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Is cognitive behavioural therapy effective for individuals experiencing thought disorder?

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ABSTRACT

Various clinical guidelines recommend cognitive behavioural therapy (CBT) to treat psychosis without reference to patients' thought disorder. However, there is a risk that disorganized thinking hampers CBT. We tested the prediction that thought disorder would interfere with the effectiveness of CBT for hallucinations and delusions, compared to treatment as usual and supportive counselling, in secondary data from two large, single blind randomised controlled trials. We fitted latent growth curve models separately for the development of frequency and distress of symptoms. CBT was significantly more successful than counselling in reducing delusional frequency in the short term and hallucinatory distress at any point, even in those with relatively high thought disorder. We found little evidence that clinicians should restrict CBT in this subgroup of patients. Nevertheless, the findings highlight the importance of effective initial treatment of thought disorder in maximising the benefit of CBT for psychosis, particularly for reducing distress from hallucinations.

1. Introduction

Various national clinical guidelines recommend cognitive behavioural therapy (CBT) for patients experiencing psychosis (e.g. Australia & New Zealand, Galletly et al., 2016; Canada, Norman et al., 2017; United Kingdom, NICE 2014). In CBT, the therapist and service user collaboratively work towards mutually defined goals, and explore how individuals' thoughts, feelings and behaviours might maintain their difficulties over time (Morrison et al., 2004). Meta-analyses have shown modest to small effects when exploring whether CBT is effective for treating psychotic disorders (Jauhar et al., 2014; Pilling et al., 2002; Turner et al., 2014; Wykes et al., 2008; Zimmermann et al., 2005). It is possible that there exist subgroups of patients, with certain symptom profiles, where CBT is less effective (Pickles and Croudace, 2010). This might include patients with high levels of hopelessness, low insight, or more severe psychosis (Birchwood et al., 2018; Brabban et al., 2009; Garety et al., 1997). It might also be true of individuals experiencing thought disorder.

Thought disorder refers to disorganised thinking, evidenced by disorganised speech. Originally described as a 'loosening of associations' (Bleuler, 1911), it likely includes a variety of cognitive and linguistic difficulties, such as tangentially, illogicality, thought block, concrete thinking and pressurised communication. Thought disorder is common in psychosis, with estimates of prevalence ranging from 5% to 91% depending on how it is defined and assessed (Roche et al., 2014). It is associated with adverse outcomes, including worse illness course (Wilcox et al., 2012), impaired functioning (Bowie and Harvie, 2008), and poor quality of life (Tan et al., 2014). Thought disorder can present as stable over time, but typically increases during periods of acute psychosis (Docherty et al., 2003).

There exists scepticism around offering CBT to individuals experiencing thought disorder, likely due to the view that communication difficulties will impede learning through verbal dialogue. In support of this view, there is evidence that higher levels of thought dsiorder are associated with lower clinician ratings of therapeutic alliance (Cavelti et al., 2016; Lysaker et al., 2011), which is a crucial factor in the efficacy of CBT and other therapies (Goldsmith et al., 2015). However, researchers have seldom investigated or verified the direct effect of thought disorder on the efficacy of CBT for psychosis. Thomas et al. (2011) found no significant relationship between thought disorder and improvement in auditory hallucinations under CBT, but the study was small, uncontrolled, and with no long-term follow up.

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Further research based upon controlled trials would allow for a more definitive answer on whether CBT is a viable treatment option in clients experiencing thought disorder. This is turn could lead to better allocation and prioritisation of treatment. This article contains re-analysis of data from two randomised controlled trials evaluating CBT for psychosis. The aim was to investigate whether thought disorder interfered with the effectiveness of CBT for key therapeutic outcomes, namely hallucinations and delusions. We hypothesised that thought disorder would reduce the effectiveness of CBT for psychosis compared to treatment as usual treatment as usual and supportive counselling.

2. Materials and methods

2.1. Participants

We used data from two randomized controlled trials of CBT conducted in the UK: The Study of Cognitive Reality Alignment Therapy in Early Schizophrenia (SOCRATES; Lewis et al., 2002, Tarrier et al., 2004), and The Assessment of Cognitive Therapy Instead of Neuroleptics (ACTION; Morrison et al., 2014). We chose these because they were both controlled trials comparing CBT with treatment as usual, using the same measures of symptoms and thought disorder, with long-term follow-ups. Many trials of CBT for psychosis exclude patients with high levels of thought disorder precluding their use for such analysis.

SOCRATES was a multicentre prospective trial of CBT or supportive counselling compared to treatment as usual. Participants were 308 patients diagnosed with schizophrenia-spectrum disorders, who had been hospitalised for a recent, acute, first or second psychotic episode. Patients were assessed at study baseline before randomization to treatment and then at 1.5, 3, 9 and 18 months. Tarrier et al. (2004) provide full details of the study and outcomes at the 18-month follow up point.

ACTION was a multicentre randomized controlled trial of CBT versus treatment as usual for 74 participants who met diagnostic criteria for schizophrenia spectrum disorders or entry criteria for an early intervention for psychosis service. All participants had chosen not to take antipsychotic drugs for at least 6 months prior to the study. Assessments were made at pre-randomisation baseline, then at 3, 6, 9, 12, 15 and 18 months. Morrison et al. (2014) provide further details.

2.2. Measures

The outcome measures were the frequency and distress scores for the paranoid delusions and auditory hallucinations scales of PSYRATS, a well-validated assessment tool for common positive psychotic symptoms (Haddock et al., 1999). Scores on both scales ranged from 0 (symptom absent/no distress) to 4 (continuous symptom experience/maximum distress). A score of 0 on the frequency scale necessarily resulted in a missing response for the associated distress scale.

The key predictor variable was thought disorder, which was based on clinician / researcher ratings using the Positive And Negative Syndrome Scale (PANSS; Kay et al., 1987). We operationalized thought disorder scores based upon psychometric analysis in Drake et al. (2003), which comprised the average of responses to the following PANSS items: P2 (conceptual disorganization), P4 (excitement), N5 (Difficulty in abstract thinking), and G11 (poor attention). These items loaded consistently onto the same principal component at each stage of follow-up of the SOCRATES study and constituted a principal component of the change scores across follow-up (Drake et al., 2003). Scores could range from 1 (absent) to 7 (extreme). Chronbach's alphas were above 0.7 in all waves.

The other predictor variables were as follows:

Treatment: Participants who received CBT were compared with those allocated to treatment as usual in both SOCRATES and ACTION datasets, and with those allocated to supportive counselling in SOCRATES.

Data collection site: Participants recruited in Manchester were compared to those recruited in other centres; with those from Nottingham and from Liverpool in the SOCRATES dataset, and from Newcastle in the ACTION dataset.

Demographics: Variables for male sex and non-white ethnicity were compared with females and white ethnicity, respectively; we also controlled for age in years at baseline.

Duration of untreated psychosis: The natural logarithm of the Duration (in months) of Untreated Psychosis was included due to the possible influence on long-term symptomatic outcomes (Marshall et al., 2005)

First episode: For the SOCRATES data only, we compared patients who had experienced more than one psychotic episode with those experiencing their first episode (first episode status was not available in the ACTION dataset).

We also included the interaction between treatment and thought disorder, to capture the moderating effect of thought disorder on treatment efficacy. All continuous predictors (thought disorder, baseline symptoms, age and log duration of untreated psychosis) were mean-centred. Age, sex and phase of illness are all well replicated predictors of outcome in models adjusting for baseline symptom severity. Ethnicity is less clear as an independent factor, but along with other demographic variables acted as a proxy for important cultural and personal variables that had the possibility of affecting the therapy process (e.g. fluency in English, conceptual model of psychosis).

2.3. Analysis

2.3.1. Statistical models

We modelled the development of the symptom outcomes using latent growth curve models (McArdle and Epstein, 1987). Separately for each symptom, we fitted parallel growth curves for the longitudinal development of frequency and distress, with latent intercepts and slopes for each. The latent intercept represented a participant's frequency or distress score at the first post-randomization assessment and the latent slope represented the linear change in symptoms from then until the final assessment 18 months later. The latent intercepts and slopes were regressed upon two nested sets of predictors, set 1 and set 2. Set 1 comprised the symptom score at baseline (i.e. pre-randomisation), age, log duration of untreated psychosis, and dummy variables for treatment, site, sex, non-white ethnicity and (SOCRATES only) whether this was the participant's first psychotic episode. Set 2 encompassed the first set plus thought disorder at baseline and the interaction between thought disorder and treatment. The second set therefore represented the additional effect that thought disorder had on the treatment-specific development of symptoms over time.

Fig. 1 shows a path diagram for the general model structure. The boxes to the left represent the observed symptom frequency scores at the post-randomisation assessments (four assessments are shown here, corresponding to the SOCRATES study; in the ACTION study there were six). To the right the boxes represent symptom distress scores, which were observed only for participants with non-zero symptom frequency. The circles represent the latent intercept and slope factors, which are indicated by the observed frequency and distress scores. Correlations among the latent growth factors are not shown to reduce clutter. The loadings for the intercept factors were fixed to one and each loading for the slope factor was fixed to its corresponding number of months since the first post-baseline assessment. Completing the picture are the boxes in the centre, representing the predictors of the latent growth intercepts and slopes; the boxes with faint outlines are the predictors in set 1, with bold outlines the predictors in set 2.

2.3.2. Estimation

The distributions of the delusions scores were positively skewed so we estimated the models using a robust maximum likelihood estimator that compensates for non-normality and heteroscedasticity (the Huber-

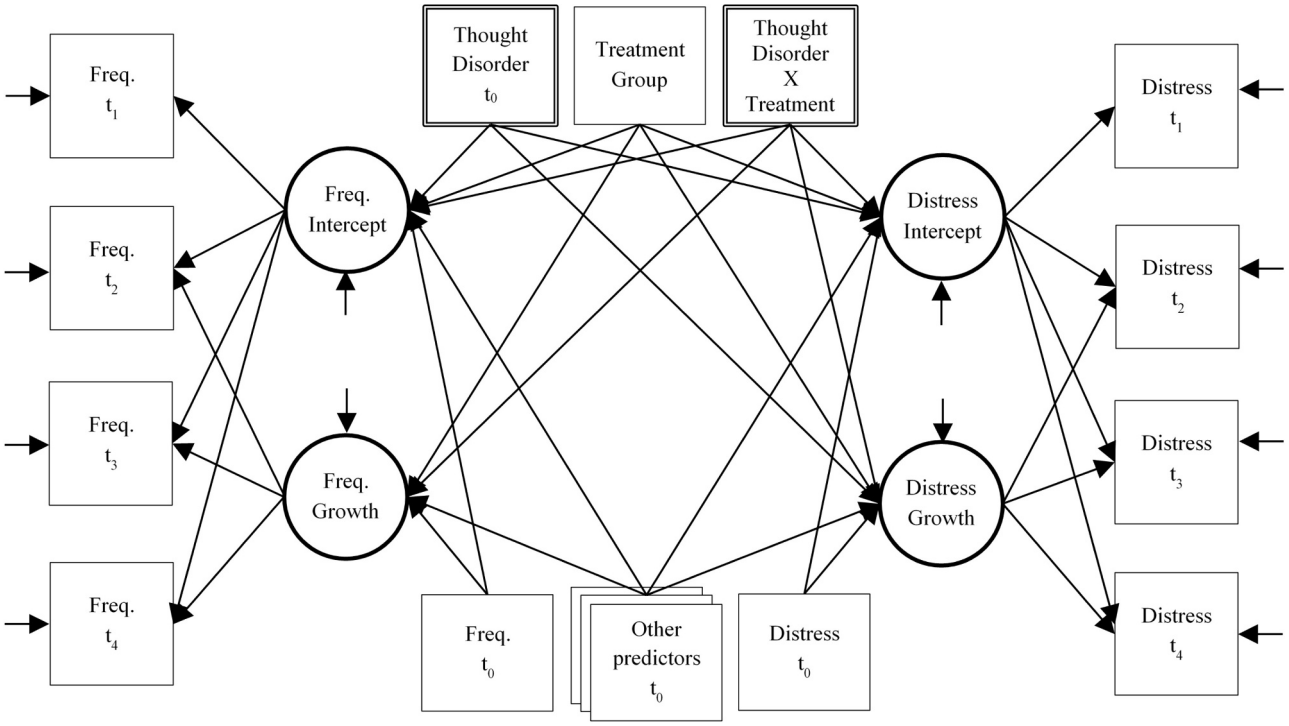

Fig. 1. Path diagram for the general model structure. The boxes to the left represent the observed symptom frequency scores at the post-randomisation assessments, those on the right represent observed distress scores. (Four assessments are shown here, corresponding to the SOCRATES study; in the ACTION study there were six). Circles represent the latent intercept and slope factors, which were regressed on key predictor variables (boxes at top-centre) and other covariates (boxes at bottom centre). Note: t_0 = baseline assessment; t_1, t_2, etc., indicate subsequent, post-randomization assessments (corresponding to 1.5, 3, 9 and 18 months for the SOCRATES study). All latent growth factors were allowed to correlate freely, but the arrows are not shown to reduce clutter.

White 'sandwich' estimator; White, 1980). The hallucination scores were severely non-normal because many participants had no hallucinations, so following Lewis et al. (2002) we dichotomized the hallucination outcomes into participants reporting no vs. any frequency or distress; we then modelled them as binary outcomes.

We fitted the models initially as Generalized Structural Equation Models (GSEMs) in Mplus 7.3 (Muthen and Muthen, 1998–2012), treating delusions as continuous measures and hallucinations as binary measures modelled using a logistic link function. Model estimation was by Monte Carlo numerical integration with 5000 integration points (necessary because of the severe computational burden of fitting the logistic models with four latent dimensions; see Asparouhov and Muthen, 2012). Because of the complexity of the models and the small sample size of the ACTION data, we also respecified[1] and fitted the models as hierarchical generalized linear mixed effects models (i.e. generalized multilevel models; GMLMs) using the `mixed` (for delusions) and `melogit` (for hallucinations) procedures in Stata 14 (StataCorp, 2015). These were fitted as a robustness check and sensitivity analysis on the GSEM results.

2.3.3. Inference

We compared the set 1 and set 2 models using likelihood ratio tests for both the GSEM and GMLM models. Outcomes showing significant differences using both modelling approaches were then evaluated in detail. This was an exploratory analysis looking for evidence of potential harm or reduced effectiveness of CBT when coupled with thought disorder. The risks associated with a type II error were therefore higher than usual, and to reflect this we set our alpha value for statistical significance at 0.1.

3. Results

Table 1 shows the SOCRATES participants were predominately white males experiencing their first psychotic episode. Average symptom levels were quite high at baseline, as would be expected for this acute sample. The ACTION participants were more evenly split by sex, with longer average duration of untreated psychosis, but generally lower symptom frequency scores. Our sample sizes were slightly smaller than the number of recruited participants because of missing covariate data.

In estimating the GSEM models, the residual variances for the delusion distress score at 18 months and the variance of the delusion distress latent slope factor were both very small and inadmissible (negative). Following accepted practice we fixed them to zero to produce an acceptable model (Chen et al., 2001). For the GMLM models, the full model for hallucination responses was too demanding for our computing facilities to estimate, even using the Laplace approximation to the standard Gauss-Hermite adaptive quadrature; we therefore divided it into separate models for the hallucination frequency and distress. Table 2 shows the likelihood ratio tests comparing the models with set 1 and set 2 predictors.

The set 2 models were a significant improvement for both the GSEM and GMLM approaches for the SOCRATES delusions outcomes. The SOCRATES Hallucination GSEM model difference was also significant, but this difference was reflected only in the distress outcomes for the GMLM. Neither of the ACTION symptom outcomes had significant differences between sets 1 and 2 for both the GSEM and GMLM approaches. We therefore went on to examine the GSEM results for the set 2 models for SOCRATES delusion frequency and distress, and hallucination distress.

[1] SEMs and multilevel models (MLMs) are distinct but comparable approaches to fitting growth curve models (Chou, Bentler, & Pentz, 1998); using both gave us a useful sensitivity analysis for the stability of our results to the different assumptions underlying model specification and approaches to model estimation of the two methods.

Table 1
Descriptive statistics for SOCRATES data (upper panel, $N = 256$) and ACTION data (lower panel, $N = 65$).

Socrates Predictor	Mean/%	SD	Symptom t_0	Mean	SD
Centre			Thought Disorder	2.95	1.03
Manchester	36.3%	-	Delusions		
Liverpool	33.6%	-	Frequency	3.06	0.78
Nottingham	30.1%	-	Distress	2.65	0.94
Male	70.0%	-	Hallucinations		
Age (years)	30.0	10.7	Frequency	2.57	0.87
Non-White	11.6%	-	Distress	2.54	1.04
First Episode	84.6%	-			
DUP (ln[months])	2.60	1.32			

ACTION Predictor	Mean/%	SD	Symptom t_0	Mean	SD
Centre			Thought Disorder	1.75	0.64
Manchester	43.4%	-	Delusions		
Newcastle	56.6%	-	Frequency	2.58	1.25
Male	49.1%	-	Distress	2.68	1.17
Age (years)	31.2	12.1	Hallucinations		
Non-White	15.1%	-	Frequency	1.66	1.40
DUP (ln[months])	3.56	1.68	Distress	2.59	1.19

Note: DUP = Duration of Untreated Psychosis; t_0 = baseline assessment; Symptoms t_0 are on a 1–7 scale.

Table 3 shows the estimates for the set 2 predictors of the latent growth factors for these models. The most clear and consistent finding was that there were no significant differences in delusion frequency or distress between CBT and treatment as usual, either at the 1.5 month post-randomisation point (i.e. on the growth intercept) or in the subsequent linear change up to the 18 month point (i.e. on the growth slope), regardless of the level of thought disorder (i.e. no significant thought disorder$_{t0}$*treatment as usual interactions).

There were significant differences between CBT and supportive counselling in delusion frequency, which are illustrated in Fig. 2. At the 1.5 month point delusion frequency was much higher with supportive counselling compared to CBT, but this difference was reduced somewhat with higher levels of thought disorder. The subsequent rate of reduction in delusion frequency to the 18 month point was more rapid for supportive counselling compared to CBT, but this difference in the rate of improvement was slower the higher the thought disorder.

The size of the effects of thought disorder on delusions were small for both the intercept and slope, with an R-square change in the latent growth factors from set 1 to set 2 of 4.2% and 7.2% respectively.

The lower panel in Table 3 shows the results for hallucination distress. There were again no significant differences between CBT and treatment as usual. There were significant effects of CBT vs. supportive counselling at the 1.5 month point, but no significant effects for subsequent change till 18 months. At 1.5 months, participants allocated to

Table 2
Comparisons for models with set 1 vs set 2 predictors.

Model type	SOCRATESDelusions	SOCRATESHallucinations	ACTIONDelusions	ACTIONHallucinations
GSEM, Likelihood-ratio chi-square, set 1 vs set 2	**32.030**df = 12 ***p*** **< 0.001**	**20.116** df = 12**p** **= 0.065**	**8.828** df = 4 ***p*** **= 0.066**	4.932 df = 4p = 0.294
GMLM, Likelihood-ratio chi-square, Set 1 vs set 2	**20.819** df = 12**p** **= 0.053**	Frequency: 5.103 df = 6,*p* = 0.954 Distress: **10.411**df = 6 ***p*** **= 0.034**	1.818 df = 4 *p* = 0.403	3.349 df = 4 *p* = 0.501

Note: GSEM = Generalized Structural Equation Models; GMLM = Generalized MultiLevel Models. **Bold** = $p < 0.1$. Set 1 predictors were site, sex, age, ethnicity, duration of untreated psychosis and first episode status; set 2 added TD, treatment, and TD*treatment interactions. The SOCRATES Hallucinations GMLM model was split into separate models by symptom type because of computational limitations fitting the full model.

Table 3
GSEM results for SOCRATES delusions and hallucinations.

Predictor	Delusion Frequency[a] Growth Intercept Est	SE	p	Growth Slope Est	SE	p	Delusion Distress[a] Growth Intercept Est	SE	p	Growth Slope Est	SE	p
TDt_0	0.155	0.107	0.147	−0.007	0.009	0.415	0.143	0.125	0.250	0.014	0.011	0.191
Treatment (vs. CBT)												
TAU	0.412	0.444	0.353	0.010	0.040	0.808	0.217	0.454	0.633	0.006	0.046	0.888
COU	**1.013**	**0.437**	**0.020**	**−0.071**	**0.038**	**0.064**	0.451	0.503	0.370	0.022	0.051	0.660
TDt_0*TAU	−0.047	0.147	0.748	−0.001	0.013	0.930	−0.063	0.150	0.673	−0.003	0.015	0.861
TDt_0*COU	**−0.367**	**0.141**	**0.009**	**0.028**	**0.013**	**0.027**	−0.207	0.173	0.231	−0.005	0.017	0.793
R^2 Change,set 2 – set 1	0.042			0.074			0.031			–		

Predictor	Hallucination Distress[b] Growth Intercept Est	SE	p	Growth Slope Est	SE	p
TDt_0	1.587	0.876	0.07	0.215	0.149	0.149
Treatment (vs. CBT)						
TAU	0.971	3.247	0.765	0.443	0.448	0.322
COU	**12.976**	**5.393**	**0.016**	−0.361	0.5	0.471
TDt_0*TAU	−0.418	1.054	0.691	−0.307	0.196	0.118
TDt_0*COU	**−4.150**	**1.648**	**0.012**	0.004	0.154	0.982
R^2 change, set 2 – set 1	0.145			0.007		

Note: N:.
Bold = $p < 0.1$. All models controlled for site, sex, age, ethnicity, duration of untreated psychosis and first episode status.
[a] = 256.
[b] = 155; TDt_0 = baseline thought disorder; TAU = Treatment As Usual; COU = counselling.

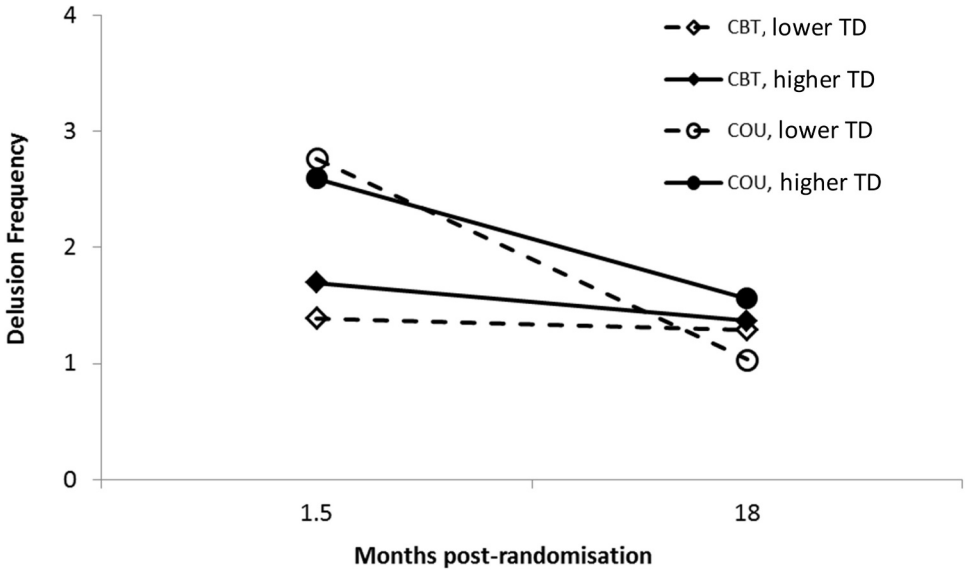

Fig. 2. SOCTRATES, model-predicted delusion frequency over time by treatment (CBT and COU) and TD. Showing linear change from 1.5 months to 18 months post-randomization.
Note: CBT = Cognitive Behavioural Therapy; COU = Counselling; TD = Thought Disorder at baseline, evaluated at 1 scale point above ("higher") and below ("lower") the average. Values conditional on the other covariates, evaluated at baseline or average value. .

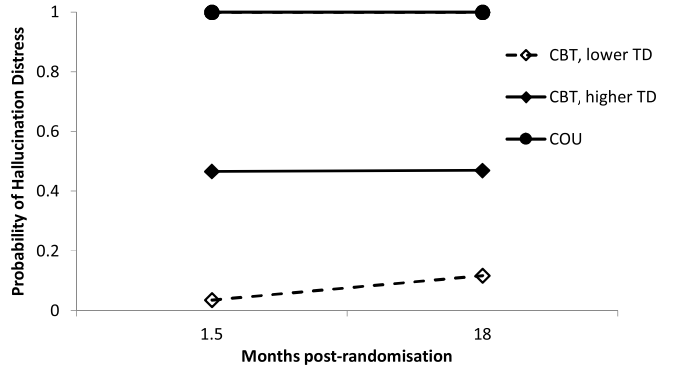

Fig. 3. SOCRATES, model-predicted hallucination distress probability over time by treatment (CBT and COU) and TD. Showing linear change from 1.5 months to 18 months post-randomization.
Note: CBT = Cognitive Behavioural Therapy; COU = Counselling; TD = Thought Disorder at baseline, evaluated at 1 point above ("higher") and below ("lower") the average. Only one line is shown for COU because the outcome did not vary by TD.

supportive counselling had significantly higher probability of hallucinations (the very high logit coefficient of nearly 13 corresponding to a near-certainty of reporting hallucinations), but the difference with CBT was smaller with increasing thought disorder. Fig. 3 illustrates these effects, showing that hallucinations were rare for participants with low thought disorder allocated to CBT, common for those with high thought disorder in the CBT group, and near certain for those allocated to supportive counselling regardless of thought disorder levels. The differences in the intercept attributable to thought disorder were of moderate size (R-square of 14.5%).

4. Discussion

4.1. Main findings

Significant findings only emerged from the larger SOCRATES dataset. Delusional frequency appeared to reduce significantly more after 6 weeks in the CBT than supportive counselling group, with a slight but significant negative impact of thought disorder; but as frequency reduced towards eighteen months initial thought disorder predicted less improvement whatever the therapy (Fig. 2). CBT produced significantly better, sustained reductions in distress from hallucinations than supportive counselling. Distress seemed worse after counselling even in those with greater thought disorder (Fig. 3), who benefitted significantly less from CBT than those without. Although CBT did not predict significantly better 6-week outcomes or changes over follow-up than treatment as usual, the trends in its favour were consistent with the original trial report's description of significant efficacy for therapy against total scores after 18 months (Tarrier et al., 2004).

4.2. Strengths and limitations

We have more trust in the relative differences found between treatments compared to the absolute differences found within treatments, because of the possibility that symptom measures and thought disorder ratings were endogenous (i.e. reflecting the same illness process). We tried to guard against this possibility by using different measures for symptoms (PSYRATS) and thought disorder (PANSS), the former being based on interpretation of self-ratings and the latter wholly observer-rated. Furthermore, the relative effects across treatments were protected by randomisation. This ensured that there would be no systematic differences in thought disorder, which was measured pre-randomisation, between the groups, and that this exogeneity across groups would also be carried over to the key interaction effects of thought disorder by treatment (Emsley et al., 2010).

The analysis used an unusually relaxed criterion for statistical significance because we wanted to have a lower than usual chance that we would miss potential evidence for the harmful effect of thought disorder on the efficacy of CBT for psychosis. Despite this, we found very little evidence for adverse effects of thought disorder; essentially no evidence for delusions and only limited evidence for hallucinations, where higher levels of thought disorder was associated with higher probability of hallucination within the CBT group. This was despite the fact that our study had the statistical power to detect reasonably small differences using the SOCRATES data (R-square of 4.2%). The smaller sample size of the ACTION data meant that our analysis would have been able to detect only larger differences (R-square > 15%). Considering the differential effects of thought disorder across treatments, we found no strong evidence that thought disorder is more problematic for CBT than for treatment as usual or supportive counselling.

Although we found essentially the same pattern of results when using the different modelling approaches in the SOCRATES data, the differences attributable to the effects of thought disorder in the ACTION data were highly sensitive to the choice of using GSEM or GMLM. We

believe this was most likely due to the much smaller sample size of the ACTION study compared to the SOCRATES study and the effect of this on the slightly different modelling assumptions and estimation methods.

We used a measure of thought disorder derived from items on the PANSS, rather than a dedicated and validated measure of thought disorder. Thought disorder is a multifaceted construct (Andreasen, 1979) and is it likely that different subtypes could differentially influence the effectiveness of CBT, as well as in those with more severe thought disorder than found in our sample.

The delivery of therapy in SOCRATES was relatively atypical in that it was front-heavy; patients received the majority of sessions at the start of the intervention. A limitation of the current analysis was that it was restricted to two datasets that were available to the authors, which may not have been representative of randomised controlled trials more generally. In the future, it will be important to evaluate the impact of thought disorder on CBT in other datasets, including a purpose-designed trial replicating how therapy could be delivered in clinical practice. It would also be useful to see whether CBT could be adapted to specifically target thought disorder, rather than focusing primarily on hallucinations and delusions (Palmier-Claus et al., 2017).

5. Conclusion

To the best of our knowledge, there is little research exploring the impact of thought disorder on the effectiveness of CBT for delusions and distress from hallucinations. Our results are novel in that they provide tentative evidence that CBT is not harmful for patients experiencing thought disorder. The UK's NICE's guidelines suggest that clinicians offer CBT to all patients with psychosis as a first line treatment. We found no compelling evidence to suggest that NICE should revise these recommendations to exclude patients experiencing thought disorder; CBT was significantly more successful than counselling in reducing delusional frequency in the short term and hallucinatory distress at any point, even in those with relatively high thought disorder. Nevertheless, these findings underscore the importance of effective initial treatment of thought disorder in maximising the benefit of CBT for psychosis, particularly for reducing distress from hallucinations.

Declaration of Competing Interest

None arising from this research.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psychres.2020.112806.

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