The data on atypical antipsychotic drugs, weight gain and metabolic dysregulation come from a heterogeneous collection of largely uncontrolled studies, but there is no doubt that these drugs induce weight gain and that some are worse than others. 'First do no harm'. There can be no justification for continuing to prescribe particular atypical antipsychotic drugs which cause serious weight gain to a population who are already at high risk of cardiovascular disease. Equally effective alternatives are readily available and are no more expensive. Obesity increases cardiovascular mortality by 50% (McGee, 2005). We must stop regarding weight gain as an acceptable price to pay for control of psychiatric symptoms.

## Declaration of interest

S.B. has attended many educational functions supported by pharmaceutical companies but has no other links with the pharmaceutical industry.

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## Akathisia as a risk factor for suicide

Hawton *et al* (2005) have produced a comprehensive, systematic review of risk factors for suicide in schizophrenia. The study questions the fundamental procedures that are an integral part of our clinical assessment of this vulnerable group of patients. Suicide is notoriously difficult to predict because of the rarity of the event, the obvious ethical problems of designing informative studies and the uncertainty about risk factors. However, although there is no study of akathisia and suicide that fulfils their strict inclusion criteria, there is more research available than the case reports mentioned (for example, Chow et al, 1997; Hansen, 2001; Hansen et al, 2004). We found no association between akathisia and suicidality in a group of 90 patients with treatment-resistant schizophrenia (Hansen et al, 2004). Akathisia may, however, have a very different impact on patients at different stages of their illness and according to the duration of treatment. Akathisia emerging early in treatment or after increases in dosages may be the more malignant in terms of distress.

Hawton *et al* also identified agitation (motor restlessness), impulsivity and depression as risk factors but not akathisia. However, akathisia could contribute to or be confused with any of these three identified risk factors.

There is also evidence that akathisia can occur as a consequence of antidepressant treatment, which is common in patients with schizophrenia (Muller-Oerlinghausen & Berghofer, 1999; Hansen & Wilkinson, 2001). Whether there is an additive effect of antipsychotic and antidepressant medication on the intensity and duration of akathisia is not yet known. None the less, in our opinion, it would be premature to exclude akathisia from a role in the complex web of factors that lead to suicide in schizophrenia and perhaps also in other conditions.

Chow, L. Y., Chung, D., Leung, V., et al (1997) Suicide attempt due to metoclopramide-induced akathisia. International Journal of Clinical Practice, **51**, 330–331.

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## Brief psychotherapy for Alzheimer's disease

I read with interest the paper by Burns *et al* (2005). This study into an under-researched and important matter is welcome. However, I would like to comment on the conclusions.

The authors quite appropriately comment that the lack of any quantifiable effect of their psychotherapy could result from the small sample size or the relative insensitivity of the outcome measures. They present qualitative data on participants' experience of the psychotherapy which show the therapy in a positive light. The collection of these data was highly biased, since participants in the 'standard care' arm of the trial were not asked about their experience of their treatment. In addition, these patients were not followed-up in the same way as those receiving the therapy. I suspect that if multidisciplinary, holistic care were being provided as it should, these patients would have made equally positive comments about their community psychiatric nurse, social worker, psychiatrist or general practitioner.

The authors of this study have neither devised the adapted therapy (this was described by Brierley *et al*, 2003), nor have they shown that the therapy works. Hence I disagree with the authors' main conclusion that 'this study shows it is possible to adapt a model of psychotherapy for those with Alzheimer's disease'. They have none the less presented some interesting preliminary data, suggesting a potential benefit of the therapy. I look forward to further research in this area.

**Brierley, E., Guthrie, E., Busby, C., et al (2003)** Psychodynamic interpersonal therapy for early Alzheimer's disease. *British Journal of Psychotherapy*, **19**, 435–446.

Burns, A., Guthrie, E., Marino-Francis, F., et al (2005) Brief psychotherapy in Alzheimer's disease.
Randomised controlled trial. *British Journal of Psychiatry*, 187, 143–147.

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Author's reply: Dr White has raised some important points. The qualitative data on the participants' experience was only a tiny part of the study and, although agreeing with the points made, I feel they are hardly relevant to the main thrust of the work. Dr White is correct that we did not devise