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.

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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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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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