specific risk for a form of psychotic illness characterised by features of both mania and mood-incongruent psychosis (Green *et al*, 2005). Other findings of a similar nature are currently emerging from our own studies and those of other groups, and we anticipate that we are entering a period during which psychiatric research and practice will be placed on much firmer nosological foundations than has been possible in the past.

Declaration of interest

N.C. and M.J.O. are consultants to GlaxoSmithKline and have received grant funding and honoraria from GlaxoSmithKline, AstraZeneca and Lilly.

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CBT for refractory symptoms in schizophrenia

Valmaggia *et al* (2005) report an interesting randomised controlled trial evaluating cognitive–behavioural therapy (CBT) for refractory psychotic symptoms of schizophrenia resistant to atypical antipsychotic medication. They conclude that patients should not be excluded from psychological help on the grounds that they are too ill to benefit from therapy, and CBT for psychotic symptoms should be available in in-patient facilities.

We feel the conclusions drawn by the authors do not truly reflect their results. Valmaggia *et al* report that their primary hypothesis was that CBT would be more effective than supportive counselling in reducing auditory hallucinations and delusional beliefs. They used the Positive and Negative Syndrome Scale (PANSS) and Psychotic Symptoms Rating Scale (PSYRATS) to measure outcomes. The post-treatment score on the PANSS positive sub-scale of those receiving CBT was not significantly different from that of the control group. On the PSYRATS no significant effect was found on the delusions. Benefits of CBT were found on the auditory hallucinations scale for physical characteristics and cognitive interpretation but not for emotional characteristics. However, the benefits noticed were not sustained at follow-up. It would have been helpful if the authors had used an *a priori* definition of what constitutes a clinically meaningful improvement and provided the actual figures for the dichotomous outcome.

Also, if we look at the numbers needed to treat (NNT) calculations, the authors have accurately reported the lack of statistical significance (PANSS positive symptom scale, NNT=8, 95% CI 3–∞; PSYRATS factor 2, NNT=6, 95% CI 2–∞; delusion scale factor 1, NNT=4, 95% CI 2–∞; factor 2, NNT=12, 95% CI 3–∞). The only finding with reasonable confidence intervals seems to be cognitive interpretation on the auditory hallucination scale of the PSYRATS (NNT=3, 95% CI 2–13). The authors also draw our attention to the fact that clozapine is effective in 32% of cases in producing a clinical improvement (NNT=5, 95% CI 4–7; Wahlbeck *et al*, 1999). They seem to suggest that the figures from the current study reveal the effects of CBT to be similar to clozapine. However, it should be noted that this figure reported by Wahlbeck *et al* is for global improvement, whereas Valmaggia *et al* do not give any figures for global improvement and hence in our opinion these results are not comparable. To conclude from these results that CBT could induce a change in psychotic symptoms seems to be overestimating the beneficial effects.

Patients with schizophrenia who are resistant to clozapine form one of the most difficult-to-treat groups. Jones *et al* (2004) concluded that trial-based data supporting the wide use of CBT for people with schizophrenia or other psychotic illnesses are far from conclusive. The randomised controlled study of Valmaggia *et al* evaluating interventions in this population is welcome.

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Olanzapine co-therapy in bipolar disorder

Baker *et al* (2004) report an interesting *post hoc* analysis from a randomised double-blind, placebo-controlled study evaluating the efficacy of olanzapine co-therapy in patients with bipolar disorder who had adequate responses to valproate or lithium monotherapy (Tohen *et al*, 2002). The authors describe a secondary analysis assessing response among dysphoric and non-dysphoric patients with bipolar I disorder.

The authors conclude that olanzapine in combination with either lithium or valproate was effective in improving the severity of depressive symptoms coexisting with acute mania. This conclusion is based on statistically significant differences in mean changes in Hamilton Rating Scale for Depression (HRSD) score. However, the authors have not reported the standard deviations for these mean changes. Hence it is difficult to ascertain whether the data are skewed. It is possible that a few patients showing large changes on the HRSD could have skewed the data. It was also puzzling that the authors reported that the difference in the HRSD score between combination and monotherapy groups was larger for dysphoric patients. One would expect participants in the non-dysphoric group to have much lower baseline scores so that there would be less chance of a significant reduction. (The mean HRSD baseline score in the non-dysphoric group was 10.42 (s.d.=5.27) and in the dysphoric group 25.18 (s.d.=4.62).)

We are also of the view that reporting study outcomes in terms of mean changes on a rating scale does not provide meaningful information for clinicians. Reporting results using dichotomous outcome

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measures such as 'improved' and 'not improved' with a meaningful cut-off point defined *a priori* would be helpful. Clinicians would be more interested in outcome measures such as complete remission of symptoms, return to premorbid levels of functioning, etc. To address the question of whether olanzapine is helpful for patients with dysphoric mania it would be helpful to know how many in the olanzapine co-therapy group achieved complete remission and whether there was any statistical difference between groups. It would have been interesting if Baker *et al* had also provided dichotomous outcomes based on the Clinical Global Impression scale for bipolar disorder (CGI–BP; Spearing *et al*, 1997), as this was administered during the course of the trial and data should be readily available.

It is not uncommon to come across reporting of various outcome measures and multiple analysis of a randomised controlled trial. However, whether this adds to clinical knowledge is questionable. We agree with Baker *et al* that it is important to explore the pharmacological options for dysphoric mania as the available options are limited. However, we need more pragmatic outcome measures that are easily understood by clinicians and can be applied in routine practice rather than being lost in multiple analysis. Systematic reviews such as that on the use of olanzapine for mania also highlight the lack of pragmatic outcome measures in the reporting of randomised controlled studies (Rendell *et al*, 2003). We hope future reports of such studies will use outcome measures that are more applicable to the real world.

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ECT in depression

Schulze-Rauschenbach *et al* (2005) found in their comparison of unilateral electroconvulsive therapy (ECT) and repetitive transcranial magnetic stimulation (rTMS) that these two procedures have similar efficacy in the treatment of major depression. However, the rate of treatment response for ECT in their study was 46%, well below the figures found in other studies (Medical Research Council, 1965). The authors state that the response rate for ECT might have been higher if a higher dosage had been used, but that this would have increased the risk of side-effects. This argument is misleading, just as comparing a sub-therapeutic dose of amitriptyline and placebo would be. The authors should have compared the incidence of side-effects between treatments, but at therapeutic doses. This comparison would probably have confirmed the prevalent belief that ECT is more effective than rTMS in the treatment of major depression (Aarre *et al*, 2003).

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Schulze-Rauschenbach *et al* (2005) compared repetitive transcranial magnetic stimulation (rTMS) and unilateral electroconvulsive therapy (ECT) and reported a similar treatment response rate. The rTMS methodology produced an impressive improvement with no cognitive side-effects.

However, the reported similar treatment effect with ECT could be misleading, as it is partly due to the rather low success rate of ECT in this study. The Hamilton Rating Scale for Depression (HRSD) score in the ECT group was reduced by a modest 35%. For comparison, the non-psychotic patients in the largest recent ECT study (the CORE study; Petrides *et al*, 2001) achieved a 74.5% reduction on the HRSD–24 (24-item version).

We started an audit of ECT at our regional psychiatric hospital 1 year ago. So far 23 consecutive patients with treatment-resistant depression, who had an HRSD–17 (17-item version) score of 15 or above (the cut-off used by Schulze-Rauschenbach *et al*), have completed at least six ECT sessions. We observed a 55% improvement on the HRSD–17: from 24.6 to 11.0 points. The decrease on the self-rated Beck Depression Inventory was 20.1 points (an improvement of 49.9%). This compares with a decrease of only 7.6 points (24%) in the ECT group of Schulze-Rauschenbach *et al*. Even more importantly, the remission rate in their study was very low. Using the remission criterion of $\leqslant 7$ points on the HRSD–17 (Thase, 2003), only one of their 13 ECT patients (8%) achieved remission (as shown in Fig. 1). This contrasts with a rate of 43.5% (10 out of 23 patients) in our study and 74.7% (189 out of 253 patients) in the CORE study. Four of our patients scored 0 or 1 point at the end of treatment.

There could be at least two reasons for the low response rate in the ECT group of Schulze-Rauschenbach *et al*. First, unilateral ECT is less effective than bilateral ECT, and when used at a simulation intensity of 100–150% above seizure threshold, it has produced only a 30% response rate (Sackeim *et al*, 2000). Only four patients in our series and none in the CORE study had unilateral ECT. Second, patients with psychotic depression respond better to ECT (Petrides *et al*, 2001). None of the patients of Schulze-Rauschenbach *et al* had psychotic symptoms, but 13 (56.5%) in our group and 77 (30.4%) in the CORE study did. This cannot explain all the difference, as the non-psychotic patients in our group still showed an improvement of 48% on both HRSD–17 and Beck Depression Inventory scores.

Properly administered bilateral ECT still remains by far the most effective treatment for severe depression.

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