methods of SANGRA do not produce unexpected findings for this group.

The comparison with our study of the National Confidential Inquiry should be made cautiously, as that study included suicides among people in contact with services rather than from all deaths reported by the ONS.[2] We would also suggest self-harm rates are not a proxy for comparative suicide rates.

Dr Aspinall makes important comments about ethnicity classification. There are no data that investigate self-assigned *v.* ascribed ethnic identity and variations of this relationship across geographical areas of the UK, over time, or the patterns of transmission of ethnic identity through the generations. There are often unpleasant trade-offs when using descriptors of ethnicity and culture from survey research.[3] Ethnicity is not a measure of cultural identity.[3] Perhaps nested within self-reported ethnic categories we need more complex models of identity that take account of acculturation, social stratification and their interaction.[4] This may help more precisely to disentangle specific influences on health. Unfortunately, the concepts and methods to do this are still being developed.

The information on the denominators so far is useful but incomplete to forge a new study design or recommend specific changes in routine data sets; for example, we would need a breakdown of self-reported ethnicity in the 'Asian Other' and 'White and Asian' categories by gender and age. Adding more ethnic categories which are imprecisely measured, or for which the difference between self-rated and ascribed may vary over time and place, may lead to more random misclassification; therefore, more ethnic categories may not always be helpful or explain any more precisely which specific ethnic identity groups are at greater or lesser risk.

The finding of high rates of suicide in young South Asian women in the UK are based mainly on papers sampling groups born in Southern Asia – using the same methodology would miss the 50% of South Asians currently in the UK.[1] Of the two studies that used different methodologies, one used names to ascertain South Asian suicides but the methodology was not validated or described so that it could be replicated, and the other studied parts of London, although we know that there are significant differences in South Asian suicide rates by geographical location.[5]

The main purpose of the study was to improve the accuracy of the estimate of suicide rates in all South Asian people living in the UK, irrespective of place of birth. We know that over 50% of the South Asian population was born in the UK and future studies need a way of accurately including them in rate calculations. We believe that ethnicity assigned on death certificates is likely to be the most useful way forward. We concur with the view that there is a need to assess trends over time.

We agree that there are caveats because of the SANGRA program. However, we do not think that the program would have worked less well in the 1999–2003 cohorts than the 1993–98, that it would work so differentially for men and women, or that artefact can explain the findings in both the older and the younger female population.

We would welcome attempts to replicate these findings using different methodologies and show the raw data to facilitate this and the calculation of other statistics such as confidence intervals (Table 1). However, in the meantime, this is the first contemporary attempt to ascertain the suicide rates for the whole of the UK South Asian population. The findings for older women should be viewed with concern and the drop in younger women should be replicated and followed over time. Policy has to be based on the best evidence available rather than the best evidence that is sought and might one day become available.

Table 1 SANGRA South Asian Suicides 1993–2003

Age range, by gender	1993–1998	1999–2003
Male		
15–24	136	72
25–34	145	158
35–44	121	109
45–54	54	72
55–64	46	39
65–74	18	18
>74	14	10
Total	534	478
Female		
15–24	60	29
25–34	75	55
35–44	54	29
45–54	28	25
55–64	12	15
65–74	13	14
>74	6	11
Total	248	178

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5 Neeleman J, Wessley S. Ethnic minority suicide: a small area geographical study in south London. *Psychol Med* 1999; **29**: 429–36.

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Risperidone for adolescent schizophrenia

We would like to make a few comments on the study by Haas *et al*[1] which compares the efficacy and safety of two dosing regimes of risperidone. First, we would like to raise concerns with regard to the design of the study. Both groups were receiving a flexible dose of risperidone in the first 4 weeks. The dose was to remain stable only during the last 4 weeks – a very short duration. Patients started showing substantial response in the first week onwards. It is not possible to rule out the placebo effect and difficult to determine the dose-related response. Surely this design cannot establish the optimal effective dose as the dose was changing very often especially in the first 4 weeks.

Second, patients in the control group were not allowed the assured effective treatment. The control group received risperidone tenfold less than the intervention group. This dose was as good as a placebo. This raises serious doubts as to whether the lower dose was also effective or whether it was a placebo effect. This is clearly evident as a substantial improvement compared with baseline was noted in both groups within 7 days. It also raises ethical issues as the authors decided to continue a presumably ineffective dose (0.15–0.6 mg/day) in the control group for 8 weeks.[2] Patients in this group had a higher discontinuation rate owing to lack of efficacy. It was unethical to continue with such

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a low dose. We also wonder why the authors arbitrarily decided to have a tenfold lower dose in the control group. We question why the authors did not try to compare the intervention drug with an existing drug such as olanzapine, as Hill[3] reports that the key point is how a new treatment compares with existing treatment rather than whether it is better than nothing.

1 Haas M, Eerdekens M, Kushner S, Singer J, Augustyns I, Quiroz J, et al. Efficacy, safety and tolerability of two risperidone dosing regimens in adolescent schizophrenia: double-blind study. *Br J Psychiatry* 2009; **194**: 158–64.

2 World Medical Association. *World Medical Association Declaration of Helsinki. Ethical Principles for Medical Research Involving Human Subjects*. WMA, 2008.

3 Hill AB. Medical ethics and controlled trials. *BMJ* 1963; **1**: 1043–9.

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Authors' reply: Several of the limitations of our study design as mentioned by Jainer & Mahood have been addressed within the publication's discussion. The study was not designed to establish an optimal dose or evaluate efficacy *v.* placebo. Thus, as we noted, no conclusions can be made in this regard. The objective of this study was to determine whether there was a difference between two dose ranges; this goal was achieved. The use of an active comparator was not possible because there was no drug approved for use in children or adolescents suffering from this disorder at the time the study was conducted.

The dose ranges were chosen to compare the adult therapeutic dose, known to be effective in schizophrenia, with a low dose. This low dose was presumed subtherapeutic, but not known to be ineffective. Notably, in studies in children with disruptive behaviour disorder where the allowable flexible dose range included doses <0.6 mg/day, risperidone was shown to be efficacious.[1,2] Additionally, at the time this study was designed, a low-dose comparator was preferred over placebo, although thinking on the appropriateness of using placebo control in studies of antipsychotics has evolved since then.[3] A placebo effect in terms of treatment response cannot be ruled out in our study, and presumably any placebo response would have affected both dose arms similarly. Numerous safeguards were implemented to minimise risk to patients in the study from the outset. The protocol was reviewed by and received approval from an independent ethics committee and individual institutional review boards. All patients and caregivers were advised that both doses were experimental and the lower dose might be an ineffective treatment. Accordingly, all enrolled patients were initially hospitalised and only adequately stabilised patients could be discharged to continue in the trial as out-patients. Patients could discontinue treatments at any time. All patients were monitored closely throughout the duration of the trial to further ensure patient safety.

Our conclusions remain valid, as they pertain to the comparative favourable efficacy benefits achieved in this study with risperidone treatment in the 1.5–6.0 mg/day dose range compared with the lower range. Both regimens were well tolerated with low discontinuation rates due to adverse events.

Declaration of interest

The study was funded by Johnson & Johnson Pharmaceutical Research & Development, LLC. M.H. and M.E. are employees of Johnson & Johnson Research & Development, Division of Janssen Pharmaceutica, NV. S.K., J.S., I.A., J.Q., G.P. and V.K. are employees of Johnson & Johnson Pharmaceutical Research & Development, LLC.

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3 Stroup TS, Alves WM, Hamer RM, Lieberman JA. Clinical trials for antipsychotic drugs: design conventions, dilemmas and innovations. *Nature Rev* 2006; **5**: 133–46.

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Time to change concepts and terminology

The proposal by van Os to introduce 'salience dysregulation syndrome'[1] to describe the psychosis spectrum, replacing schizophrenia and bipolar disorder, represents an acceptance that such terms have outlived their usefulness. But by introducing three subcategories, 'with affective expression', 'with developmental expression' and not otherwise specified, he simply replaces outdated terms but retains the invalid and unreliable concepts – schizophrenia and bipolar disorder re-emerge with different names.

The evidence for a psychosis spectrum, as he describes, now seems irrefutable. At one end, manic symptoms 'represent the greatest diagnostic value' and this end of the continuum seems relatively recognisable and clinically relevant. Moving towards the other end takes us into Bleuler's schizophrenias and the more recently emerged area of drug-related psychosis. We have argued the case that rather than simply continuing to try to homogenise the schizophrenias, we should listen to what patients tell us led to their first episodes. Dudley *et al*[2] have recently used Q-sort methodology to elicit this and found similarities to concepts developed empirically from clinical practice.[3] We have used these concepts of drug-related, traumatic, stress-sensitivity (early-onset) and anxiety (late-onset) psychoses successfully with patients[4] and also found them to be destigmatising.[5] They are derived from work which Van Os himself has been pre-eminent in developing and we suggest to him that he has the courage of his convictions and use aetiological concepts rather than nebulous descriptive ones.

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