related study (O'Connor *et al*, 2003), which we regretfully overlooked when we wrote our article. They point to similarities between the studies, but speculate about the reasons for the discrepant findings regarding the clinical efficacy of rTMS. We believe the following methodological differences might contribute.

First, in the study by O'Connor *et al* (2003), the level of baseline depression was different in the treatment groups: those receiving rTMS were significantly less depressed than those receiving ECT. Furthermore, those treated with rTMS were medication-free for at least 2 weeks but those receiving ECT continued to receive antidepressant medication. Finally, the duration of treatment – and the interval between initial and follow-up measurements – tended to be longer (2–4 weeks) in the ECT group than in the rTMS group (2 weeks). These features most likely contributed to the better clinical efficacy of unilateral ECT compared with rTMS in the study by O'Connor *et al*, where not a single patient treated with rTMS showed a clinically significant (50% reduction in the Hamilton Rating Scale for Depression) response.

In contrast, those treated with either rTMS or ECT in our study were matched for baseline levels of depression. They were treated for about 5 weeks on average. Antidepressant medication was kept constant in both ECT and rTMS treatment arms, and both treatments were clinically effective in about half of the patients. In principle, a comparative study of side-effects of two treatments only seems to be relevant when both modalities have a measurable clinical effect.

We agree that the effects of rTMS on mood and cognition may be independent of each other, and may point to different neural networks mediating these effects. However, the better retrograde memory performance after treatment, even in patients lacking an antidepressant response to rTMS, reported by O'Connor *et al*, is not necessarily suggestive of such a dissociation. It might also be explained by test repetition effects, which were masked in the ECT group because of enduring memory impairments. A healthy control group assessed repeatedly can be used to control for this confounding variable. We noted that patients receiving rTMS did not show stronger improvements over time than the control group for any objective cognitive measure, effectively ruling out a genuine memory-enhancing effect of rTMS as used in our study.

With the development of magnetic seizure therapy as possibly yet another form of brain stimulation for depression, the issue of relative benefits, side-effects and the duration of both will need further careful assessment. We have highlighted some of the methodological issues to be considered when studying the effects of different treatments on cognition.

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CBT for refractory psychotic symptoms

We read with interest the study of Valmaggia *et al* (2005), particularly noting that the interventions delivered were based on a comprehensive treatment manual and delivered by therapists specifically trained in the protocol.

By the authors' admission, some aspects of the intervention showed only modest benefit over supportive counselling; indeed the only outcomes when examining the 95% CI that provide support for cognitive–behavioural therapy (CBT) are physical characteristics of hallucinations and cognitive interpretation of hallucinations. At the same time, the 95% CI for negative symptoms (Positive and Negative Syndrome Scale) suggest that supportive counselling is more effective than CBT. In addition, the effects of 16 sessions of highly structured CBT disappeared at follow-up. We were therefore very surprised at the authors' conclusions that this therapy should be available within in-patient facilities. As experienced CBT clinicians and nurses, we are acutely aware that there is a serious shortage of CBT therapists and nursing staff available to provide therapist or 'manualised' CBT. Indeed, waiting lists of over 12 months are common for therapist-provided out-patient CBT. In turn, a very large number of in-patient wards rarely, if ever, see a psychologist, let alone have the capacity to train therapists and provide 16 h of therapy! Should we not be more prudent when making claims on such scant resources by first ensuring that we have adequate evidence to support such claims? Perhaps the editor should consider making obligatory a section in every paper relating to real-world implications.

Valmaggia, L. R., Van der Gaag, M., Tarrier, N., *et al* (2005) Cognitive–behavioural therapy for refractory psychotic symptoms of schizophrenia resistant to atypical antipsychotic medication. Randomised controlled trial. *British Journal of Psychiatry*, **186**, 324–330.

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Urban environment and schizophrenia

Selten *et al* (2005) cite two reasons for the increased risk of schizophrenia in Surinamese immigrants to The Netherlands. These are an increased base rate in the Surinamese population and exposure to an urban competitive Dutch society. These findings are of particular interest to researchers in Trinidad and Tobago because both countries share a similar mix of African and East Indian population and historically were simultaneously but independently developed by British and Dutch colonisers.

Interestingly, the authors noted that in their own study of Surinam and studies from Jamaica, Trinidad and Barbados no excess of schizophrenia was reported in the native countries. In addition, they argue that an overrepresentation of patients resident in Paramaribo points to an urban causation. The two reasons cited by the authors need further analysis.

The concept of urban environment causing disease is complex. Van Os (2004) proposes that the urban environment with a set of environmental factors acting between birth and the onset of illness is a risk factor for psychotic illness. However, Hutchinson & Morgan (2005) argue that the risk for psychosis is not specifically the urban environment but the social disadvantages and isolation experienced by vulnerable individuals in an urban society. These interact with perceptions of self, transgenerational expectations, cognitive processes and the urban environment to confer risk. Although both these views are tenable, is it not fair to assume that the variables described as associated with an urban environment will also be present in sub-urban or rural environments? It appears, then, that the effect lies in the confounding variables described rather than the urban effect.

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