clozapine in such patients may be unjustified as the patients may be denied proper treatment.

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M. D. Adityanjee, V. Chowdary Jampala Psychiatry Service, Veterans Affairs Medical Center, 4100W. Third Street, Dayton, OH 45434, USA

Madan S. Mohan Department of Psychiatry, Wright State University School of Medicine

Establishing cost-effectiveness of antipsychotic drugs

Sir: In commenting on the work of Aitchison & Kerwin (1997), Robert & Kennedy (1997) make a plea for more long-term, large-sample, randomised, controlled trials evaluating the health–economics of antipsychotic prescribing. While I agree that such studies would indeed be useful, I feel that open, mirror-image studies like that of Aitchison & Kerwin have greater practical value. As a manager of a trust drugs budget, I need to know the cost-effectiveness of drugs used under normal clinical circumstances in an environment similar to, or the same as, my trust. Indeed, purchasers of health care often demand parochial health-economic trials before they will consider funding, showing only a passing interest in large-scale multi-centre, randomised controlled trials, despite their scientific rigour. This is because, with all their inclusions, exclusions and intensive observations, these studies only indirectly reflect clinical practice.

Let us conduct more long-term, controlled studies, but let us also note carefully the results of open, uncontrolled trials which better reflect the real world.

Aitchison, K. J. & Kerwin R. W. (1997) Cost-effectiveness of clozapine: a UK clinic-based study. *British Journal of Psychiatry*, **171**, 125–130.

Robert, G. & Kennedy, P. (1997) Establishing cost-effectiveness of atypical neuroleptics. *British Journal of Psychiatry*, **171**, 103–104.

David M. Taylor Bethlem and Maudsley NHS Trust, Maudsley Hospital, Denmark Hill, London SE5 8AZ

Sir: In their excellent article, Robert & Kennedy (1997) reference two reviews in the Cochrane Library (*http://archie.cochrane.co.uk,info/*) (Adams & Soares, 1997). These are now completed. One, on clozapine, has been available for some time and has been updated every three months since publication (Wahlbeck *et al*, 1997). This will soon contain what data are available from recent trials that have, at last, looked at economic outcomes. The review on risperidone is also complete (Kennedy *et al*, 1997) and the next version of the Cochrane Library will contain this as well. What few data there are on cost-effectiveness will be presented. A similar review on olanzapine is starting. Every attempt is made to keep the systematically conducted reviews on the Cochrane Library up-to-date. Abstracts of completed reviews and titles of those underway are readily available (*http://archie.cochrane.co.uk/info/abstracts/abidx.htm#G06@*).

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Clive E. Adams Summertown Pavilion, Middle Way, Oxford OX2 7LG

Disorders of the mother–infant relationship

Sir: Professor Kumar's (1997) questionnaire study of 44 women with disorders of maternal 'bonding' has confirmed the duration of the disorder and the importance of parturition stress. I am surprised that he failed to find a link with unwanted pregnancy and disorders of prepartum affiliation because these are supported by other studies (Arbeit, 1975). He seems not to have considered the child's contribution to this relationship.

My most serious concern, however, is his remark about treatment. There is no discussion of treatment in the text, but the table of "clinical implications" states that "there are as yet no specific therapies". The word 'specific' presumably means therapies tailor-made to address this particular problem, not any other. The principles of treatment were set out in my earlier account (Brockington, 1996, pp. 347–360): they include specific elements directed at mother–infant interaction. Professor Kumar's negative remark about therapy may give a misleading impression to sufferers and their therapists. These are severe disorders, they are common and neglected, but, provided the disordered mother–infant relationship is identified, and not subsumed under a label such as 'postnatal depression', the treatment response is excellent.

Arbeit, S. A. (1975) *A Study of Women During their First Pregnancy*. PhD Thesis, Yale University.

Brockington, I. F. (1996) *Motherhood and Mental Health*. Oxford: Oxford University Press.

Kumar, R. C. (1997) "Anybody's child": severe disorders of mother-to-infant bonding. *British Journal of Psychiatry*, **171**, 175–181.

I. F. Brockington Department of Psychiatry, The University of Birmingham, Queen Elizabeth Psychiatric Hospital, Mindelsohn Way, Birmingham B15 2QZ

Neuropsychological and imaging differences in dementia with Lewy bodies and Alzheimer's disease

Sir: We agree with the recent editorial by Miller (1997) that recognition of the characteristic features of the different dementias has become increasingly important, especially in view of new treatment possibilities. We are surprised, however,

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by the statement that "Neuropsychological and neuroimaging studies have not shown robust differences between DLB and AD subjects". There is considerable evidence from published data to suggest that there is a clear difference in the neuropsychological profile between dementia with Lewy bodies (DLB) and Alzheimer's disease (AD) and that this could contribute to making the differential diagnosis.

The literature on the neuropsychological profile of DLB has been extensively reviewed by Salmon & Galasko (1996). They summarise earlier work by Hansen's group which showed greater deficits in attention, verbal fluency and visuospatial processing in nine pathologically confirmed Lewy body variant patients compared with 10 AD patients, as well as data from the Newcastle Group, using the CANTAB, a computerised battery of tests. In the latter study DLB patients were more impaired on delayed matching to sample, conditional learning paired associate tasks and spatial working memory. This difference in visuospatial ability was thought to reflect greater dysfunction of frontal lobe structures. The visuospatial differences have recently been shown to be of sufficient magnitude to be detected even by tests commonly used in clinical practice, e.g. on the CAMCOG (Walker *et al*, 1997*a*) and the Clock Face Test (Gnanalingham *et al*, 1997), suggesting those differences could be utilised to distinguish DLB from other dementias in a clinical setting.

As for neuroimaging studies, both perfusion studies (Albin *et al*, 1996) and dopamine receptor imaging (Walker *et al*, 1997*b*) have shown significant differences in the two conditions, suggesting that those might be a further aid to differentiate the two conditions.

Albin, R. L., Minoshima, M. D., D'Amato, B. S., *et al* (1996) Fluorodeoxyglucose positron emission tomography in diffuse Lewy body disease. *Neurology*, **47**, 462–466.

Gnanalingham, K. K., Byrne, E. J., Thornton, A., *et al* (1997) Motor and cognitive function in Lewy body dementia: comparison with Alzheimer's and Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **62**, 243–252.

Miller, B. L. (1997) Clinical advances in degenerative dementias. *British Journal of Psychiatry*, **171**, 1–3.

Salmon, D. P. & Galasko, D. (1996) Neuropsychological aspects of Lewy body dementia. In *Dementia with Lewy Bodies* (eds R. Perry, I. McKeith & E. Perry), pp. 99–113. Cambridge: Cambridge University Press.

Walker, Z., Allen, R. L., Shergill, S. S., *et al* (1997*a*) Neuropsychological performance in Lewy body dementia and Alzheimer's disease. *British Journal of Psychiatry*, **170**, 156–158.

—, Costa, D. C., Jansen, A. G., *et al* (1997*b*) Dementia with Lewy bodies: a study of post synaptic dopaminergic receptors with iodine-123 iodobenzamide single-photon emission tomography. *European Journal of Nuclear Medicine*, **24**, 609–614.

R. L. Allen Essex and Herts Community NHS Trust
Z. Walker, C. L. E. Katona Department of Psychiatry, University College London Medical School, Wolfson Building, Riding House Street, London W1N 8AA

Tests of 'dissociation' and mood disorder

Sir: Nijenhuis *et al* (1997) maintain that what Fahy (1988) and Merskey (1992) regard as the misdiagnosis of some cases of bipolar disorder as dissociative conditions (particularly multiple personality disorder) permits the hypothesis that, if we are right, instruments measuring dissociative pathology should give high scores, both in patients with supposed dissociative disorders and in those with bipolar affective disorder. Their claim is invalid. Patients with bipolar disorder may be misdiagnosed in at least two ways. First, as in the double consciousness cases, the existing natural phenomena are simply misread for honest, but antiquated reasons. Second, patients with bipolar disorder may be educated into producing the desired states that are to be labelled dissociative. It was not to be expected that patients with bipolar disorder would match the dissociative disorder group on these scales unless the former had been indoctrinated. Nijenhuis *et al* have compared un-indoctrinated subjects with others whom they consider to be dissociative and have obtained a number of highly significant statistical results which are predictable, but not for the reason they suppose. They comparison that has been offered is worthless.

Fahy, T. A. (1988) The diagnosis of multiple personality disorder. A critical review. *British Journal of Psychiatry*, **153**, 597–606.

Merskey, H. (1992) The manufacture of personalities. The production of multiple personality disorder. *British Journal of Psychiatry*, **160**, 327–340.

Nijenhuis, E. R. S., Spinhoven, P., Van Dyck, R., *et al* (1997) Dissociative pathology discriminates between bipolar mood disorder and dissociative disorder (letter). *British Journal of Psychiatry*, **170**, 581.

H. Merskey London Psychiatric Hospital, University of Western Ontario, 850 Highbury Avenue, PO Box 2532, London, Ontario, Canada N6A 4H1

Large same-year effects: fact or artefact?

Sir: Kessler *et al* (1996) report inordinately large odds ratios for 'same-year' effects for major depressive disorder (MDD) and comorbid disorders (see Table 4). An examination of the estimation method indicates that the large odds are likely to be an artefact.

The authors used survival models to estimate time-lagged, same-year, and time-trend effects (Table 4). The three time-dependent covariates were: (a) same-year – z_1=1 if MDD and prior disorder occurred in the same year, 0 otherwise; (b) time-lagged – z_2=1 if prior disorder occurred one or more years prior to onset of MDD, 0 otherwise; (c) time-trend – x=number of years since onset of prior disorder.

The estimation of the odds ratio is similar to that of the Mantel–Haenszel approach with two-by-two tables defined at each event (MDD) time (Crowley, 1975, 1980). For z_1 this table would be:

		MDD	
		Yes	No
Other disorder and MDD in same year	Yes	A	B
	No	C	D

By definition, cell B in the table is always 0; no subject can be both 'no' for MDD and 'yes' for other disorder and MDD in the same year. This 'structural zero' inflates the odds ratio for the 'same-year' effect, which could account for the high odds ratios in Table 4.

An approach that avoids this artefact (although still problematic, as indicated below) would be to include two binary-time dependent variables: z_3=1 if time since prior disorder is less than one year, 0 otherwise; z_4=1 if time since prior disorder is greater than one year, 0 otherwise.

Variable z_3, which measures within-year effect, does not result in a structural zero. A subject may have a prior disorder for less than a year but not have MDD when evaluated at another subject's event time. In other words, whereas z_1 requires that the onsets of the two disorders *within an individual* occur in the same year, z_3 does not.

To illustrate the two approaches, a simulation study was run. The generated data consisted of 1000 observations from an exponential distribution. The prevalence of MDD and prior disorder were set at 20%. Distributions were chosen so that the MDD odds ratio for all years including

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