# Correspondence

EDITED BY MATTHEW HOTOPF



## Psychiatrists can cause stigma too

The Royal College of Psychiatrists' campaign to reduce the stigma of mental illness needs to examine the role that we play in maintaining stigma as well as reducing it. The negative attitudes of members of the public (Crisp *et al*, 2000) towards people with mental illness were mirrored by some psychiatrists (Farrell & Lewis, 1990). The latter authors found that psychiatrists held significantly more negative attitudes towards patients with a prior history of alcohol dependence. This included the view that they would not like these patients in their clinics. Similar findings apply to other groups of patients. Lennox & Chaplin (1996) surveyed the attitudes of Australian consultant psychiatrists. They found that 39% agreed with the statement 'personally I would prefer not to treat patients with learning disability and mental illness'.

The very nature of our job can be powerfully stigmatising in a way that cannot be underestimated. While engaging in debate with the public via the media and other means to inform and change attitudes, performing our clinical duties can have exactly the opposite effect. A Mental Health Act assessment at a patient's residence can be a cause of tremendous stigma to the patient and the family. This is especially so because of the highly visible involvement of the ambulance and police services whose help is often essential. It is against such almost routine community experiences that a broader national campaign has to compete.

Another very real source of stigma may be the side-effects of the medications that we prescribe. People with schizophrenia may not appear any different to the general public. However, side-effects such as drooling and tardive dyskinesia immediately point out an individual as being socially undesirable. Obesity, often a result of antipsychotic treatment, has been described as being seen as unattractive and unlikeable and has been linked with impaired employment and education opportunities (Crandall, 1994).

Psychiatrists have a clear duty to reduce stigma at the individual level. We must be prepared to identify and challenge our own prejudices and attempt to modify our clinical practice. Consideration also needs to be given to how we can carry out Mental Health Acts assessments, potentially the most stigmatising event that any family with a member with mental illness will suffer.

**Crandall, C. S. (1994)** Prejudice against fat people: ideology and self-interest. *Journal of Personality and Social Psychology*, **66**, 882–894.

**Crisp, A. H., Gelder, M. G., Rix, S., *et al* (2000)** Stigmatisation of people with mental illnesses. *British Journal of Psychiatry*, **177**, 4–7.

**Farrell, M. & Lewis, G. (1990)** Discrimination on the grounds of diagnosis. *British Journal of Addiction*, **85**, 883–890.

**Lennox, N. & Chaplin, R. (1996)** The psychiatric care of people with intellectual disability: the perceptions of consultant psychiatrists in Victoria. *Australian and New Zealand Journal of Psychiatry*, **30**, 774–780.

**R. Chaplin** South West London and St George's Mental Health Trust, 61 Glenburnie Road, London SW17 0JB

## Stigmatising pharmaceutical advertisements

The general public holds stigmatising attitudes toward those with mental disorder, with schizophrenia being rated as highly associated with dangerousness and unpredictability (Crisp *et al*, 2000). The authors mention that health professionals may share some of these views. After reading their article, I was struck by a number of pharmaceutical advertisements elsewhere in the same issue of the *Journal*, that appeared to perpetuate a negative image of schizophrenia. My curiosity thus stimulated, I performed a cursory lunchbreak study examining the portrayal of people with mental disorder in pharmaceutical advertising in three recent issues of international psychiatric journals (Table 1). It was notable that all the advertising for antidepressants had positive imagery. Indeed this was also largely true for the 'other' category, with only one negatively rated advertisement.

By contrast, three out of five advertisements for antipsychotic medications in this *Journal* were negative. One was particularly striking, a fearful young man peering through a door, his house covered in foil. The copy included the following: "His parents have to withstand torrents of verbal abuse. And Constant threats of violence". This small sample also suggests that there may be international variations in advertising in the field; what underlies this is unclear. It is intriguing, however, that the British advertising mirrors the attitudes of surveyed householders.

**Table 1** Pharmaceutical advertisements in three psychiatry journals

| | *British Journal of Psychiatry*, July 2000 | *American Journal of Psychiatry*, June 2000 | *Australian and New Zealand Journal of Psychiatry*, June 2000 |
|---|---|---|---|
| Antidepressants | | | |
| No. advertisements | 2 | 5 | 5 |
| No. rated as negative | 0 | 0 | 0 |
| Example of imagery | | | Smiling woman |
| Antipsychotics | | | |
| No. advertisements | 5 | 3 | 2 |
| No. rated as negative | 3 | 0 | 0 |
| Example of imagery | Wan young woman, dishevelled hair | Family photos of happy family | Attractive young woman putting lipstick on |
| Other | | | |
| No. advertisements | 1 | 5 | 0 |
| No. rated as negative | 0 | 1 | |
| Example of imagery | | Smiling children (advert for stimulants) | |



https://doi.org/10.1192/bjp.177.5.467-a Published online by Cambridge University Press

How can we expect the general public to have a rational and informed approach to people with schizophrenia when learned journals accept advertisements that promote a product through negative stereotyping? Perhaps our willingness to allow this to happen is in accord with work in the field which suggests that health professionals may have even more negative attitudes to mental disorder than the general public (Jorm *et al*, 1999). A public campaign to combat stigma is undoubtedly important, but perhaps we should be prepared to examine our own beliefs about serious mental illness as a prelude to changing attitudes in society at large.

**Crisp, A. H., Gelder, M. G., Rix, S., *et al* (2000)** Stigmatisation of people with mental illnesses. *British Journal of Psychiatry*, **177**, 4–7.

**Jorm, A. F., Korten, A. E., Jacomb, P. A., *et al* (1999)** Attitudes towards people with a mental disorder: a survey of the Australian public and health professionals. *Australian and New Zealand Journal of Psychiatry*, **33**, 77–83.

**D. McKay** Department of Psychological Medicine, The University of Sydney, Block 4 Level 5, Royal North Shore Hospital, St Leonards, NSW 2065, Australia

## Serum cholesterol and parasuicide

Garland *et al* (2000) reignited the various controversies on the role of cholesterol in psychiatric disorders. The methodology used was similar to those in previous studies (Asberg *et al*, 1976) which did not control for the substances used in parasuicide. This may affect the levels of the chemical or metabolites being researched. Garland *et al* (2000) did not mention the methods used in those parasuicides and whether they would have affected serum cholesterol.

Engelberg (1992) and Block & Edwards (1987) held contrasting views on the relationship between cholesterol and serotonin uptake. The work by Heron *et al* (1980) used to support the hyposerotonergic function caused by low cholesterol appeared flawed. The serotonin site labelled by Heron *et al* (1980) is not the uptake site (Hawton *et al*, 1993), and therefore changes in brain serotonin content cannot be explained on the basis of their data. Furthermore, the serotonin stored within brain cells is not accumulated from blood but synthesised *in situ* from L-tryptophan.

Plasma cholesterol is in a dynamic state, entering the blood complexed with lipoproteins that keep it in solution and leaving the blood as tissues take up cholesterol. High-density lipoprotein (HDL)-cholesterol that transports circulating cholesterol to the liver for clearance plays a crucial role. Excess HDL can result from excess alcohol (Parkes *et al*, 1989). This increases the amount of cholesterol transported peripherally, causing low serum cholesterol. Alcohol, drugs and poisons are usually involved in parasuicides (Asberg *et al*, 1976) and low cholesterol level may therefore be due to ethanol misuse or poisoning. It is unlikely that cholesterol would provide the needed answers to parasuicide. It would only reduce this complex human behaviour to a 'matter to mind' paradigm.

**Asberg, M., Traskman, L. & Thoren, P. (1976)** 5-HIAA in the cerebrospinal fluid: a biochemical suicide predictor? *Archives of General Psychiatry*, **33**, 1193–1197.

**Block, E. R. & Edwards, D. (1987)** Effect of plasma membrane fluidity on serotonin transport by endothelial cells. *American Journal of Physiology*, **253**, 672–678.

**Engelberg, H. (1992)** Low serum cholesterol and suicide. *Lancet*, **339**, 727–729.

**Garland, M., Hickey, D., Corvin, A., *et al* (2000)** Total serum cholesterol in relation to psychological correlates in parasuicide. *British Journal of Psychiatry*, **177**, 77–83.

**Hawton, K., Cowen, P., Owens, D., *et al* (1993)** Low serum cholesterol and suicide. *British Journal of Psychiatry*, **162**, 818–825.

**Heron, D. S., Shinitzky, M., Herschkowitz, M., *et al* (1980)** Lipid fluidity markedly modulates the binding of serotonin to mouse brain membranes. *Proceedings of the National Academy of Sciences of the USA*, **77**, 7463–7467.

**Parkes, J. G., Hussain, R. A. & Goldberg, D. M. (1989)** Effect of alcohol on lipoprotein metabolism. I. High density lipoprotein binding. *Clinical Physiology and Biochemistry*, **7**, 269–277.

**O. J. Famoroti** Lishman Brain Injury Unit, The Maudsley Hospital, Denmark Hill, London SE5 8AZ

## Transcranial magnetic stimulation: asymmetrical excitability and depression

Maeda *et al* (2000) have succeeded in demonstrating the interhemispheric asymmetry of motor cortical excitability in major depression, using transcranial magnetic stimulation (TMS). This is an important finding that raises questions not only about the pathophysiology of major depression, but also about the state or trait nature of the results.

In discussing possible explanations for this functional asymmetry the authors consider the activity of inhibitory interneurons between cortical output cells, as proposed by Wasserman *et al* (1996), but it is not clear whether this mechanism is thought to act within the hemisphere being stimulated. The role of transcallosal inhibitory mechanisms has been demonstrated in schizophrenia (Davey *et al*, 1997; Boroojerdi *et al*, 1999) and is likely to be relevant to understanding asymmetrical motor thresholds in depression. In support of this view, Menkes *et al* (1999) hypothesised that depression is associated with decreased left hemisphere excitability with respect to the right hemisphere. They successfully showed that inhibitory low-frequency repetitive TMS applied to the right frontal lobe produced a significant antidepressant effect, in contrast to exciting the left frontal lobe by means of fast-frequency repetitive TMS, the antidepressant effects of which have been known for some years.

Furthermore, Maeda *et al* report mean motor thresholds in the depression group of 41.13% for the left hemisphere and 37.63% for the right hemisphere, and in the healthy group of 48.29% for the left hemisphere and 52.7% for the right hemisphere. This gives a mean motor threshold of 39.38% for the depression group and 50.50% for the controls, which suggests important differences in both absolute threshold and laterality between the groups. Any changes to either of these parameters in subjects recovered from depression, and possibly in their first-degree relatives, not only promises new insights into the pathophysiology of depression, but also may provide clues about the most elusive object, a biological marker for depression.

**Boroojerdi, B., Töpper, R., Foltys, H., *et al* (1999)** Transcallosal inhibition and motor conduction studies in patients with schizophrenia using transcranial magnetic stimulation. *British Journal of Psychiatry*, **175**, 375–379.

**Davey, N. J., Puri, B. K., Lewis, H. S., *et al* (1997)** The effects of antipsychotic medication on electromyographic responses to transcranial magnetic stimulation of the motor cortex in schizophrenia. *Journal of Neurology, Neurosurgery and Psychiatry*, **63**, 468–473.

**Maeda, F., Keenan, J. P., Pascual-Leone, A. (2000)** Interhemispheric asymmetry of motor cortical excitability in major depression as measured by transcrannial magnetic stimulation. *British Journal of Psychiatry*, **177**, 169–173.

**Menkes, D. L., Bodnar, P., Ballesteros, R. A., *et al* (1999)** Right frontal lobe slow frequency repetitive transcranial magnetic stimulation (SF r–TMS) is an effective treatment for depression: a case–control pilot study of safety and efficacy. *Journal of Neurology, Neurosurgery and Psychiatry*, **67**, 113–115.

**Wasserman, E. M., Samii, A., Mercuri, B., *et al* (1996)** Responses to paired transcranial magnetic stimuli in resting, active and activated muscles. *Experimental Brain Research*, **109**, 158–163.

**B. J. Moore** The University Department of Psychiatry, Royal Liverpool University Hospital, Liverpool L69 3GA



https://doi.org/10.1192/bjp.177.5.467-a Published online by Cambridge University Press