Sensky *et al*[6] (non-significant) end-of-study results contribute to the findings, but the (significant) 9-month and 5-year follow-up results do not.[7]

Meta-analyses can be highly informative, but they are highly prone to bias.[8] Those with a 'washing machine' approach, such as this one (i.e. amalgamating different populations – from acute in-patients to chronic out-patients, from young people with a first episode of psychosis to older adults; different therapies – from 3 sessions of acceptance and commitment therapy to 18 months of weekly cognitive therapy; different modalities – groups or individual; different targets – from compliance with command hallucinations to emotional dysfunction), tell us very little about what works for whom. Unsurprisingly, the heterogeneity statistics were highly significant for all analyses, with I^2 being at 50% or above (i.e. representing 'substantial heterogeneity'), suggesting that there was too much heterogeneity to obtain meaningful pooled estimates, and that the necessary criteria for rendering a meta-analysis appropriate were not met.[9]

The field of CBTp has now progressed such that it is no longer appropriate to simply lump together psychosis patients assuming that clinical presentations are the same, that therapy is for the same problem, and that the type of CBT is the same. Other recent meta-analyses, which focus on treatment-resistant patients,[10] or on individually tailored, formulation-based CBT for hallucinations and delusions,[11] will be more informative to clinicians and researchers about the specific effects of CBTp.

To conclude, the reported analyses reflect an over-simplification of the complexities of psychosis and psychological interventions. The biggest challenges in psychological therapy trial methodology (and in clinical practice) are the quality of/adherence to the therapy delivered and the competence of the therapists, none of which was taken into account in this study. A more meaningful reading of the existing research is that the next steps are to investigate which patients benefit on which outcomes at which stages with which types of therapy, and how to ensure therapist competence (and availability).

Declaration of Interest

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Authors' reply: One of the founding principles of meta-analysis is to pool data from as many studies as possible.[1] Among other benefits this prevents studies being preselected for consideration on arbitrary grounds. It is difficult to imagine anything more arbitrary than restricting a meta-analysis of CBT for schizophrenia to studies that conform to some notional interpretation of the NICE guideline, as Peters seems to be suggesting, not to mention excluding any that were in Chinese.

Similarly, it would be wrong to exclude studies that used group CBT *a priori*. Here, though, it is entirely legitimate to examine this issue *post hoc*; that is, to ask whether use of group *v.* individual CBT significantly moderates effect size. Carrying out this analysis on our data reveals that the pooled effect sizes for both types of intervention were very similar in the meta-analysis of overall symptoms (effect size in 7 group studies −0.24 *v.* −0.23 in 24 individual studies; $Q = 0.006$; $P = 0.94$); for positive symptoms, group CBT had a non-significantly smaller effect size than individual CBT (effect size in 8 group studies −0.08 *v.* −0.25 in 23 individual studies; $Q = 1.73$; $P = 0.19$) (across both analyses, one study employed both group and individual CBT and three were rated as 'unclear'). This might or might not be considered evidence that group CBT is less effective than individual CBT, but what it does not mean is that inclusion of group studies in our original meta-analyses somehow acted to dilute the pooled estimate – the effect sizes for studies using individual CBT are similar or lower to those we reported for all studies combined (effect sizes were −0.33 for overall symptoms and −0.25 for positive symptoms).

With regard to some of the other points raised by Peters, our diagnostic criteria were broad and similar to those used by NICE, Wykes *et al* and the Cochrane Collaboration. We recognised the possibility that Acceptance and Commitment Therapy might be different from regular CBT and presented an analysis in the article excluding two studies using this[2,3] and another where CBT took the form predominantly of coping skills enhancement;[4] this did not materially affect the results. Peters expresses surprise over our decision to exclude studies that specifically targeted hallucinations from the meta-analysis of positive symptoms. As it happens, only three studies of hallucination-directed CBT also reported outcomes for positive symptoms. Adding the data from two of them[5,6] (data cannot be extracted from one study[7]) to the positive symptoms dataset makes no difference to the pooled effect size (−0.25; CI −0.36/−0.13).

Peters argues that there was too much heterogeneity among the results to obtain meaningful pooled estimates. In fact, the Cochrane Collaboration article she cites[8] recommends (a) not pooling data using meta-analysis, (b) investigating heterogeneity using subgroup analysis or meta-regression or (c) using a

random-effects model for meta-analysis, as this includes consideration of heterogeneity in the effect-size estimate. The authors also note that 'even though a random-effects model helps to consider heterogeneity, it does not remove it – heterogeneity still needs to be considered in interpreting the results'. We used a random-effects model and examined heterogeneity.

We would like to reiterate that for those who wish to examine for themselves other points of the type raised by Peters, a detailed database of the studies we included is available online (http://www.cbtinschizophrenia.com/).

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Borderline personality disorder and mood

Parker examined whether borderline personality disorder (BPD) is a bipolar or unipolar mood condition and concluded by suggesting that it is probably neither.[1] I would like to offer a supplementary interpretation of the literature; that is BPD is in large part a mood disorder but is not necessarily a bipolar or unipolar mood variant.

Borderline personality disorder is highly comorbid with bipolar disorder[2] and depression,[3] and those who develop bipolar disorder have early temperamental markers of emotional dysregulation.[4] Support that BPD is a mood disorder is also aligned with the fact that affective instability is a core feature of the syndrome. While under- investigated, there is emerging evidence that affect or mood instability, as opposed to mood episodes, might be the core feature of bipolar disorders.[5] The majority of patients with established bipolar disorder, even after symptomatic control continue to experience daily or weekly mood swings.[6] Further, the prevalence of mood instability and cyclothymic temperament is increased in unaffected bipolar probands[7] and it predicts functioning in those with bipolar disorder.[5] Mood instability is highly prevalent in unipolar depression[8] and independently links to suicidality and health-service use. Furthermore in BPD, affective instability is the least stable of the 'trait-like' features of the syndrome over 2 years.[9] Thus, all three disorders share mood instability as a clinical component and this all points to BPD, at least in part, being a disorder of mood.

However BPD does not exactly fit into the bipolar or depressive affect rubric, given that the affective shifts do not last long enough for either diagnosis. Detailed studies of the nature of affective instability in mood disorders and BPD using the same measurement methods are limited. However, as Parker states, there are differences. Those with bipolar disorder have greater levels of euthymia–elation and affect intensity. In BPD there are more shifts between anxiety, depression and euthymia–anger.[10] Negative emotionality is a critical feature of BPD but it is changeable, as is obvious to clinicians who have been charged with the care of people with BPD on in-patient wards.

Affect can be studied on the basis of intensity, frequency of shift, rapidity of rise-times and return to baseline, reactivity to psychosocial cues or whether endogenously driven, and the extent to which there is overdramatic expression.[11] To this could be added valence. Using this framework, BPD could be conceptualised as a disorder of mood in which affect changes are intense, frequent, rapid to occur, slow to dissipate and in which the valence of the mood state is typically negative incorporating depression, anxiety and anger. This pattern of difficulties although related to mood, do not appear to overlap to a significant extent with how depression or bipolar disorder might be described using the same affective framework. Though it is clear that terms such as 'intensity', 'frequency' and 'rapidity of rise' need to be better specified, experience-sampling methods analysing affective patterns in the three disorders might further illuminate this area and indeed the debate.

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Gordon Parker makes a powerful case against the hypothesis that borderline personality disorder is really a form of bipolar or unipolar disorder.[1] In so doing he is tilting at a windmill in whose