Finally, Das *et al* recommend that the advantages of medication should be discussed without dismissing or challenging indigenous explanatory models. We cannot assume that the challenge is not inherent in the underlying principles of the belief systems themselves. Investigating ways in which biomedical explanations can be discussed in conjunction with cultural beliefs is a constant challenge that will not be helped by reducing the prevalence of one set of beliefs.

Angermeyer, M. & Matschinger, H. (2003) Public beliefs about schizophrenia and depression: similarities and differences. *Social Psychiatry and Psychiatric Epidemiology*, **38**, 526–534.

Das, S., Saravanan, B., Karunakaran, K. P., et al (2006) Effect of a structured educational intervention on explanatory models of relatives of patients with schizophrenia. Randomised controlled trial. *British Journal of Psychiatry*, **188**, 286–287.

Harrison, G., Hopper, K., Craig, T., et al (2001) Recovery from psychotic illness: a 15- and 25-year international follow-up study. *British Journal of Psychiatry*, **178**, 506–517.

Read, J. & Haslam, N. (2004) Public opinion: bad things happen and can drive you crazy. In *Models of Madness* (eds J. Read, R. Bentall & L. Moshier), pp. 133–146. Hove: Routledge.

Read, J., Haslam, N., Sayce, L., et al (2006) Reducing negative attitudes towards people diagnosed 'schizophrenic': evaluating the 'mental illness is an illness like any other' approach. *Acta Psychiatrica Scandinavica* (in press).

M. Taitimu, J. Read Private Bag 92019, Department of Psychology, University of Auckland, New Zealand. Email: m.taitimu@auckland.ac.nz
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Authors' reply: We agree with Taitimu & Read that discussing biomedical beliefs in conjunction with indigenous beliefs in the clinical setting is challenging. However, patients, their relatives and the general public seem to simultaneously hold multiple and contradictory beliefs related to

mental illness and its treatment (Joel *et al*, 2003). Biomedical explanations (e.g. disease, abnormality, infection, degeneration, etc.) often coexist with indigenous beliefs (e.g. supernatural causation, sin and punishment, karma, etc.) in many cultures (Saravanan *et al*, 2004). It is common for people in India to simultaneously seek help and treatment from practitioners of modern medicine and from traditional and faith healers (Jacob, 1999). This may not lead to conflict providing that each practitioner does not claim exclusivity. We have hypothesised that such multiple models may be advantageous, 'buffering' notions of loss and stigma and preventing social disintegration (Saravanan *et al*, 2004).

We agree that the acceptance of mental illness labels may increase perceived stigma. Nevertheless, holding alternative beliefs of causality also has costs. This is particularly true for people with chronic psychosis for whom antipsychotic medication has a powerful effect on outcome. Studies which have reported a better outcome for people with schizophrenia from low- and middle-income countries included many patients on psychotropic medication. The complete failure to subscribe to a disease model often results in a delay in seeking treatment and a poorer outcome.

The acknowledgement that individual health systems do not comprehensively address every issue for all mental disorders is useful in patient care (Jacob, 1999). It provides for alternatives in clinical situations, especially for psychiatrists practising in non-Western cultures, and allows the use of regional therapies, yoga and meditation, and respects folk beliefs and religions. Many experienced psychiatrists working in non-Western cultures employ cultural constructs and local treatments in their practice. Although psychological constructs are easily incorporated, traditional physical therapies are seldom used owing to the poor understanding of their active principles. Only a minority of mental health professionals in low- and middle-income countries rigidly function within Western frameworks. The majority acknowledge the ethnocentricity of psychiatry and its treatment techniques and the equally effective traditional alternatives. An eclectic approach and a liberal framework will enable psychiatrists to incorporate local cultural beliefs and traditional psychological treatments in therapy, thus increasing the therapeutic armamentarium.

Jacob, K. S. (1999) Mental disorders across cultures: the common issues. *International Review of Psychiatry*, **11**, 111–115.

Joel, D., Sathyaseelan, M., Jayakaran, R., et al (2003) Explanatory models of psychosis among community health workers in South India. *Acta Psychiatrica Scandinavica*, **108**, 66–69.

Saravanan, B., Jacob, K. S., Prince, M., et al (2004) Culture and insight revisited. *British Journal of Psychiatry*, **184**, 107–109.

B. Saravanan Institute of Psychiatry, London SE5 8AF, UK.

K. S. Jacob Department of Psychiatry, Christian Medical College, Vellore 632002, India.
Email: ksjacob@cmcvellore.ac.in

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Restarting clozapine following leucopenia or neutropenia

Dunk *et al* (2006) report rechallenge with clozapine of people with either treatment-resistant or treatment-intolerant schizophrenia. A proportion of these may lack insight and capacity and may therefore be detained under the Mental Health Act 1983 but the authors do not indicate the proportion of patients in this group. If a person has been compulsorily detained, the treating clinician may require a second opinion from the Mental Health Act Commission. We are interested in whether Dunk *et al* have any data on this, as in the *British National Formulary* clozapine is contraindicated in those who have previously developed dyscrasia. The Mental Health Act Commission may not provide a second opinion for drugs that are contraindicated.

Dunk *et al* report a possible alternative explanation for dyscrasia during first exposure to clozapine in 25 patients. There was no alternative explanation in the remaining 28 patients. An obvious question that arises is whether a patient is more or less likely to develop dyscrasia on rechallenge if they have a history of an alternative explanation. This would be a very useful predictor and would be helpful when discussing the options with the patient prior to rechallenge.

British Medical Association & Royal Pharmaceutical Society (2005) *British National Formulary*. BMJ Publishing Group & Pharmaceutical Press.

Dunk, L. R., Annan, L. J. & Andrews, C. D. (2006) Rechallenge with clozapine following leucopenia or

neutropenia during previous therapy. *British Journal of Psychiatry*, **188**, 255–263.

P. Soma, P. C. Naik Lyndon Clinic, Birmingham and Solihull Mental Health NHS Trust, Hobs Meadow, Solihull, West Midlands B92 8PW, UK.
Email: prakash.naik@bsmht.nhs.uk

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Authors' reply: The Clozaril Patient Monitoring Service (CPMS) does not record which patients are being treated under the Mental Health Act 1983 and we are therefore unable to say what proportion of patients in our cohort were compulsorily detained. We are not aware of any studies regarding second opinions from the Mental Health Act Commission in patients undergoing rechallenge with clozapine but would be interested to hear of any.

We have re-examined our data to determine whether patients were more or less likely to develop dyscrasia on rechallenge if they had a history of an alternative explanation for the first episode of dyscrasia. Out of 53 patients in the cohort, 25 had an alternative explanation for the first episode and 6 of these (24%) developed a second episode on rechallenge. Out of the 28 patients with no alternative explanation for the first episode of dyscrasia, 14 (50%) experienced dyscrasia on rechallenge. The difference was not significant ($P=0.05914$). The relative risk of 2.08 indicated that patients with no alternative explanation may be twice as likely to have a second episode of dyscrasia on rechallenge as those with an alternative explanation, but the 95% confidence interval was 0.98–6.2. We must stress that alternative explanations for dyscrasia may not always be reported to the CPMS, therefore these figures may not represent the true picture and this aspect of our work should be interpreted with caution.

Declaration of interest

L.D. has undertaken consultancy for Novartis UK and Novartis Australia and received a fee from Novartis Australia for the preparation of this paper; she was formerly employed by Novartis UK. L.A. and C.A. are employed by Novartis UK.

L. R. Dunk Department of Histopathology, Leicester Royal Infirmary, Leicester LE1 5WW, UK.
Email: louisa.dunk@btinternet.com

L. Annan, C. Andrews Novartis Pharmaceuticals UK Ltd, Camberley, UK
doi: 10.1192/bjp.189.3.285a

Risk factors for coronary heart disease in people with severe mental illness

Osborn *et al* (2006) compared risk factors for coronary heart disease (CHD) in people with and without severe mental illness (SMI) in primary care.

A number of points in the results, discussion and conclusions seem unjustified and are potentially misleading. For example, the statement that patients with SMI had a significantly raised CHD risk score is based upon the unadjusted risk. After adjustment for age and gender the odds ratio dropped below the level of statistical significance and fell further to a non-significant value of 1.3 (95% CI 0.7–2.7) after considering employment status. The authors' claim that 'we have demonstrated that SMI itself can incur CHD risk, *over and above that associated* with the socio-economic deprivation experienced by these patients' is not justified.

This claim is repeated in the abstract: 'excess risk factors for CHD are not wholly accounted for by medication or socio-economic deprivation'. This statement seems either unproven or reducible to the fact that smoking is more common among people with SMI. Such a conclusion is scarcely novel and clearly does not explain the excess mortality observed in patients with SMI (Joukamaa *et al*, 2006). The fact that diabetes is both more common among people with SMI and much less explicable in terms of their deprivation or demographics receives relatively little comment, despite having particular relevance for their healthcare needs.

Joukamaa, M., Heliövaara, M., Knekt, P., et al (2006) Schizophrenia, neuroleptic medication and mortality. *British Journal of Psychiatry*, **188**, 122–127.

Osborn, D. P. J., Nazareth, I. & King, M. B. (2006) Risk for coronary heart disease in people with severe mental illness: cross-sectional comparative study in primary care. *British Journal of Psychiatry*, **188**, 271–277.

C. Gilleard Department of Psychology and Psychotherapies, Springfield University Hospital, Tooting, London SW17 7DJ, UK. Email: Chris.Gilleard@swlstg-tr.nhs.uk
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