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

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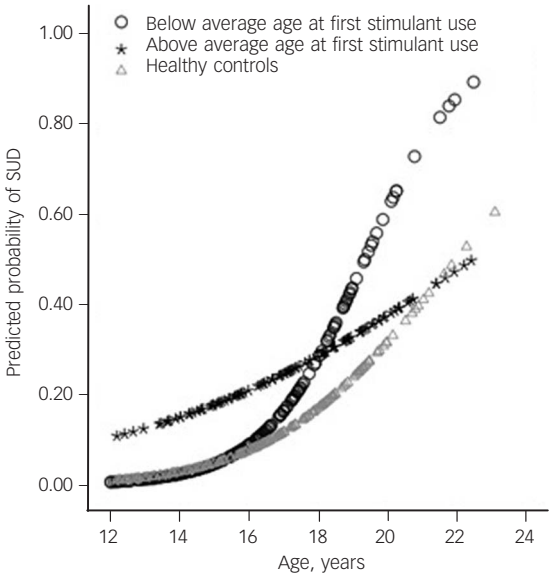

Fig. 1 Predicted probability of substance use disorders (SUDs).

Purely for illustrative purposes, we plotted the predicted probability of substance use disorder according to a generalised estimated equation model, that included age, gender and the interaction between age and age at first stimulant use. For healthy controls the model only includes age and gender. Below average age at first stimulant use: participants started before age 8.1 years; above average age at first stimulant use: participants started after age 8.1 years. Please note that predictions for healthy controls are not the product of the same model as prediction for stimulant groups.

ratio (OR) = 0.95, Wald $\chi^2 = 13.78$, $P < 0.001$). Verma *et al* are concerned that we excluded patients with shorter treatment durations. However, when we include all individuals who ever used stimulants, we find the same effect (OR = 0.95, Wald $\chi^2 = 11.89$, $P = 0.001$). Purely for illustrative purposes, we plotted the predicted probability of substance use disorder for the control group in Fig. 1. The figure shows that delay in the first age at stimulant use leads to marked increases in the probability of developing substance use disorder. In our article, we examined whether the effect of stimulant treatment depended on other factors (i.e. current use of stimulants, age at stimulant treatment initiation, age-adjusted duration of stimulant use and age-adjusted cumulative dosage), but found no other significant predictors than age at first stimulant use.

Verma and colleagues refer to a meta-analysis, but provide the wrong citation. Recently, a meta-analysis on this topic was published.[2] Here no difference was found between treated and untreated patients in risk of developing substance use disorder (including alcohol, marijuana, cocaine and non-specific drugs) and nicotine use. Unfortunately, in this meta-analysis specific moderator variables such as age at first stimulant use were not taken into account, probably because of the relatively low numbers of studies to date that include such variables.

We thank the authors for discovering the mistake in the table, 9% should have read 59%.

1 Groenman AP, Oosterlaan J, Rommelse NN, 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 Humphreys KL, Eng T, Lee SS. Stimulant medication and substance use outcomes; a meta-analysis. *JAMA Psychiatry* 2013; **70**: 740–9.

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Liaison services for older adults

Professor Sharpe's editorial summarises elegantly the latest developments in psychological medicine.[1] The economic evaluation of the liaison services that started with the evaluation of the Birmingham Raid Model has naturally progressed with the recent National Institute for Health Research Health Services and Delivery Research Programme on commissioning research grants for 'Organisation, quality and cost-effectiveness of psychiatric liaison services in acute settings'. This call was also accompanied by another one on 'Assessing alternatives to face-to-face contact with patients'. The outcomes of these two calls will undoubtedly bring a new wave of changes to our current liaison services that are already undergoing remodelling. The editorial argues that 'small' liaison subspecialties should 'join forces under a single banner' to provide 'flexible and shared service provision'. Liaison Services for Older Adults (LSOA) are among those that are numbered in the list of small subspecialties. Our analysis of the LSOA within our locality[2] and wider[3] confirms that the LSOAs appear to be the fastest growing liaison discipline. In Newcastle alone we witness a steady 10% yearly increase of older people referred to our service, with the overall numbers being very close to those of our Deliberate Self Harm (DSH) team (37% LSOA *v.* 39% DSH).[3] Those of us who already work in the newly integrated liaison services are under increasing pressure to become more generalist, shadow our DSH colleagues to 'broaden' our clinical experiences, while at the same time the suitability of referrals to our 'small' subspecialty is frequently scrutinised. And yet, the majority of hospital beds are occupied by older people who are physically compromised and cognitively impaired, who are either known to old age psychiatry services or are referred to the LSOA as a result of the Dementia Commissioning for Quality and Innovation (CQUIN). For many of them, our subspecialty would facilitate the diagnosis and initiate the treatment for their cognitive impairment, challenging behaviour and/or depression, and our expertise would aid the decision about their long-term needs and placement and enable/maintain that essential continuity of care that is currently failing them.[4,5] In addition, the LSOA medical expertise is not confined to our old age psychiatric knowledge, but many of us are also dual trained (e.g. family medicine, neurology) and/or hold diplomas in geriatric medicine.

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