Authors' reply: Birchwood *et al* make two points that require clarification. First, their statement that our findings from studies with high methodological rigour, particularly masking, imply that cognitive–behavioural therapy (CBT) has small but by no means negligible effects on positive and total symptoms 'broadly in line with the National Institute for Health and Care Excellence (NICE) review and particularly that of Wykes *et al*,' seems to us questionable. Wykes *et al*[1] reported an effect size of 0.37 for positive symptoms, which reduced slightly to 0.31 in masked studies. This latter value was four times larger than the value of 0.08 we found for masked studies of positive symptoms. Ratings of bias were made for the studies included in the 2009 NICE guideline;[2,3] however, no analyses excluding low-quality studies or otherwise examining methodological rigour were actually carried out.

Second, Birchwood *et al*'s argument that a finding of significant heterogeneity among studies implies that CBT is effective in certain subgroups of patients is not formally correct. It could simply mean that there are systematic differences in effect size between studies at high and low risk of bias. Tending to support this latter interpretation, in our meta-analysis of positive symptoms there was no significant heterogeneity in either the masked ($n=20$, effect size 0.08, $I^2=0\%$, $Q=18$, $P=0.49$) or unmasked studies ($n=8$, effect size 0.57, $I^2=23\%$, $Q=9$, $P=0.24$) when they were considered separately. Heterogeneity was also not significant in the masked studies of overall symptoms ($n=20$, effect size 0.15, $I^2=25\%$, $Q=25$, $P=0.15$), although it remained significant in the unmasked studies ($n=10$, effect size 0.62, $I^2=71\%$, $Q=31$, $P<0.001$).

Byrne argues that our findings are limited by not considering follow-up data. We presume he is arguing here for a 'delayed action' effect of CBT, as found in the 2000 study of Sensky *et al*[4] and an early meta-analysis by Pilling *et al*.[5] However, the meta-analyses carried out for the 2009 NICE guideline[2] provide only lukewarm support for such a view: the pooled effect sizes for overall symptoms were 0.27, 0.23, 0.40 and 0.19 at end of treatment, 6 months', 12 months' and 12–18 months' follow-up respectively, when CBT was compared with standard care; they were 0.13 at end of treatment and 0.18 at 12 months when CBT was compared with other active treatments.

Among the other issues raised, whether there is evidence for a dose effect for CBT seems to us essentially imponderable, since none of the 50+ published randomised controlled trials to date has manipulated dose or duration of the intervention. Such an effect would also likely be difficult to detect using meta-analytic methods, given the many other sources of variation among the existing studies. With respect to whether or not CBT should be considered a 'quasi-neuroleptic', we simply note that CBT was originally developed for and continues to be promoted as a treatment for positive symptoms.

1 Wykes T, Steel C, Everitt B, Tarrier N. Cognitive behavior therapy for schizophrenia: effect sizes, clinical models, and methodological rigor. *Schizophr Bull* 2008; **34**: 523–37.

2 National Institute for Health and Clinical Excellence. *Schizophrenia: Core Interventions in the Treatment and Management of Schizophrenia in Adults in Primary and Secondary Care (Update)* (Clinical guideline CG82). NICE, 2009.

3 National Institute for Health and Clinical Excellence. *Schizophrenia (Update). Appendix 15c: Psychological Therapies and Psychosocial Interventions Study Characteristics Tables*. NICE, 2009.

4 Sensky T, Turkington D, Kingdon D, Scott JL, Scott J, Siddle R, et al. A randomized controlled trial of cognitive-behavioral therapy for persistent symptoms in schizophrenia resistant to medication. *Arch Gen Psychiatry* 2000; **57**: 165–72.

5 Pilling S, Bebbington P, Kuipers E, Garety P, Geddes J, Orbach G, et al. Psychological treatments in schizophrenia: I. Meta-analysis of family intervention and cognitive behaviour therapy. *Psychol Med* 2002; **32**: 763–82.

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Stimulant treatment for ADHD

We read with great interest the article by Groenman *et* al,[1] which highlights an important facet concerning substance use in attention-deficit hyperactivity disorder (ADHD).

The authors suggested, through the generalised estimating equation model, that the risk of developing substance use disorder reverses after 18 years of age, indicating that it may be mediated by modulation in parental support. However, we wish to raise concern for this conclusion as a possible biased finding since the researchers have included patients exposed to stimulants intermittently or for short durations along with those exposed continuously ($n=358$), which may have falsely led to the results. Possibly, analysis of the combined no-stimulant treatment group (stimulant-naive and those with short or inconsistent stimulant use) against the stimulant treatment group for age variable (as had been done in the correlation analysis) may have validated the statement.

In what appears to be a printing mistake, Table 1 incorrectly shows the percentage of males in the no-stimulant group as being 9.0%, which must be higher given the *n* in this group (36/61).

Meta-analysis also concludes that treating ADHD during childhood reduces the incidence of substance use disorder by half, whereas failure to treat doubles the risk for substance use disorder.[2] We concur with the authors that stimulant treatment impact on nicotine dependence should be interpreted with caution, warranting future larger-sample, longer-term prospective studies inspecting the role of non-stimulant medications in modulating substance use disorder in ADHD.

1 Groenman AP, Oosterlaan J, Rommelse NNJ, Franke B, Greven CU, Hoekstra PJ, et al. Stimulant treatment for attention-deficit hyperactivity disorder and risk of developing substance use disorder. *Br J Psychiatry* 2013; **203**: 112–9.

2 Verma R, Balhara YP, Mathur S. Management of attention-deficit hyperactivity disorder. *J Pediatr Neurosci* 2011; **6**: 13–8.

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Authors' reply: In their letter, Verma and colleagues make the interesting point that possibly the age at first stimulant use × current age interaction effect found in our paper[1] might be influenced by our selection of patients. Including individuals with stimulant treatment duration longer than 12 months in our analyses, we found a protective effect of earlier age at first stimulant use on the development of substance use disorder (odds