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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'Toxicity' of any environment is determined by the stability of the social framework that governs the lives of individuals. It is debatable whether the variables of racism, alienation, political discrimination, unemployment, lack of opportunity, crime and fear of crime are more common in urban areas in developing countries. There is often no means of rural living for urban dwellers in these countries and many opt to escape through migration to foreign lands. Migration from the native country is therefore associated with a release from these stressful factors, as is the case of some ethnic groups in the Caribbean. In societies where environmental factors confer greater stress either in the native or the receiving country, the rates of psychosis will be higher and should not be attributed only to the base rates of the native country as proposed by Selten *et al*. If a social model were to be developed, consideration must be given to the time between assault and disease manifestation with a formula for lag time, rather than equating disease with geographical location at the time of manifestation.

The degree of urbanisation cannot simply be judged by the number of households per square kilometre. In developing countries, the division of areas into urban and rural is arbitrary; consideration must be given to the availability of basic amenities, geographical distance from cities and towns, the availability of newspapers and electronic media, the degree of literacy, transportation systems and the presence of household amenities. The fact that all people in Surinam have access to psychiatric care except for two remote districts that are looked after by medical missions suggests a movement away from rurality, since access to psychiatric care is a good index of development. Nevertheless, in many rural communities there is a distrust of Western psychiatric services and, as pointed out by Selten *et al*, help is often sought from traditional healers. This can result in statistical inaccuracies in both directions, through leakage of cases and delay in first contact with the psychiatric services.

Our findings in Trinidad suggest that gender and ethnicity are important variables in 'urbanisation'. In more urbanised areas, more males aged between 15 and 29 years presented with schizophrenia than females. The affected young males were more likely to be of African descent. A neuroprotective effect of oestrogen in females could be responsible for their low rates of schizophrenia, and neuronal plasticity in response to exposure to a new environment and its effects on the disease process is another area of possible future research.

Selten *et al* and others have raised important questions that are relevant to Caribbean people and those who have migrated and settled abroad. Cross-cultural differences, environmental factors and gender affect the risk for the development of psychosis but the final common pathway of any disease is at the molecular level. Genetic factors must therefore also be taken into consideration.

Hutchinson, G. & Morgan, C. (2005) Social development, urban environment and psychosis. *British Journal of Psychiatry*, **186**, 76–77.

Selten, J.-P., Zeyl, C., Dwarkasing, R., *et al* (2005) First-contact incidence of schizophrenia in Surinam. *British Journal of Psychiatry*, **186**, 74–75.

Van Os, J. (2004) Does the urban environment cause psychosis? *British Journal of Psychiatry*, **184**, 287–288.

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Authors' reply: We thank Dr Maharajh for his reaction. We agree with his observation that it is uncertain whether the urban effect is also operative in Surinam. The sample size of our study was too small for definitive conclusions and the possibility remains that some patients in rural areas do not see doctors.

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Advance directives and advance agreements

The paper by Amering *et al* (2005) adds to the growing literature on advance directives. The main difficulty with advance directives seems to be that with the available training programmes very few service users can be enthused to draft one. The authors recommend more training of service users and substantial administrative commitment from service providers.

The same could be said about advance agreements, another tool to empower patients to become partners in negotiating individualised treatment and care in time of crisis. Advance agreements (*Behandlungsvereinbarungen*) are widely used in German-speaking countries and according to a quick web search are offered routinely in at least 50 psychiatric hospitals in Austria, Switzerland and Germany.

Unfortunately no systematic research on advance agreements has been conducted in these countries; the only trial that has been published is from the UK (Henderson *et al*, 2004) and showed a significant reduction in the use of compulsory admission and treatment. Interestingly, advance agreements are seen as legally binding in Germany but not in the UK. Thomas & Cahill (2004) sceptically commented on the Henderson study that 'Liberation cannot be handed to the oppressed by the oppressor'. Basaglia (1979) would probably answer that this is precisely what the psychiatrist is supposed to do: 'to enter a dialogue with the patient, a dialogue not between subject and object, but between two human beings, who have become subjects. If we don't accept this logic of contradictions between two individuals, we should better trade bananas than work as doctors'.

Advance agreements, from the experience in German-speaking countries, are usually initiated by nurses and doctors working in in-patient settings, who have perhaps the strongest incentive to reduce compulsion in mental health (as those who restrain, detain and enforce treatment). Negotiating job plans with senior and junior doctors, with ward managers and nurses where time is allocated to discuss and draft advance agreements might be a way forward.

Amering, M., Stastny, P. & Hopper, K. (2005) Psychiatric advance directives: qualitative study of informed deliberations by mental health service users. *British Journal of Psychiatry*, **186**, 247–252.

Basaglia, F. (1979) The power of the state and psychiatry. Quoted and translated from: *Die Entscheidung des Psychiaters, Bilanz eines Lebenswerks*. Bonn: Psychiatrie Verlag, 2000.

Henderson, C., Flood, C., Leese, M., *et al* (2004) Effect of joint crisis plans on use of compulsion in psychiatric treatment: single blind RCT. *BMJ*, **329**, 136–138.

Thomas, P. & Cahill, A. B. (2004) Compulsion and psychiatry – the role of advance statements. *BMJ*, **329**, 122–123.

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Authors' reply: In practice, rights are only as visible as the mechanisms put in place

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