Correspondence

EDITED BY MATTHEW HOTOPF

Contents ■ Psychiatrists can cause stigma too ■ Stigmatising pharmaceutical advertisements ■ Serum cholesterol and parasuicide ■ Transcranial magnetic stimulation: asymmetrical excitability and depression ■ Child abuse and the clinical course of drug misuse ■ Apolipoprotein E, Alzheimer’s disease and Down’s syndrome ■ Chronic fatigue syndrome and depression

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.


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

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antidepressants</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. advertisements</td>
<td>2</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>No. rated as negative</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Example of imagery</td>
<td></td>
<td>Smiling woman</td>
<td></td>
</tr>
<tr>
<td><strong>Antipsychotics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. advertisements</td>
<td>5</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>No. rated as negative</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Example of imagery</td>
<td>Wan young woman, dishevelled hair</td>
<td>Family photos of happy family</td>
<td>Attractive young woman putting lipstick on</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. advertisements</td>
<td>1</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>No. rated as negative</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Example of imagery</td>
<td>Smiling children</td>
<td>(advert for stimulants)</td>
<td></td>
</tr>
</tbody>
</table>
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.


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.


O. J. Famoroti Liahman 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 anti-depressant 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 52.77% 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.


B. J. Moore The University Department of Psychiatry, Royal Liverpool University Hospital, Liverpool L69 3GA
Child abuse and the clinical course of drug misuse

Charnaud & Griffiths (2000) in response to the finding of increased psychiatric symptoms in female drug users by Marsden et al (2000) postulate that this finding may be a sequela of earlier child abuse. It is interesting to note the high incidence of childhood sexual abuse found in their study population based in Cornwall. In a Dublin sample, the level of sexual abuse for both males and females was considerably lower (21%). However, the effects of abuse appear to have a significant influence in subsequent clinical progression of substance misuse. Those patients with a history of sexual abuse in the past had a significantly younger mean age of first opiate use (16.7 years v. 19.1 years for those without a history of sexual abuse) (Browne et al, 1998). The duration of drug misuse was also considerably longer (mean 10.8 v. 8.4 years).

We would support the suggestion of Charnaud & Griffiths (2000) that the evaluation of previous history of sexual abuse can predict the best plan of treatment for these patients. We would suggest that the long-term clinical progression of sexually abused drug misusers is that of more rapid progression to intravenous drug misuse with all the prognostic features that this implies.


R. Browne, J. O’Connor Drug Treatment Centre, Trinity Court, 30/31 Pearse St, Dublin 2, Republic of Ireland

Apolipoprotein E, Alzheimer’s disease and Down’s syndrome

We read with interest the article by Deb et al (2000) apparently demonstrating findings contrary to our own (Prasher et al, 1997). Overall, we agree with the findings by Deb et al, although clarification on several important points is required.

The principle reason why we did not find a statistically significant association (at the 5% significance level) between apolipo-

protein E (ApoE) ε4 and Alzheimer’s disease in adults with Down’s syndrome was because at that time there was a much smaller sample size of adults with Down’s syndrome and dementia available for meta-analysis (102 subjects previously included compared to 158 in Deb et al’s report). The three additional reports included in Deb et al’s meta-analysis are of significantly larger samples. However, even with this greater number of subjects available for meta-analysis the power remains at 76%. Given the proportions of ε4 in the groups with and without dementia in the Deb et al paper, for a power of 90%, a minimum of 224 adults with Down’s syndrome and dementia are required to demonstrate statistical significance at the 5% level. Furthermore, the ε4 allele frequency in the different studies varies from 5.9% to 33.4% in subjects with dementia (Deb et al, 2000) and therefore future studies are still required if an association between ApoE ε4 genotype and Alzheimer’s disease in adults with Down’s syndrome is to be established.

Deb et al are incorrect to exclude the study by Winsiewski et al (1995) because “they diagnosed Alzheimer’s disease on the basis of neuropathological findings alone”. Winsiewski et al (1995) made a diagnosis of dementia (not Alzheimer’s disease) by a clinical assessment alone “as judged by the physician following the patient”. However, the inclusion of this study in the present meta-analysis makes little difference to the findings by Deb et al (2000) as only one person with an ε4 allele was present.

The increase in risk of developing dementia in adults with Down’s syndrome (odds ratio 2.02) appears to be less than that in populations with no learning disability where it can be increased by as much as 30 times for people with two copies of the ε4 allele (Swartz et al, 1999). From the allele frequency given by Deb et al (2000) the diagnostic accuracy of ApoE ε4 for adults with Down’s syndrome and dementia is of some clinical value. The sensitivity is 18% (95% CI 13.5–22%) and specificity 90% (95% CI 88–92%). The absence of an ε4 allele strongly suggests the absence of Alzheimer’s disease. ApoE genotyping in the Down’s syndrome population may possibly be used to screen for dementia.

We conclude, as previously (Prasher et al, 1997), that the presence of an ε4 allele is neither sufficient nor necessary to cause Alzheimer’s disease but ApoE ε4 genotype does have a role to play in the presentation of Alzheimer’s disease in adults with Down’s syndrome. The effect is, however, ‘overwhelmed’ by the excessive amyloidosis due to the triplication of the amyloid precursor gene.


V. P. Prasher Department of Psychiatry, University of Birmingham, Queen Elizabeth Psychiatric Hospital, Mindelshon Way, Birmingham B1 2QZ

M. S. Haque Research & Development Unit, South Birmingham Mental Health Trust, Birmingham

Authors’ reply: We thank Drs Prasher & Haque for their interest in our paper and are pleased that they agree with our conclusions. It is quite obvious that the difference in findings in the meta-analysis between our study and Prasher et al’s study was due to the inclusion of data in our study that were not available at the time of Prasher et al’s study. According to our calculation, our meta-analysis has 92% power (95% CI 88–96%) at the 5% level. However, traditional power calculation is not applicable in this case because instead of simply adding allele frequencies among all studies, we have used the computerised version of the Woolf (1995) method of meta-analysis that takes account of each study individually. Also, because of the varied nature of studies included in the meta-analysis we did not feel it appropriate to calculate specificity and sensitivity in the traditional way.

It was not stated in Prasher et al’s (1997) paper which 31 patients (15 with and 16 without dementia) out of 40 patients with Down’s syndrome, presented in Winsiewski et al’s (1995) study, were included in their meta-analysis. The age of death of patients reported in Winsiewski et al’s study ranged widely between 15 and 69 years. They mentioned at the bottom of their table that “The presence of dementia is defined as a deterioration of competence, as judged by the physician following the patient”. No detail...
about diagnosis was mentioned in the text and no patient over age 30 had an e4 allele. For these reasons we chose not to include this study in our meta-analysis. However, as Prasher & Haque point out inclusion of this study would have made little difference to our findings.

Whereas Prasher & Haque rightly suggest that further research is needed to clarify the role of ApoE e4 in Alzheimer’s disease in people with Down’s syndrome, we were surprised to see that they have recommended ApoE genotyping as a possible screening test for dementia in this population. This will be totally inappropriate at this stage considering the uncertain relationship between Alzheimer’s neuropathology and ApoE genotype in people with Down’s syndrome, as we mentioned in the last paragraph of the Discussion in our paper.

We agree with Prasher & Haque that the presence of e4 allele is neither necessary nor sufficient for the development of Alzheimer’s disease.


S. Deb, J. Williams & M. J. Owen Division of Psychological Medicine, University of Wales College of Medicine, Heath Park, Cardiff CF14 4XN

Chronic fatigue syndrome and depression

I found MacHale et al’s (2000) discussion of their results confusing. According to the abstract and methods, they screened their patients with chronic fatigue syndrome (CFS) to exclude those with depression. Then they examined this group further using a standardised psychiatric interview (Schedule for Affective Disorders and Schizophrenia), in order to “exclude subjects with current psychiatric illness, with a particular emphasis on depression”. The data from the Hamilton Rating Scale for Depression are difficult to interpret given the number of ill-structured items, but the scores did not indicate a significant degree of depression either. So, having excluded “subjects with depression or anxiety”, why did the authors claim in their discussion that “the main limitation of the present study is that our CFS subjects had high levels of depression”?

If this is correct, why was their depression not picked up by the three measures? Why were these patients not excluded from the research as stated by the authors or, funds permitting, used as a comparison group (Costa et al., 1995; Fischler et al., 1998)? How depressed were the 10 patients on antidepressants and, if these were not effective, could their suboptimal treatment have contributed to their ongoing fatigue?

I was also baffled by the authors’ suggestion that the thalamic hyperperfusion may reflect “increased attention to motor and cognitive tasks”. What were the patients doing? The abstract states that the scans were conducted at rest. If the subjects had just completed a battery of cognitive tests, why did the authors not check to see whether the data available supported their hypothesis (Fischler et al., 1998)?

If this paper was subjected to peer review, why did no one query the selective discussion of the findings and the misrepresentation of the literature on CFS and psychopathology?


E. Goudsmit 23 Melbourne Road, Teddington, Middlesex TW11 9QX

Authors’ reply: As explained in the method section, the potential participants were screened by excluding those scoring above case threshold in the Hospital Anxiety and Depression (HAD) scale, a self-rating scale that does not require a detailed interview. The remaining participants were then interviewed using the Schedule for Affective Disorders and Schizophrenia to further exclude any current mental illness.

First, in the discussion we say: “The main limitation of the present study is that our CFS subjects had high levels of depression: almost half were on psychotropic medication and five had a previous history of depression.” “High levels of depression” is defined by what follows the colon. There is, therefore, no contradiction. Participants were not currently depressed, but some were receiving antidepressant medication and some had previously been depressed.

Second, regarding that point made relating to our comment that “thalamic over-activity in CFS (and depression) may, therefore, reflect increased attention to motor and cognitive tasks . . .”. The perceived contradiction is that participants were at rest during uptake of the tracer, i.e. not currently engaged in motor or cognitive tasks. It is clearly speculative that increased thalamic activity at rest will also mean increased thalamic activity during tasks. What was implied, however, was that increased baseline or resting activity of the thalamus may be an underlying brain marker that is related to patients being more attentive to motor and cognitive activity, as they occur.

S. M. MacHale, S. M. Lawrie, J. T. Cavanagh, M. P. Glabus, C. L. Murray, K. P. Ebmeier Department of Psychiatry and MRC Brain Metabolism Unit, University of Edinburgh, Kennedy Tower, Royal Edinburgh Hospital, Morningside Park, Edinburgh EH10 5HF

G. M. Goodwin University Department of Psychiatry, Warneford Hospital, Oxford OX3 7JX
Psychiatrists can cause stigma too
R. Chaplin
BJP 2000, 177:467.
Access the most recent version at DOI: 10.1192/bjp.177.5.467

References
This article cites 4 articles, 1 of which you can access for free at:
http://bjp.rcpsych.org/content/177/5/467.1#BIBL

Reprints/permissions
To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at
/letters/submit/bjprcpsych;177/5/467

Downloaded from
http://bjp.rcpsych.org/ on April 13, 2017
Published by The Royal College of Psychiatrists

To subscribe to The British Journal of Psychiatry go to:
http://bjp.rcpsych.org/site/subscriptions/