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Metacognitive Therapy in Treatment-Resistant Psychosis: A Multiple-Baseline Study

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Background: More effective psychological treatments for psychosis are required. Case series data and pilot trials suggest metacognitive therapy (MCT) is a promising treatment for anxiety and depression. Other research has found negative metacognitive beliefs and thought-control strategies may be involved in the development and maintenance of hallucinations and delusions. The potential of MCT in treating psychosis has yet to be investigated. **Aims:** Our aim was to find out whether a short number of MCT sessions would be associated with clinically significant and sustained improvements in delusions, hallucinations, anxiety, depression and subjective recovery in patients with treatment-resistant long-standing psychosis. **Method:** Three consecutively referred patients, each with a diagnosis of paranoid schizophrenia and continuing symptoms, completed a series of multiple baseline assessments. Each then received between 11 and 13 sessions of MCT and completed regular assessments of progress, during therapy, post-therapy and at 3-month follow-up. **Results:** Two out of 3 participants achieved clinically significant reductions across a range of symptom-based outcomes at end-of-therapy. Improvement was sustained at 3-month follow-up for one participant. **Conclusions:** Our study demonstrates the feasibility of using MCT with people with medication-resistant psychosis. MCT was acceptable to the participants and associated with meaningful change. Some modifications may be required for this population, after which a controlled trial may be warranted.

Keywords: Metacognitive therapy, psychosis, schizophrenia, hallucinations, delusions.

Introduction

The last 30 years have seen many developments in the treatment of psychosis (NICE, 2009). However, the extent to which the main pharmacological intervention, antipsychotic medication, provides a clinically meaningful benefit above placebo has recently been questioned (Lepping, Sambhi, Whittington, Lane and Poole, 2011; Leucht, Arbter, Engel, Kissling and Davis, 2009; Rattehalli, Jayaram and Smith, 2010), while very high rates of

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drop-out from short and long-term clinical trials suggest low acceptability (Leucht, Arbter et al., 2009; Lieberman et al., 2005). According to some estimates, between one-fifth and one-third of patients will not have a good response to antipsychotic medication (NICE, 2009).

Psychosocial interventions appear to be highly acceptable to most service-users with psychosis (Villeneuve, Potvin, Lesage and Nicole, 2010; Warner, Mariathasan, Lawton-Smith and Samele, 2006). A recent review of one of these treatments, cognitive behavioural therapy (CBT), found it was moderately effective at reducing positive and negative symptoms, improving functioning and improving mood (Wykes, Steel, Everitt and Tarrier, 2008), although there is not complete consensus on this (Lynch, Laws and McKenna, 2010; Newton-Howes and Wood, 2011). Unfortunately, the effects were smaller in rigorous and adequately-powered trials that attempted to control for non-specific aspects of therapy (e.g. increased social interaction). However, as outlined elsewhere (Tai and Turkington, 2009), CBT for psychosis is evolving and new treatments are in development. Preliminary efficacy data from a multiple-baseline case-series study of one of these new treatments, metacognitive therapy (MCT), will be reported here.

Metacognition in psychological disorder

Traditional CBT assumes that emotional distress is strongly influenced by the way a person makes sense of their experiences (Beck, 1976). Unhelpful or distorted beliefs about self, others and the world are thought to be maintained by counterproductive attempts at coping, such as avoidance. Reductions in distress are thought to follow the development of new, more helpful beliefs. This is achieved through collaborative examination of the evidence, which is facilitated by behavioural change.

MCT, on the other hand, assumes emotional distress is related to activation of unhelpful forms of information-processing such as worry and rumination, "metacognitive" beliefs about the usefulness, controllability and danger of these processes, and counterproductive strategies of thought-control (Wells, 2009) as outlined in the S-REF model (Wells and Matthews, 1994). Negative metacognitive beliefs (e.g. "My worry is uncontrollable") are thought to play a central role in the onset and maintenance of many different emotional disorders and problems, including generalized anxiety (GAD) (Wells, 1995), social anxiety (Clark and Wells, 1995), post-traumatic stress disorder (PTSD) (Wells and Sembi, 2004), obsessive-compulsive disorder (OCD) (Fisher and Wells, 2005), health anxiety (Bouman and Meijer, 1999), depression (Papageorgiou and Wells, 2001) and anger (Simpson and Papageorgiou, 2003). Metacognition has also been linked to the development of psychotic symptoms, specifically hallucinations (Baker and Morrison, 1998) and delusions (Freeman and Garety, 1999; Laroi and van der Linden, 2005; Morrison, 2001; Morrison, French and Wells, 2007; Morrison et al., 2011; Startup, Freeman and Garety, 2007). Recent work has suggested metacognitive beliefs may be more important in understanding the distress associated with hallucinations, rather than hallucination occurrence per se (Varese and Bentall, 2011).

Rather than challenging the validity of thoughts and beliefs about the self and world MCT encourages a person to develop a detached awareness of their thoughts with concomitant control of worry/rumination and unhelpful attentional strategies. This facilitates: (1) appropriate executive control over maladaptive processes and (2) challenging of metacognitive beliefs that worry and rumination are uncontrollable or dangerous. This, in turn, is thought to lead to a significant reduction in distress (Wells, 2008). Early pilot trials

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and case studies suggest MCT (or components of MCT) is a promising treatment for OCD (Fisher and Wells, 2008), PTSD (Wells et al., 2008), GAD (Wells and King, 2006) treatment-resistant depression (Wells et al., 2009) and auditory and visual hallucinations in psychosis (Hutton, Morrison and Taylor, 2012; Valmaggia, Bouman and Schuurman, 2007). Results from controlled trials suggest MCT may be superior to exposure therapy in treating PTSD (Proctor, 2008), and superior to relaxation therapy or CBT in treating GAD (van der Heiden, Muris and van der Molen, 2011; Wells et al., 2010). In psychosis, a 4-session MCT-informed intervention, focused on reducing worry and challenging metacognitions, was found to be superior to treatment as usual in reducing persecutory delusions (Foster, Startup, Potts and Freeman, 2010).

A metacognitive approach to psychosis

Although existing cognitive models clearly identify metacognition and worry as important factors in psychosis (Garety, Kuipers, Fowler, Freeman and Bebbington, 2001; Morrison, 2001), they also incorporate many non-metacognitive components, such as core beliefs (i.e. about self, others and the world), a focus on the content of appraisals, self-esteem and safety behaviours including avoidance. It remains to be seen whether a "pure" metacognitive model and treatment approach is of value in psychosis. We will now describe the key features of the model used in the current study.

We adapted Wells' metacognitive model of GAD (Wells, 1995) in a similar way to that proposed by Morrison and colleagues (Morrison et al., 2011). To maximize fidelity with the metacognitive model (Wells, 2009) we conceptualized negative thoughts rather than psychotic experiences as the trigger for the development of distress (see Figure 1 for an example of such a formulation). This reflects the fact that many people have psychotic-like experiences but are not distressed by them, and are not receiving help from services (Pechey and Halligan, 2011, in press). In the metacognitive model, distress is a consequence of sustained processing in the form of worry and rumination and counterproductive mental regulation strategies.

Psychotic experiences were not conceptualized as direct triggers for the development of further worry; rather the trigger was instead an initial worrying thought, such as "Oh no, I have to leave the house" or "God is talking to me again". If an individual held positive metacognitive beliefs about the usefulness of worry (e.g. "Worrying keeps me safe", "If I analyse my problems, I will find answers"), then this activated a chain of further worries or rumination which, as in GAD, varied in content depending on the initial trigger. Unusual thoughts, commonly viewed as delusions or paranoia, were classified as the content or output of worry, which equates with Wells' concept of Type 1 worry in his GAD model. Whilst this worry contributes to distress, negative metacognitive beliefs and "worry about worry" (Type 2 worry) contribute to this becoming persistent and intense. Type 2 worries such as "My worry is uncontrollable" and "I'll go mad if I can't stop worrying" give rise to a further imminent sense of threat and interfere with attempts at regulating cognition.

Type 2 worries are thought to be closely linked to pre-existing negative metacognitive beliefs about worry. Typical negative metacognitive beliefs include "Thinking too much will drive me mad" and "Worry is uncontrollable". Activation of Type 2 worries leads to an escalation of anxiety symptoms, which are then misinterpreted as further evidence that worry is uncontrollable or harmful (Wells, 2009).

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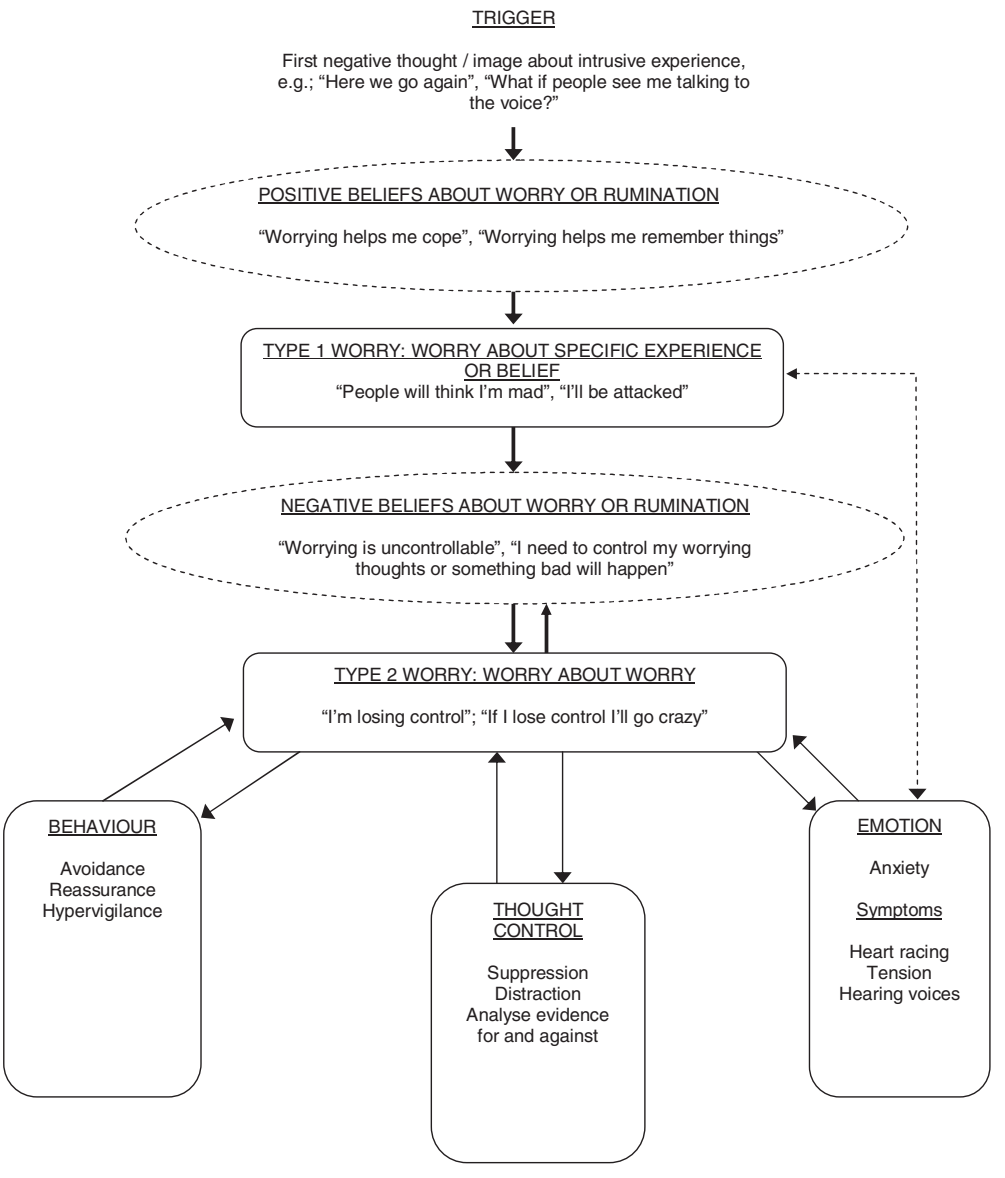


Figure 1. A metacognitive case formulation

Negative metacognitions about worry are also thought to be maintained by counterproductive attempts at thought-control (Wells, 2009). Thought suppression, a typical thought-control strategy in psychosis (Morrison and Wells, 2000), can lead to a paradoxical increase in the frequency of intrusive thoughts and/or associated distress (Lin and Wicker, 2007; Najmi, Riemann and Wegner, 2009; Wegner, Schneider, Carter and White, 1987). Other

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strategies such as reassurance, distraction and avoidance all prevent an individual discovering they can control their thoughts by internal means, while evidential analysis drains resources required for adequate control of cognition and attention (Wells, 2009). Some people may also engage in self-attacking or self-harm as a way of controlling thoughts, thus heightening their distress (Morrison and Wells, 2000). Some may consume alcohol or other substances. Most thought control strategies prevent an individual finding out that worry is not dangerous and does not need to be controlled.

The pattern of worry and rumination is also accompanied by changes in the direction of attention in which individuals begin to focus on potential sources of threat (Wells, 2009). Overall the pattern of worry/rumination, threat monitoring and counterproductive self-regulation strategies is termed the Cognitive Attentional Syndrome (CAS) (Wells and Matthews, 1994). MCT aims to reduce the likelihood of CAS activation by modifying metacognitive control strategies and restructuring positive and negative metacognitive beliefs (Wells, 2009).

The aim of the current study was to conduct a case series of MCT with people meeting criteria for treatment-resistant schizophrenia. The goal was to address a preliminary question: Can a brief course of MCT work in schizophrenia? In order to test this we selected severe, complex and treatment resistant cases to provide rigour. Our main hypothesis was that MCT could be associated with, at end-of-therapy and 3-month follow-up, a clinically significant reduction in the severity of both delusions and hallucinations. Our secondary hypotheses were that MCT would be associated with reduced anxiety, improved mood and a greater sense of subjective recovery. Finally, we hypothesized that symptomatic change would closely follow change in CAS activity, as predicted by the S-REF model.

Method

Design

Following eligibility checks and a full initial assessment, three participants were randomly allocated to receive 2, 3, or 4 weeks of brief weekly baseline assessments. Treatment commenced at the end of the baseline periods if stability in at least one of the primary symptoms was demonstrated. In line with other multiple baseline studies of MCT, stability was defined as an absence of improvement in the primary outcome over at least two consecutive assessments (Wells et al., 2009). Conducting multiple baseline assessments controls for the effect of increased social contact, natural improvement and regression to the mean, allowing change following onset of treatment to be more confidently attributed to the intervention.

We planned for a maximum of 12 weekly 1-hour treatment sessions over a 3-month period. Participants received full assessments again at end of treatment and 3 months later, with no treatment provided in the interim period. Each participant also completed a brief assessment prior to each therapy session, allowing us to closely chart their progress.

Participants

The first three people who met our inclusion and exclusion criteria were included in the study. Since this was a pilot study, our aim was to recruit symptomatic, yet clinically stable

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and treatment-adherent individuals whose symptoms were not attributable to brain injury or alcohol/drug use, who wanted help and whose social care needs were being adequately met. By reducing the likelihood of symptomatic variance we hoped to increase our confidence that any subsequent change could be attributed to MCT.

Our inclusion criteria were therefore that participants had to: (1) have an identified care coordinator and be in regular contact with mental health services; (2) meet either ICD-10 criteria for schizophrenia, schizoaffective disorder or delusional disorder; (3) score at least 4 on PANSS delusions or hallucinations and/or 5 on suspiciousness; (4) be judged by their clinician to be clinically stable for at least the preceding 4 weeks; (5) be judged by their clinician to be taking prescribed antipsychotic medication for at least the previous 3 months; and (6) have distress ratings of at least 3 on either the auditory hallucinations or delusions subscale of the PSYRATS.

Participants were unable to take part if they: (1) had moderate to severe learning disability; (2) had evidence of clear organic neurological impairment (e.g. head injury or dementia); (3) were non-English speaking in so far as this would prevent the use of standardized assessment instruments; (4) had received inpatient/acute care immediately prior to or during baseline assessments; (5) were judged by their clinician to be clinically unstable within the preceding 4 weeks; (6) were judged by their clinician to be taking prescribed antipsychotic medication for less than 3 months preceding referral; (7) were not receiving care coordination; (8) had a primary diagnosis of substance misuse dependency; (9) had a score of 5 or more on conceptual disorganization on the PANSS.

Key participant details have been changed in the following descriptions in order to preserve anonymity. All diagnoses were made by the patients' psychiatrists as documented in the case notes.

Participant 1

Participant 1 was a 45-year old female who presented with persecutory delusions, anxiety and suicidal ideation. She had a diagnosis of paranoid schizophrenia and had experienced persecutory delusions for approximately 20 years. She believed with very strong conviction that her family, work colleagues and strangers were very critical of her and believed she was a terrorist. She used to drink heavily to cope with her high levels of anxiety, but had managed to stop this. She had tried a number of typical and atypical antipsychotics over a 15-year period, and was currently taking 20 mg aripiprazole. She had acquired tardive akathesia which had resisted treatment with procyclidine. She was also taking 40 mg citalopram, 30 mg mirtazapine and varying doses of pregabalin. She had seen at least two therapists in the past where she had received CBT and motivational interviewing. Although she had found this beneficial she was concerned that the effects had not lasted.

Participant 2

Participant 2 was a 30-year old male with a diagnosis of schizophrenia. He had experienced auditory and visual hallucinations for 15 years. He believed with very high conviction that aliens were appearing to him, talking to him and interfering with his thoughts. He was also fearful that strangers would attack him or his family. He also suffered from chronic sleep deprivation partly attributable to unsuccessfully treated sleep apnoea. He took pregabalin

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(150 mg), intermittently before and during therapy. He had also been taking olanzapine (20 mg), citalopram (20 mg) and zopiclone (7.5 mg) for a number of years. He was switched from olanzapine to quetiapine (600 mg) during therapy and then to amisulpride (400 mg). He had not received a psychological intervention before.

Participant 3

Participant 3 was a 55-year old female who had heard voices since she was an adolescent some 40 years ago. The voices, which she believed were the thoughts of other people intruding on her mind, were continuous despite treatment with various antipsychotics. She had a diagnosis of paranoid schizophrenia, and was taking olanzapine (20 mg) and citalopram (60 mg) at the start of therapy. Her antipsychotic was changed to risperidone (6 mg) during therapy. She believed with very high conviction that other people could read her thoughts and that her experiences were the result of a conspiracy involving MI5. She had taken illegal drugs in the past to cope with her experiences and was currently addicted to several prescription drugs. She also drank heavily, used illegal stimulants and was a carer for her adult son, who also experienced mental health problems. She had not received a psychological intervention before.

Outcome measures – primary

Psychotic Symptom Rating Scales (PSYRATS; Haddock, McCarron, Tarrier and Faragher, 1999). The PSYRATS is a widely-used clinician-administered assessment of various dimensions of auditory hallucinations and delusions. Dimensions assessed include distress, frequency, loudness, intensity, perceived control and disruption to life for auditory hallucinations and distress, conviction, preoccupation and disruption to life for delusions. Each dimension is assessed on a 0–4 point scale, with higher scores indicating greater severity. Total scores can range from 0–44 for auditory hallucinations and 0–24 for delusions. We used the PSYRATS as the primary outcome in this study because it takes less time to administer on a weekly basis.

Outcome measures - secondary

Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein and Opler, 1987). The PANSS is a structured clinical interview that assesses positive and negative symptoms of schizophrenia, as well as general psychopathology, occurring over the preceding week. Each of the 30 items is normally scored on a 1–7 point scale, with higher scores indicating greater severity. Following recent recommendations (Leucht, Kissling and Davis, 2010), we changed this to a 0–6 scale. Total scores normally range from 30–210, but this was adjusted to 0–180 for the current study.

Beck Depression Inventory, 2nd edition (BDI-2; Beck, Steer and Brown, 1996). This is widely-used self-report measure of depressive symptoms occurring in the preceding 2 weeks, where 21 items are rated on a 0–3 scale, allowing a range of 0–63. Higher scores indicate greater severity.

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Beck Anxiety Inventory (BAI; Beck, Epstein and Brown, 1988). The BAI is a self-report measure of anxiety symptoms occurring during the preceding week. Each of the 21 items is rated on 0–3 scale, allowing a maximum score of 63. Higher scores indicate greater severity.

Questionnaire about the Process of Recovery (QPR; Neil et al., 2009). The QPR is a 22-item self-report measure of recovery from psychosis developed in close collaboration with service-users. It measures two domains of recovery, intrapersonal and interpersonal functioning, on a 5-point scale, from 0 (strongly disagree) to 4 (strongly agree) allowing a range of 0–96. Higher scores correspond to greater subjective recovery.

Cognitive Attention Syndrome Scale (CAS-1; Wells et al., 2009). The CAS is a short self-report questionnaire that measures cognitive attentional syndrome activation in the preceding week. It provides a working measure of the frequency of worry and dwelling, threat-focused attention and counterproductive attempts at thought-control. It also provides a measure of current conviction in various positive and negative metacognitive beliefs. Thus, the CAS provides a short measure of the main features of the metacognitive model of psychosis we used in the current study.

We also administered other measures of metacognition, namely the Metacognitions Questionnaire (MCQ-30) (Wells and Cartwright-Hatton, 2004), the Interpretation of Voices Inventory (IVI) (Morrison, Wells and Nothard, 2002) and the Beliefs about Paranoia Scale (BAPS) (Gumley, Gillan, Morrison and Schwannauer, 2010), as well as a measure of social functioning, the Personal and Social Performance Scale (PSP) (Morosini, Magliano, Brambilla, Ugolini and Pioli, 2000). However, outcome data for these measures will be reported elsewhere, as they were administered as part of a larger study.

Procedure

Assessment. The PSYRATS, BDI-2, BAI and CAS-1 were completed during every assessment and treatment session. This allowed an accurate assessment of change in both our primary outcome and in our hypothesized mechanism of change (reduction in CAS scores). These assessments took on average 30 minutes to complete. The PANSS and QPR were completed at initial assessment, end-of-therapy and 3-month follow-up only. Administration of the PANSS increased the time taken for these assessments to 1–2 hours.

Participant 1 received 11 sessions of MCT lasting approximately 1 hour, participant 2 received 13, and participant 3 received 12. Therapy lasted longer than expected, mainly due to missed appointments but also because we decided to have longer gaps between sessions near the end of therapy. Participant 1's treatment lasted for 5 months, participant 2's for 8 months, and participant 3's for 10 months.

Treatment

Treatment followed the general principles of MCT for GAD, as outlined in Wells (2009). In accordance with the model we have presented, we did not attempt to engage in any direct treatment of hallucinations or delusions. The focus instead was on reducing worry, developing alternative ways of responding to thoughts and achieving metacognitive change. We attempted to achieve several goals in our first two sessions of MCT. These included generating an idiosyncratic formulation of a recent time when the participant was very distressed (see Figure 1 for an example), helping participants understand the counterproductive nature of

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existing thought-control strategies (e.g. suppression, self-harm, using worry to control worry) and encouraging participants to view their worrying thoughts as unimportant mental events that did not require further conceptual processing.

The second and third sessions involved teaching participants a new way of relating to their thoughts. A variety of exercises were used within and between sessions to help participants grasp the idea of detached mindfulness; these involved them developing a decentred awareness of their thoughts and letting them come and go without engaging with them (Wells, 2005).

The evidence for and against negative metacognitive beliefs concerning the uncontrollability of worry was then examined. Participants were encouraged to test these metacognitive beliefs by practising worry postponement and detached mindfulness between sessions. Every session they were encouraged to increase the proportion of worrying thoughts they could postpone or simply let go. Once participants believed they could control their thoughts, we moved on to examining negative metacognitive beliefs about danger. The experience of intrusive or racing thoughts was normalized using published data or idiosyncratic surveys. Participants were also encouraged to engage in symptom provocation experiments, where they increased their worry and attempted to induce their feared catastrophe (e.g. going mad, having a heart attack). The implications of non-occurrence of the catastrophe were discussed in detail. Examination of negative metacognitive beliefs often took 4–5 sessions.

Positive metacognitive beliefs about worry were then examined using a cost-benefit analysis and verbal restructuring. Participants were asked how long they had been worrying, and whether they had achieved their goals (e.g. finding answers). When participants did identify benefits of worrying (e.g. keeping themselves safe), they were asked whether they needed to do this now, or whether the costs now outweighed the benefits. Examination of positive metacognitive beliefs often took 2–3 sessions.

The aim of the final two sessions was to construct a comprehensive blueprint and relapse prevention plan. This contained a written and diagrammatic formulation, a summary of the strategies used, an overview of progress and a future plan. Old strategies of thought-control and information processing were contrasted with the new way taught in therapy.

Training

The first author treated all three participants. He received training and tape-based supervision during monthly meetings with Adrian Wells and several other MCT therapists-in-training.

Data analysis procedure

Session-by-session PSYRATS, BDI-2 and BAI scores were graphically represented along with time spent worrying and degree of conviction in negative and positive metacognitive beliefs. Visual inspection of these data was used to determine whether therapy onset was associated with an improvement in these domains.

Clinically significant change

Following previous clinical trials (Durham et al., 2003) we defined a clinically significant reduction as at least a 25% reduction in either PSYRATS auditory hallucination or delusions scores from baseline assessment to end-of-treatment. Following Leucht, Arbter et al. (2009),

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Table 1. Mean scores and standard deviations for each outcome measure across participants at baseline, end-of-therapy and follow-up

	Pre-treatment		Post-treatment		3-month follow-up	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
PSYRATS Voices	26.33	8.08	17.67	15.95	19.00	16.82
PSYRATS Delusions	17.33	0.58	13.33	1.15	14.33	3.51
PANSS Positive	15.33	4.93	10.00	1.00	9.33	2.52
PANSS Negative	15.33	5.03	7.67	2.31	8.33	4.16
PANSS General	19.67	8.62	14.67	3.79	15.67	6.11
PANSS Total	50.33	7.51	32.33	6.11	33.33	11.24
BDI-2	18.00	3.61	10.33	9.24	21.00	7.21
BAI	18.67	9.29	10.00	8.19	16.00	6.00
QPR	45.67	16.80	59.67	12.50	41.00	27.62
Duration of worry	4.67	1.15	4.33	1.53	4.00	2.00
Worry is uncontrollable (% conviction)	50.00	0	26.67	30.55	73.33	20.82
Worry is harmful (% conviction)	41.57	24.66	10.00	10.00	10.00	10.00
Worry helps me cope (% conviction)	10.00	17.32	16.67	15.28	40.00	36.06
Worry helps me find answers (% conviction)	66.67	25.17	20.00	26.46	46.67	35.12

Table 2. Proportion of PANSS "resolution" items scored less than 3

	Participant 1	Participant 2	Participant 3
PANSS "resolution" items $\leqslant$ 3 at baseline (number, %)	4, 50%	3, 37.5%	4, 50%
PANSS "resolution" items $\leqslant$ 3 at end-of-therapy (number, %)	7, 87.5%	6, 75%	6, 75%
PANSS "resolution" items $\leqslant$ 3 at follow-up (number, %)	7, 87.5%	7, 87.5%	5, 62.5%

we defined a response to treatment on the PANSS as a 25% reduction in PANSS total scores, from initial baseline to end-of-treatment (Leucht, Davis, Engel, Kissling and Kane, 2009). Following Peuskens, Kaufman and Van Vleymen (2007), we defined "resolution" of symptoms as a score of less than 3 on 8 selected PANSS remission items at post-treatment. Analysis of remission rates was not possible in the current study due to the limited duration of the follow-up period and limited number of assessment points.

Results

Each participant's PSYRATS, BDI-2 and BAI scores for every baseline assessment, therapy session, end-of-therapy and 3-month follow-up are displayed in Figure 2. Figure 3 shows each participant's scores on selected CAS-items measuring time spent worrying and conviction in key negative and positive metacognitions, over the same assessment points. Means and standard deviations for each outcome at baseline, end-of-therapy and follow-up are shown in Table 1. Table 2 shows the proportion of PANSS "resolution" items where participants scored less than 3.

As shown in Figure 2, participant 1's PSYRATS auditory hallucination scores dropped substantially during the baseline period. This was because she reported voices that

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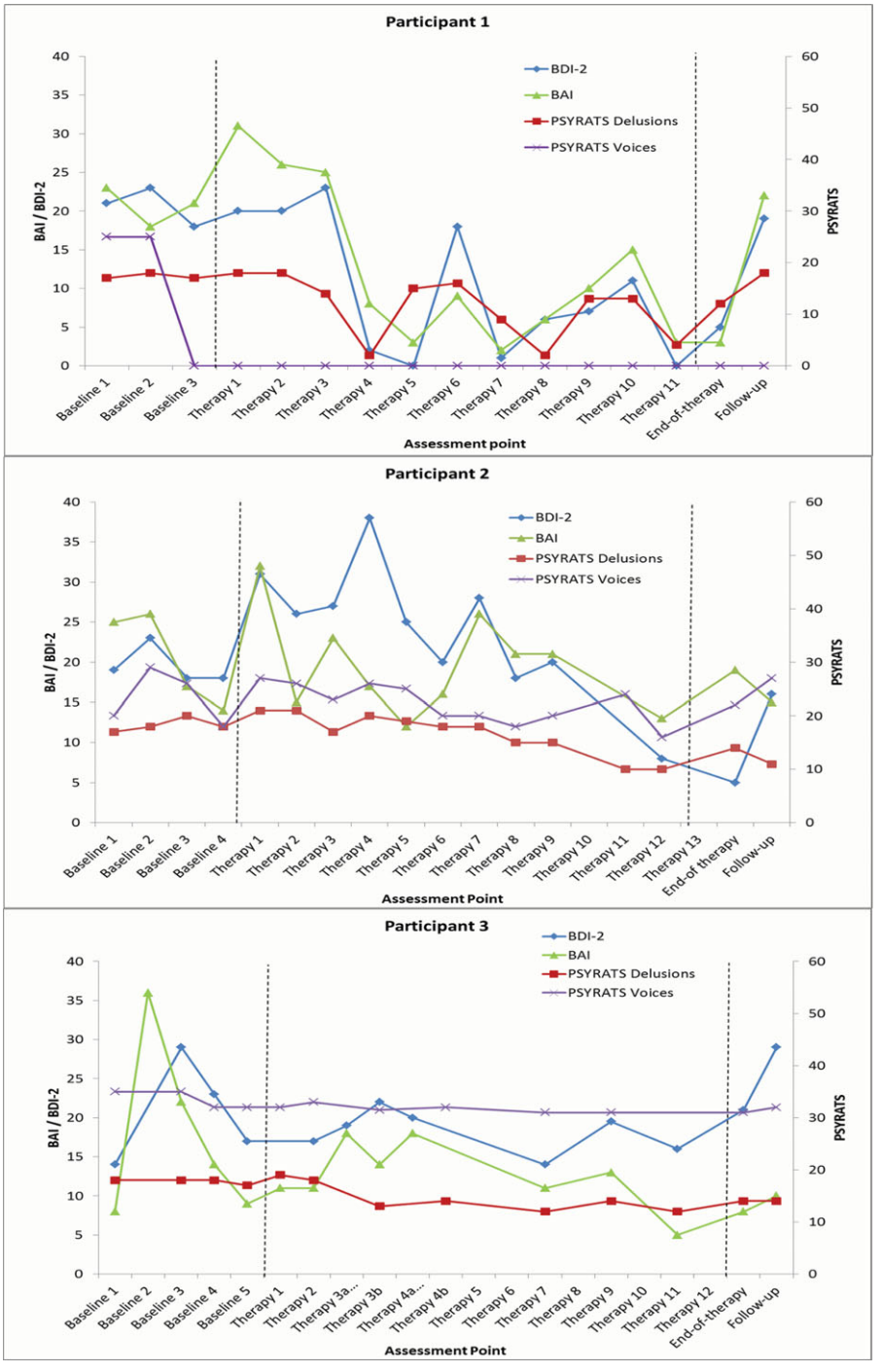


Figure 2. (Colour online) Each participant's ratings on the BDI, BAI and PSYRATS, from baseline to follow-up

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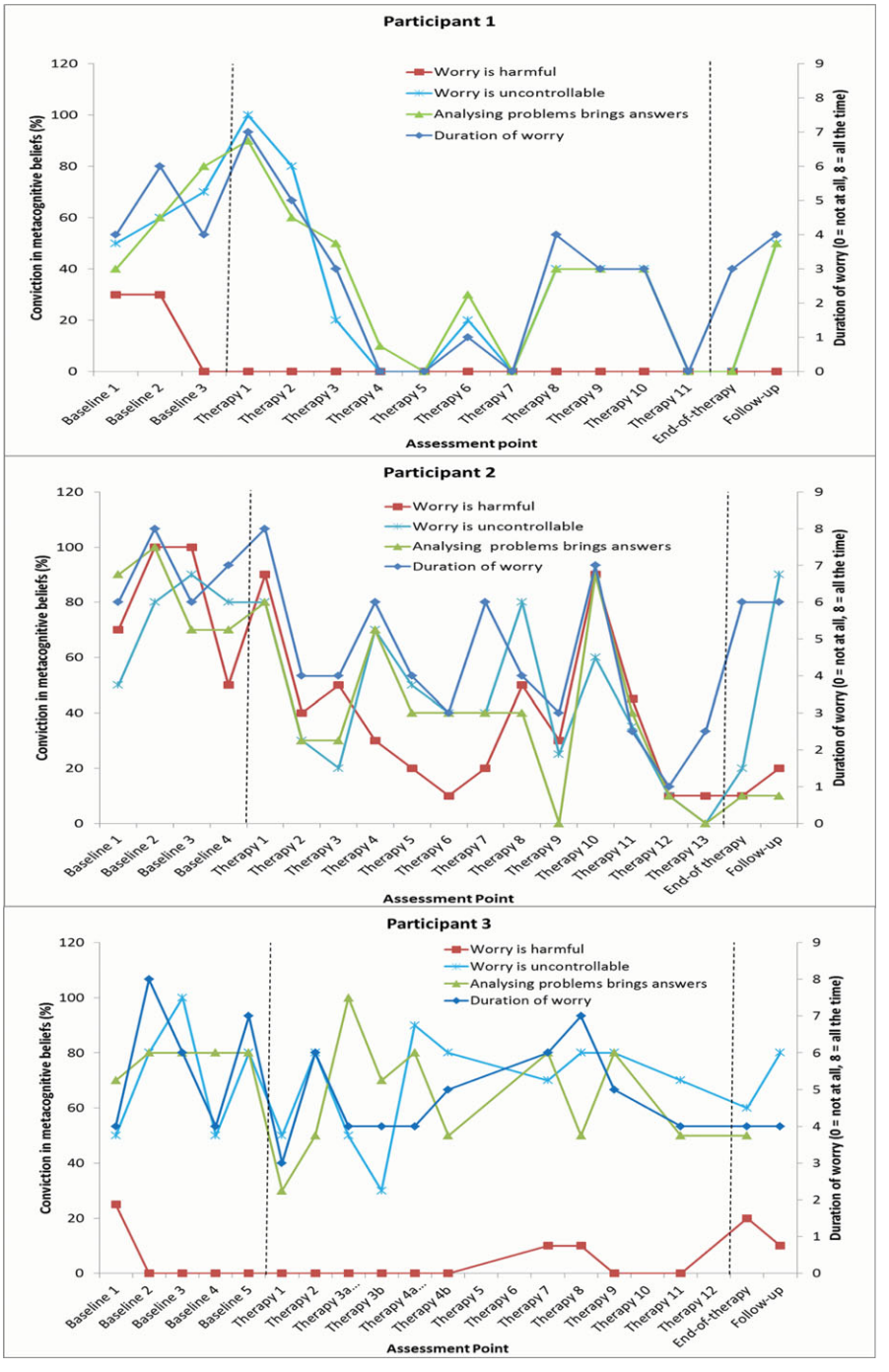


Figure 3. (Colour online) Each participant's ratings on key items from the Cognitive Attentional Syndrome Scale, from baseline to follow-up

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spontaneously remitted during the baseline assessments, before any therapy took place. Although we can speculate that therapy reduced the likelihood of recurrence, we suggest that focusing on PSYRATS delusion scores allows a more accurate assessment of treatment-attributable change. These scores are high and stable across the baseline period and become lower but more unstable throughout therapy. End-of-therapy scores suggest a substantial reduction of almost 30% from initial baseline; however follow-up scores suggest this improvement was not sustained. This same pattern was observed for BAI and BDI-2 scores as well as time spent worrying and degree of conviction in positive and negative metacognitions. The CAS and metacognition scores appear to decrease before psychotic symptom changes. This is consistent with the hypothesis that symptomatic change is achieved via modification of metacognitive beliefs and CAS deactivation. As shown in Table 2, BAI and BDI-2 scores at end-of-therapy were reduced by 87% and 76% respectively, compared to initial baseline. This improvement was not sustained; at follow-up her BDI-2 and BAI scores had returned to near baseline levels.

Participant 2's PSYRATS scores appeared to fluctuate to some degree over the baseline period and early stages of therapy. Change started to occur around therapy session 5 and by session 12 scores had reduced by almost 30% compared to baseline. This was not sustained by end-of-therapy and there was a slight increase in symptoms at follow-up. There was a large reduction of 63% in BDI-2 scores at end-of-therapy, despite these increasing markedly in the early stages of therapy. However this reduction was much smaller at follow-up (16%). There was a small 24% reduction in BAI scores at end-of-therapy compared to baseline, which grew to 40% at follow-up, suggesting any potential benefit was sustained. Although there was substantial variability in BAI scores throughout therapy, there was a clear and sustained reduction in conviction in target metacognitive appraisals. This was closely mirrored by a similar reduction in time spent worrying. This is again consistent with the hypothesis that metacognitive change leads to symptomatic improvement.

Participant 3 experienced a small but sustained reduction in PSYRATS auditory hallucination and delusion scores of at least 9% and 22% respectively. However this was associated with a concurrent 50–107% increase in BDI-2 scores and 25% worsening in BAI scores - the latter were very low at start and end, indicating only mild anxiety. The substantial variance in BDI-2 scores across baseline prevents us from confidently attributing the increase to potential negative effects of therapy. This fluctuation was mirrored by fluctuating conviction in key metacognitive appraisals and time spent worrying. Therapy did not appear to produce any major change in these indicators of CAS activation. This might indicate that metacognitive change is required for symptomatic improvement.

Clinically significant change

Our primary outcome measure was the PSYRATS. We decided, a-priori and on the basis of previous trials (Durham et al., 2003), that a 25% reduction would be clinically worthwhile. On this basis, two out of our three participants achieved a clinically worthwhile improvement in PSYRATS delusion scores, one at end of treatment only, with a reduction of 29%, and another at follow-up only, with a reduction of 35%. For the latter participant, this improvement in delusions was accompanied by a comparable increase in hallucinations, as measured by PSYRATS hallucinations scores.

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Our secondary measures suggest a somewhat different picture of change. As discussed, a reduction of 25% in PANSS total scores indicates clinically significant change in a group of patients whose symptoms are refractory to long-term antipsychotic treatment, while a 50% reduction reflects "much improvement" in this group (Leucht, Davis et al., 2009). Under this criterion, two participants experienced clinically significant change, one of whom also experienced much improvement. The latter went on to experience a further reduction in symptoms at follow-up, while the former returned to near baseline levels. Overall, participant 3 experienced little change in PANSS total scores, participant 1 experienced clinically significant change, which was not sustained at follow-up, while participant 2 experienced much improvement, which was sustained at 3-month follow-up. Looking at symptom subgroups, all participants experienced at least a 25% reduction in positive and negative symptoms by follow-up, compared to baseline; one experienced over a 50% reduction in negative symptoms, which was sustained at follow-up.

This picture of recovery is also reflected in the pattern of change on the QPR, our self-report measure of subjective recovery. Higher scores on this measure indicate greater subjective recovery. At end-of-therapy, participant 1 had a 43% increase, participant 2 had a 132% increase but participant 3 experienced a 27% reduction. At follow-up, participant 1's QPR scores had dropped by 76% compared to baseline, reflecting a substantial worsening. Participant 2's improvement appeared to be largely sustained, as reflected by a 90% increase compared to initial baseline. The reduction experienced by participant 3 had reduced somewhat at follow-up to 9%. Overall, one person had a significant improvement in recovery followed by a significant reduction, one had a large sustained improvement, and another experienced a significant reduction, which had returned to near baseline levels by follow-up.

Table 2 shows the proportion of resolution items scored 3 (mild) or less for each participant at baseline, end-of-therapy and follow-up. Although no participant achieved complete resolution (score of $\geqslant 3$ on each item), the pattern of change is clearly moving towards this, with improvement on these items continuing at follow-up for two out of three.

Discussion

Summary of results

Based on our primary outcome measure, we found that a relatively short number of sessions of MCT were associated with a large, clinically significant reduction in the severity of delusions for two out of our three participants. Unfortunately, this change was not sustained for one person and for the other it was accompanied by an increase in hallucinations.

With the secondary outcome measures we found two out of three patients experienced large and clinically significant reductions in PANSS total scores, while all three experienced significant reductions ($>25\%$) in PANSS positive and PANSS negative scores. We found two out of three experienced a large increase in self-rated recovery and a large decrease in depression and anxiety. These improvements continued at 3-month follow-up for one person, while the other lost the gains made and actually experienced a significant reduction in their sense of recovery, relative to baseline. Third, all patients moved towards resolution of

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symptoms, as indicated by their scoring 3 or less on a greater proportion of eight key PANSS items. This change largely persisted at follow-up.

We hypothesized that symptomatic change would closely follow change in metacognitive beliefs. This was supported by the pattern of responses on the CAS. The two participants who achieved symptomatic improvement also achieved a drop in conviction in positive and negative metacognitions. Participant 3, who did not respond, showed at best minimal change in conviction in negative metacognitions as well as an increase in positive beliefs about worry. In this case, treatment may have failed because of participant 3's long duration of illness, ongoing stimulant use and adverse home environment. We also note she was the only participant not to have a care coordinator, meaning she received little support in her role as a carer or with her complex social issues.

Although our results are encouraging, we must also note the risk of adverse effects, since participant 3 had a large reduction in mood, increase in anxiety and reduction in subjective recovery. Participant 1 also experienced a large reduction in self-rated recovery at follow-up when compared to baseline.

Limitations

Several factors limit our ability to generalize from our study. First, there were only three participants. Although case studies and case series such as ours are an essential first step in the process of developing and testing a new intervention, only a clinical trial can provide definitive evidence of efficacy. Second, although the main assessments were completed by an independent experienced assessor, our design meant they could not be blinded. Given that use of non-blinded assessors is associated with inflated estimates of effect size in clinical trials of CBT for psychosis (Wykes et al., 2008), future studies of MCT for psychosis should, if the design allows, employ independent and blinded raters. Third, our lack of a comparison group reduces our ability to confidently attribute change to therapy. Definitive conclusions must await the results of a randomized controlled trial. Changes in medication during treatment in two of the cases means that any observed effects could be attributed to this factor. It is also the case that the baseline PANSS assessment was only administered at the start of the multiple baseline period, and therefore any change in this measure includes change occurring during the baseline. Inspection of the graphs suggests this is an issue for interpreting participant 1's scores, whose auditory hallucinations resolved spontaneously during the baseline period. Our assessment of clinically significant change on the PSYRATS was also based on change from initial baseline, rather than last baseline. This could lead to an inaccurate estimate of treatment-attributable change; however, inspection of the graphs suggests this is only an issue for interpreting change in PSYRATS hallucinations of participant 1 (as discussed in the results section).

Conclusion and recommendations

Two out of three participants with long-standing, treatment-resistant psychotic symptoms appeared to achieve a clinically worthwhile benefit associated with MCT; all three experienced a reduction in positive and negative symptoms according to the PANSS. Treatment lasted between 11 and 13 hours, suggesting MCT may be a less time-intensive option for service-users. However, one participant experienced minimal improvement in

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symptoms, together with a reduction in mood and subjective sense of recovery. Furthermore, one participant who did respond did not maintain gains at follow-up. In interpreting these results, the obvious differences between the person who worsened and the two who recovered or stayed the same was a longer duration of illness (40 years versus 15–20), presence of complex substance-use problems, and a highly stressful home environment. In the therapist's clinical judgement, the latter was the primary reason for the deterioration. However, it is notable that MCT did not help this individual cope with these circumstances. On the other hand, there are also factors that made it more probable that participants 1 and 2 may have been more likely to improve, regardless of whether they received therapy. Both had supportive families, both abstained from drug and alcohol use, and both had a change in medication.

It is also important to set these results in context. For example, according to a recent meta-analysis, some 24% of participants demonstrated a clinically significant response to placebo alone in short-term trials of modern antipsychotics (Leucht, Arbter et al., 2009). In the largest, longest and arguably best-designed study of CBT, some 40–50% of participants receiving befriending demonstrated at least a 50% improvement in symptoms (Sensky et al., 2000). Non-specific factors (e.g. hope, expectation, companionship), natural recovery or other variables (e.g. rater bias) seem to contribute in large part to the efficacy of existing treatments. Our multiple baseline period may not have been long enough to control for these effects.

Another issue is whether our working formulation was appropriate and acceptable. We conceptualized negative thoughts rather than psychotic experiences as the trigger for the development of distress. It could be argued that this fails to recognize that for many people the content of their experiences (e.g. derogatory voices, or visions of evil entities) can be very distressing, even if one is not prone to worry. Clinical experience suggests that the person who believes the world is about to end, or that they have been possessed by a demon, may find it somewhat difficult to detach from what might be perceived as life or death concerns. A metacognitive model that acknowledges the importance of experience, cognition *and* metacognition in determining distress may have greater face validity with patients and providers.

Our results suggest MCT for psychosis should be evaluated further. A larger case series might be useful to see if any modifications are required for this group. Such studies might consider incorporating a qualitative element, allowing careful assessment of participants' views on the acceptability and accuracy of the metacognitive model and therapy approach. Our results might suggest MCT may need to be delivered for longer if change is to be sustained. The role of insight and conviction in responsiveness to MCT might also be important to consider. Perhaps it would be easier for patients to detach from their thoughts if they are able to contemplate that they might not be entirely realistic? Another important issue is whether all people with psychosis have the requisite capacity to think about their thoughts and feelings and, if not, whether this is something MCT can help to improve (Lysaker et al., 2011). Although we did not detect major problems in self-reflectivity, these may become apparent in a larger case-series.

If and when the active ingredients of MCT for psychosis are identified, comparison to treatment as usual using a randomized controlled design and blind raters would help to establish benefits and costs of the treatment, as well as ensuring it does not cause harm. For definitive assessment of value, MCT should at some stage be compared to the most established psychological treatment for psychosis, currently cognitive behavioural therapy.

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References

Baker, C. A. and Morrison, A. P. (1998). Cognitive processes in auditory hallucinations: attributional biases and metacognition. *Psychological Medicine*, *28*, 1199–1208.

Beck, A. T. (1976). *Cognitive Therapy and the Emotional Disorders*. New York: International Universities Press.

Beck, A. T., Epstein, N. and Brown, G. (1988). An inventory for measuring clinical anxiety: psychometric properties. *Journal of Consulting and Clinical Psychology*, *56*, 893–897.

Beck, A. T., Steer, R. A. and Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Bouman, T. K. and Meijer, K. J. (1999). A preliminary study of worry and metacognitions in hypochondriasis. *Clinical Psychology and Psychotherapy*, *6*, 96–101.

Clark, D. M., and Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg and M. R. Liebowitz (Eds.), *Social Phobia: diagnosis, assessment, and treatment* (pp. 69–93). New York: Guilford Press.

Durham, R. C., Guthrie, M., Morton, R. V., Reid, D. A., Treliving, L. R., Fowler, D., et al. (2003). Tayside-Fife clinical trial of cognitive-behavioural therapy for medication-resistant psychotic symptoms: results to 3-month follow-up. *British Journal of Psychiatry*, *182*, 303–311.

Fisher, P. L. and Wells, A. (2005). Experimental modification of beliefs in obsessive-compulsive disorder: a test of the metacognitive model. *Behaviour Research and Therapy*, *43*, 821–829.

Fisher, P. L. and Wells, A. (2008). Metacognitive therapy for obsessive-compulsive disorder: a case series. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*, 117–132.

Foster, C., Startup, H., Potts, L. and Freeman, D. (2010). A randomized controlled trial of a worry intervention for individuals with persistent persecutory delusions. *Journal of Behavior Therapy and Experimental Psychiatry*, *41*, 45–51.

Freeman, D. and Garety, P. A. (1999). Worry, worry processes and dimensions of delusions: an exploratory investigation of a role for anxiety processes in the maintenance of delusional distress. *Behavioural and Cognitive Psychotherapy*, *27*, 47–62.

Garety, P. A., Kuipers, E., Fowler, D., Freeman, D. and Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine*, *31*, 189–195.

Gumley, A. I., Gillan, K., Morrison, A. P. and Schwannauer, M. (2010). The development and validation of the Beliefs about Paranoia Scale (Short Form). *Behavioural and Cognitive Psychotherapy*, *39*, 35–53.

Haddock, G., McCarron, J., Tarrier, N. and Faragher, E. B. (1999). Scales to measure dimensions of hallucinations and delusions: the psychotic symptoms rating scales (PSYRATS). *Psychological Medicine*, *29*, 879–889.

Hutton, P., Morrison, A. P. and Taylor, H. (2012). Brief cognitive-behavioural therapy for hallucinations: can it help people who decide not to take antipsychotic medication? A case report. *Behavioural and Cognitive Psychotherapy*, *40*, 111–116.

Kay, S. R., Fiszbein, A. and Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*, 261–276.

https://doi.org/10.1017/S1352465812001026 Published online by Cambridge University Press

Laroi, F. and van der Linden, M. (2005). Metacognitions in proneness towards hallucinations and delusions *Behaviour Research and Therapy*, *43*, 1425–1441.

Lepping, P., Sambhi, R. S., Whittington, R., Lane, S. and Poole, R. (2011). Clinical relevance of findings in trials of antipsychotics: systematic review. *British Journal of Psychiatry*, *198*, 341–345.

Leucht, S., Arbter, D., Engel, R. R., Kissling, W. and Davis, J. M. (2009). How effective are second-generation antipsychotic drugs? A meta-analysis of placebo-controlled trials. *Molecular Psychiatry*, *14*, 429–447.

Leucht, S., Davis, J. M., Engel, R. R., Kissling, W. and Kane, J. M. (2009). Definitions of response and remission in schizophrenia: recommendations for their use and their presentation. *Acta Psychiatrica Scandinavica Suppl.*, *438*, 7–14.

Leucht, S., Kissling, W. and Davis, J. M. (2010). The PANSS should be rescaled. *Schizophrenia Bulletin*, *36*, 461–462.

Lieberman, J. A., Stroup, T. S., McEvoy, J. P., Swartz, M., Rosenheck, R., Perkins, D., et al. (2005). Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *New England Journal of Medicine*, *353*, 1209–1223.

Lin, Y. J. and Wicker, F. W. (2007). A comparison of the effects of thought suppression, distraction and concentration. *Behavior Research and Therapy*, *45*, 2924–2937.

Lynch, D., Laws, K. R. and McKenna, P. J. (2010). Cognitive behavioural therapy for major psychiatric disorder: does it really work? A meta-analytical review of well-controlled trials. *Psychological Medicine*, *40*, 9–24.

Lysaker, P. H., Buck, K. D., Carcione, A., Procacci, M., Salvatore, G., Nicolo, G., et al. (2011). Addressing metacognitive capacity for self reflection in the psychotherapy for schizophrenia: a conceptual model of the key tasks and processes. *Psychology and Psychotherapy: Theory Research and Practice*, *84*, 58–69.

Morosini, P. L., Magliano, L., Brambilla, L., Ugolini, S. and Pioli, R. (2000). Development, reliability and acceptability of a new version of the DSM-IV Social and Occupational Functioning Assessment Scale (SOFAS) to assess routine social functioning. *Acta Psychiatrica Scandinavica*, *101*, 323–329.

Morrison, A. P. (2001). The interpretation of intrusions in psychosis: an integrative cognitive approach to hallucinations and delusions. *Behavioural and Cognitive Psychotherapy*, *29*, 257–276.

Morrison, A. P., French, P. and Wells, A. (2007). Metacognitive beliefs across the continuum of psychosis: comparisons between patients with psychotic disorders, patients at ultra-high risk and non-patients. *Behaviour Research and Therapy*, *45*, 2241–2246.

Morrison, A. P., Gumley, A. I., Ashcroft, K., Manousos, R., White, R., Gillan, K., et al. (2011). Metacognition and persecutory delusions: tests of a metacognitive model in a clinical population and comparisons with non-patients. *British Journal of Clinical Psychology*, *50*, 223–233.

Morrison, A. P. and Wells, A. (2000). Thought control strategies in schizophrenia: a comparison with non-patients. *Behaviour Research and Therapy*, *38*, 1205–1209.

Morrison, A. P., Wells, A. and Nothard, S. (2002). Cognitive and emotional predictors of predisposition to hallucinations in non-patients. *British Journal of Clinical Psychology*, *41*, 259–270.

Najmi, S., Riemann, B. C. and Wegner, D. M. (2009). Managing unwanted intrusive thoughts in obsessive-compulsive disorder: relative effectiveness of suppression, focused distraction, and acceptance. *Behavior Research and Therapy*, *47*, 494–503.

Neil, S. T., Kilbride, M., Pitt, L., Welford, M., Nothard, S., Sellwood, W., et al. (2009). The Questionnaire about the Process of Recovery (QPR): a research instrument developed in collaboration with service users. *Psychosis*, *1*, 145–155.

Newton-Howes, G. and Wood, R. (2011). Cognitive behavioural therapy and the psychopathology of schizophrenia: systematic review and meta-analysis. *Psychology and Psychotherapy: Theory, Research and Practice*. doi: 10.1111/j.2044-8341.2011.02048.x

https://doi.org/10.1017/S1352465812001026 Published online by Cambridge University Press

NICE (2009). *Schizophrenia: core interventions in the treatment and management of schizophrenia in adults in primary care and secondary care (update)*: London: National Institute for Health and Clinical Excellence.

Papageorgiou, C. and Wells, A. (2001). Metacognitive beliefs about rumination in recurrent major depression *Cognitive and Behavioral Practice*, *8*, 160–164.

Pechey, R. and Halligan, P. (2011). The prevalence of delusion-like beliefs relative to sociocultural beliefs in the general population. *Psychopathology*, *44*, 106–115.

Pechey, R. and Halligan, P. (in press). Prevalence and correlates of anomalous experiences in a large non-clinical sample. *Psychology and Psychotherapy: Theory, Research and Practice*.

Peuskens, J., Kaufman, L. and Van Vleymen, B. (2007). Analysis of resolution criteria in patients with schizophrenia treated with olanzapine for an acute psychotic episode. *Schizophrenia Research*, *95*(1–3), 169–173.

Proctor, D. (2008). *A Randomized Controlled Trial of Metacognitive Therapy versus Exposure Therapy for Posttraumatic Stress Disorder.* Thesis submitted to the University of Manchester for the degree of Doctor of Clinical Psychology in the Faculty of Medical and Human Sciences.

Rattehalli, R. D., Jayaram, M. B. and Smith, M. (2010). Risperidone versus placebo for schizophrenia. *Cochrane Database System Review*, *1*, CD006918.

Sensky, T., Turkington, D., Kingdon, D., Scott, J. L., Scott, J., Siddle, R., et al. (2000). A randomized controlled trial of cognitive-behaviour therapy for persistent symptoms in schizophrenia resistant to medication. *Archives of General Psychiatry*, *57*, 165–172.

Simpson, C. and Papageorgiou, C. (2003). Metacognitive beliefs about rumination in anger. *Cognitive and Behavioral Practice*, *10*, 91–94.

Startup, H., Freeman, D. and Garety, P. A. (2007). Persecutory delusions and catastrophic worry in psychosis: developing the understanding of delusion distress and persistence. *Behavior Research and Therapy*, *45*, 523–537.

Tai, S. and Turkington, D. (2009). The evolution of cognitive behavior therapy for schizophrenia: current practice and recent developments. *Schizophrenia Bulletin*, *35*, 865–873.

Valmaggia, L., Bouman, T. and Schuurman, L. (2007). Attention training with auditory hallucinations: a case study. *Cognitive and Behavioral Practice*, *14*, 127–133.

van der Heiden, C., Muris, P. and van der Molen, H. T. (2011). Randomized controlled trial on the effectiveness of metacognitive therapy and intolerance-of-uncertainty therapy for generalized anxiety disorder. *Behavior Research and Therapy*, *50*, 100–109.

Varese, F. and Bentall, R. P. (2011). The metacognitive beliefs account of hallucinatory experiences: a literature review and meta-analysis. *Clinical Psychology Review*, *31*, 850–864.

Villeneuve, K., Potvin, S., Lesage, A. and Nicole, L. (2010). Meta-analysis of rates of drop-out from psychosocial treatment among persons with schizophrenia spectrum disorder. *Schizophrenia Research*, *121*(1–3), 266–270.

Warner, L., Mariathasan, J., Lawton-Smith, S. and Samele, C. (2006). *A Review of the Literature and Consultation on Choice and Decision-making for Users and Carers of Mental Health and Social Care Services*. London: King's Fund/Sainsbury Centre for Mental Health.

Wegner, D. M., Schneider, D. J., Carter, S. R. III and White, T. L. (1987). Paradoxical effects of thought suppression. *Journal of Personality and Social Psychology*, *53*, 5–13.

Wells, A. (1995). Meta-cognition and worry: a cognitive model of generalized anxiety disorder. *Behavioural and Cognitive Psychotherapy*, *23*, 301–320.

Wells, A. (2005). Detached mindfulness in cognitive therapy: a metacognitive analysis and ten techniques. *Journal of Rational-Emotive and Cognitive-Behavioral Therapy*, *23*, 337–355.

Wells, A. (2008). *Metacognitive Therapy for Anxiety and Depression*. New York: Guilford Press.

Wells, A. (2009). *Metacognitive Therapy for Anxiety and Depression*. New York: Guilford Press.

Wells, A. and Cartwright-Hatton, S. (2004). A short form of the metacognitions questionnaire: properties of the MCQ-30. *Behaviour Research and Therapy*, *42* 385–396.

https://doi.org/10.1017/S1352465812001026 Published online by Cambridge University Press

Wells, A., Fisher, P., Myers, S., Wheatley, J., Patel, T. and Brewin, C. R. (2009). Metacognitive therapy in recurrent and persistent depression: a multiple-baseline study of a new treatment. *Cognitive Therapy and Research*, *33*, 291–300.

Wells, A. and King, P. (2006). Metacognitive therapy for generalized anxiety disorder: an open trial. *Journal of Behavior Therapy and Experimental Psychiatry*, *37*, 206–212.

Wells, A. and Matthews, G. (1994). *Attention and Emotion: a clinical perspective*. Hillsdale NJ: Lawrence Erlbaum Associates.

Wells, A. and Sembi, S. (2004). Metacognitive therapy for PTSD: a core treatment manual. *Cognitive and Behavioral Practice*, *11*, 365–377.

Wells, A., Welford, M., Fraser, J., King, P., Mendel, E., Wisely, J., et al. (2008). Chronic PTSD treated with metacognitive therapy: an open trial. *Cognitive and Behavioral Practice*, *15*, 85–92.

Wells, A., Welford, M., King, P., Papageorgiou, C., Wisely, J. and Mendel, E. (2010). A pilot randomized trial of metacognitive therapy vs applied relaxation in the treatment of adults with generalized anxiety disorder. *Behaviour Research and Therapy*, *48*, 429–434.

Wykes, T., Steel, C., Everitt, B. and Tarrier, N. (2008). Cognitive behavior therapy for schizophrenia: effect sizes, clinical models, and methodological rigor. *Schizophrenia Bulletin*, *34*, 523–537.

https://doi.org/10.1017/S1352465812001026 Published online by Cambridge University Press