Correspondence

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Problem substance use and schizophrenia

McCreadie *et al* (2002) report on the problem use of drugs and alcohol by people with schizophrenia. This is an excellent study as the pattern of use by patients is compared with controls from the general population, although the findings – that problem use is greater among patients – are unsurprising. It is impressive that the study included tobacco use, often disregarded as a 'problem' drug despite the obvious financial implications for patients surviving on state benefits. Previous studies quoted in the paper indicate that patients with schizophrenia often have been smoking for many years prior to the onset of the illness.

We are very interested to know whether the data collected for the study show that particular groups of patients appear to be more at risk from problem substance use, in order to focus efforts on helping them. Our experience is that admission to a psychiatric ward leads to increased tobacco use, and patients who have given up smoking recommence and continue smoking post-discharge, despite anti-smoking strategies. Also, we would like to know whether the study shows, or the authors know of, cultures that may be at lesser risk for developing problem use, accepting that numbers of ethnic minorities in the study sample may be small.

McCreadie, R. on behalf of the Scottish Comorbidity Study Group (2002) Use of drugs, alcohol and tobacco by people with schizophrenia: case–control study. *British Journal of Psychiatry*, **181**, 321–325.

P. Bates, J. Rutherford Tolworth Hospital, Red Lion Road, Surbiton KT6 7QU, UK

Author's reply: Bates & Rutherford raise some interesting points. I am impressed by their experience that admission to a psychiatric ward leads to increased tobacco use; this is certainly worthy of more-detailed study.

Smoking habits of people with schizophrenia probably do differ in different cultures. Colleagues in south India, where I have carried out much research, have found that people with schizophrenia probably smoke less than the general population. This is largely for economic reasons. Most are unemployed or in part-time employment. There are no state benefits, and therefore patients cannot afford to buy cigarettes.

R. G. McCreadie Department of Clinical Research, Crichton Royal Hospital, Dumfries DG1 4TG, UK

Betel use and schizophrenia

The Scottish Comorbidity Study Group has highlighted again the problem of greater use of drugs and alcohol, and especially tobacco, among patients with schizophrenia (McCreadie *et al*, 2002). An underrecognised comorbidity, however, especially in developing countries, is that of chewing betel nut (*Areca catechu*), along with the betel leaf (*Piper betle*) and lime.

In a preliminary study conducted in the North Colombo Teaching Hospital, Sri Lanka, we observed that a higher proportion of patients with schizophrenia chewed betel compared with control subjects. The frequency of chewing betel was also higher among the patients with schizophrenia. A recent study from Micronesia (Sullivan *et al*, 2000) has shown that betel chewing may in fact have a beneficial effect on patients with schizophrenia in terms of reducing both positive and negative symptoms. They postulate that the muscarinic agonist action of the betel nut alkaloid, arecoline, may provide an explanation.

However, betel chewing is an important risk factor for oro-pharyngeal carcinoma, and contributes significantly to oral health-related morbidity and mortality (Trivedy *et al*, 2002). Thus, the dual diagnosis of schizophrenia and betel chewing should not be missed, and services to address this problem should receive priority in many developing countries.

McCreadie, R. G. on behalf of the Scottish Comorbidity Study Group (2002) Use of drugs, alcohol and tobacco by people with schizophrenia: case–control study. *British Journal of Psychiatry*, **181**, 321–325.

Sullivan, R. J., Allen, J. S., Otto, C., *et al* (2000) Effects of chewing betel nut (*Areca catechu*) on the symptoms of people with schizophrenia in Palau, Micronesia. *British Journal of Psychiatry*, **177**, 174–178.

Trivedy, C. R., Craig, G. & Warnakulasuriya, S. (2002) The oral health consequences of chewing areca nut. *Addiction Biology*, **7**, 115–125.

K. A. L. A. Kuruppuarachchi, S. S. Williams Faculty of Medicine, University of Kelaniya, PO Box 6, Thalagolla Road, Ragama, Sri Lanka

ECT and old age psychiatry

The editorial on electroconvulsive therapy (ECT) by Eranti & McLoughlin (2003) describes the current status of ECT as a treatment option. They have noted that the use of ECT is declining and also highlight that in a study from Edinburgh (Glen & Scott, 1999) a reduction in the number of recipients aged 18–65 was noted. While discussing the future of the ECT clinic, they raise concerns about how this reduced use could lessen clinical interest and how this could, in turn, affect future psychiatric trainees with respect to obtaining experience in ECT. They rightly highlight the need for this treatment option to be readily available for our patient group.

In this context, I want to report some of the findings from a study my colleagues and I presented as a poster at the annual meeting of the Royal College of Psychiatrists in Edinburgh in July 2000. The study looked at trends in ECT usage in the busy ECT clinic at the Royal London (St Clement's) Hospital. It was a retrospective, chart-based study, covering a 3-year period during 1996–1999. Demographic and clinical data, which included response to ECT, were noted. There was a reduction in the number of patients who received ECT from 25 (171 total ECT episodes) in the year 1996/1997 to 12 (113 ECT episodes) in the year 1998/1999. Of the patients who received ECT, 70% were women and about 65% of the sample were aged above 60 years. A good response was noted in 45% of patients and, of this group, 70% were aged more than 60 years. The most common indication was depression and most of the findings of the practice of

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ECT at the unit were in keeping with national trends reported by the Department of Health (1999). Over this 3-year period, consultant groups in the unit remained largely unchanged.

Concluding from this study, I feel that ECT is more commonly used in treating older people with depression. Availability of newer antidepressants and other treatment modalities, as highlighted by Eranti & McLoughlin (2003), could be some of the reasons why there is a decline in the number of patients under 65 who receive ECT. Furthermore, the limited response to ECT in the subjects of our study could be due to the fact that these patients had been treatment-resistant. On the other hand, in the case of older people suffering from severe depression, there are other factors that tilt the treatment options towards ECT. Factors such as physical frailty, propensity to develop side-effects from antidepressants, and the serious effects of dehydration and weight loss (as a result of severe depression) make it imperative that depression is controlled rapidly.

I feel that in the future, it will be old age psychiatrists who will be using ECT more commonly as a treatment option for depression. Old age psychiatrists could take a leading role in ensuring that psychiatric trainees have the opportunity to obtain experience in ECT. The effective (albeit reduced) use of ECT resulting in good clinical outcomes will ensure that clinical interest in this treatment modality is maintained.

Department of Health (1999) *Electro Convulsive Therapy: Survey Covering the Period from January 1999 to March 1999, England* (Bulletin 1999/22). London: Department of Health.

Eranti, S. V. & McLoughlin, D. M. (2003) Electroconvulsive therapy – state of the art. *British Journal of Psychiatry*, **182**, 8–9.

Glen, T. & Scott, A. I. F. (1999) Rates of electroconvulsive therapy use in Edinburgh (1992–1997). *Journal of Affective Disorders*, **54**, 81–85.

M. Bhat South Essex Partnership NHS Trust, Basildon Hospital, Basildon, Essex SS16 5NL, UK

Lithium augmentation in treatment-refractory unipolar depression

Stimpson *et al* (2002) have taken an 'all or nothing' approach to evaluating randomised controlled trials (RCTs) for their systematic review. Their rigorous procedures eliminated over 98% of the 919 RCTs considered (although we note that the flow chart in Fig. 1 appears to 'lose' 166 of them without explanation). As a consequence, they have provided a matchless summary of the very best evidence about intervention for treatment-refractory unipolar depression but have left undescribed the very large quantity of remaining levels of evidence.

In 1999 Bauer and Dopfmer identified 11 placebo-controlled studies of lithium augmentation. As always, the trials were of varying quality; nevertheless, they concluded (using the three studies of highest quality, two of which were used by Stimpson *et al*) that there is 'firm evidence' in favour of lithium as an augmentation strategy for treatment-refractory unipolar depression, with a number needed to treat of 3.7. They supported their conclusion by performing a separate analysis adding a further six studies (that used either lower doses or shorter duration of lithium augmentation) and found a similar, indeed slightly stronger, effect size (Bauer & Dopfmer, 1999).

We note that there have been no studies of lithium augmentation against placebo for treatment-resistant unipolar depression that are of a suitable quality for a systematic review in the approximately 3-year period between the acceptance dates of the two papers cited above. We suggest that many clinicians now consider the weight of evidence (at many levels) supporting the use of lithium as an augmentation strategy for treatment-refractory unipolar depression sufficiently compelling. Thus, it is unusual for our service dedicated to treatment-resistant depression to receive referrals of patients not yet tried on lithium. Although further and better RCTs of lithium augmentation would be welcome (even Bauer & Dopfmer identified only 234 subjects studied), many would feel that other questions now have more clinical salience. Pressing examples might include whether psychological treatments are effective in these patients, how they compare with lithium augmentation, and how olanzapine augmentation (for which a large body of evidence is emerging; see Dube *et al*, 2002) compares with both.

Bauer, M. & Dopfmer, S. (1999) Lithium augmentation in treatment-resistant depression: meta-analysis of placebo-controlled studies. *Journal of Clinical Psychopharmacology*, **19**, 427–434.

Dube, S., Anderson, S. W., Paul, S., *et al* (2002) Metaanalysis of olanzapine–fluoxetine use in treatment resistant depression. *International Journal of Neuropsychopharmacology*, **5** (suppl. 1), 105–106.

Stimpson, N., Agrawal, N. & Lewis, G. (2002) Randomised controlled trials investigating pharmacological and psychological interventions for treatment-refractory depression. Systematic review. *British Journal of Psychiatry*, **181**, 284–294.

W. Lee Department of Psychological Medicine, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK

A. Cleare Department of Psychological Medicine, Institute of Psychiatry, London, and Maudsley Hospital, London, UK

Authors' reply: According to Drs Lee and Cleare 'many clinicians' regard the current evidence for lithium augmentation in treatment-refractory depression as 'compelling'. They are correct in repeating one of the principles of evidence-based medicine, that all levels of evidence need to be taken into account when making clinical decisions.

Previous systematic reviews of this area have included patients who have had $\leqslant 3$ weeks' treatment with an antidepressant or who have bipolar disorder. We do not think that many UK psychiatrists would consider lithium augmentation in unipolar depression that had not responded to an antidepressant for only 3 weeks. For patients with bipolar disorder, most UK psychiatrists, we think, would in any case be treating with lithium or another mood-stabiliser. Our inclusion criteria, which were set before the review started, were based therefore upon sensible and pragmatic clinical considerations.

We too were surprised and shocked by the lack of randomised evidence to support lithium augmentation; but it is also important to remember that lithium may well be effective, even though the evidence to support its use is extremely weak.

Lithium has a number of potentially serious side-effects, even at normal therapeutic doses (Bell *et al*, 1993). When we discuss the advantages and disadvantages of lithium with our patients we are unable to provide them with much more than clinical anecdote in its favour. We certainly have no idea from empirical research about the severity of depression for which lithium augmentation might be effective.

We have a collective responsibility to our patients to provide them with good-quality research evidence to justify the treatments we recommend. As a profession we need to address areas of uncertainty

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