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

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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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