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Rumination-focused cognitive behaviour therapy for residual depression: A case series

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Abstract

The treatment of chronic and recurrent depression is a priority for the development of new interventions. The maintenance of residual symptoms following acute treatment for depression is a risk factor for both chronic depression and further relapse/recurrence. This open case series provides the first data on a cognitive-behavioural treatment for residual depression that explicitly targets depressive rumination. Rumination has been identified as a key factor in the onset and maintenance of depression, which is found to remain elevated following remission from depression. Fourteen consecutively recruited participants meeting criteria for medication-refractory residual depression [Paykel, E.S., Scott, J., Teasdale, J.D., Johnson, A.L., Garland, A., Moore, R. et al., 1999. Prevention of relapse in residual depression by cognitive therapy—a controlled trial. *Archives of General Psychiatry 56,* 829–835] were treated individually for up to 12 weekly 60-min sessions. Treatment specifically focused on switching patients from less helpful to more helpful styles of thinking through the use of functional analysis, experiential/imagery exercises and behavioural experiments. Treatment produced significant improvements in depressive symptoms, rumination and co-morbid disorders: 71% responded (50% reduction on Hamilton Depression Rating Scale) and 50% achieved full remission. Treating depressive rumination appears to yield generalised improvement in depression and co-morbidity. This study provides preliminary evidence that rumination-focused CBT may be an efficacious treatment for medication-refractory residual depression.

Keywords: Rumination; Cognitive behaviour therapy; Behavioural activation; Residual depression; Case series

Introduction

In recent years, there has been growing recognition that depression is a chronic and recurring condition (Judd, 1997), with improved relapse/recurrence prevention identified as a priority for future treatment research (e.g., Hollon et al., 2002). An important risk factor for relapse and recurrence is partial remission, that is, the maintenance of residual symptoms following acute treatment for depression. Residual symptoms of

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depression repeatedly predict increased risk for depressive relapse in prospective longitudinal studies (Fava, 1999; Judd, 1997; Judd et al., 1999; Paykel et al., 1995). Residual depression is a common problem, with one-third of patients not responding fully to acute treatments, most commonly antidepressant medication (Cornwall & Scott, 1997; Paykel et al., 1995). Residual symptoms are also an important clinical target because they produce substantial distress and are associated with marked functional impairments and increased health care utilization (Cornwall & Scott, 1997; Judd, 1997; Paykel et al., 1995).

Cognitive-behavioural therapy (CBT) for the residual symptoms of depression remaining after antidepressant medication has been found to be effective at reducing subsequent depressive relapse over the next 4–6 years in randomised controlled trials (Fava, Grandi, Zielezny, Canestrari, & Morphy, 1994; Fava, Grandi, Zielezny, Rafanelli, & Canestrari, 1996; Fava, Rafanelli, Grandi, Canestrari, & Morphy, 1998; Paykel et al., 1999). Nonetheless, CBT's relapse/recurrence prevention effect has its limits: approximately one-third of patients have relapsed by 68 weeks (Paykel et al., 1999) and the effect fades 3.5 years after the completion of CBT (Paykel et al., 2005). The impact of CBT on acute residual symptoms is less clear-cut. In patients with moderate levels of residual symptoms, Paykel et al. (1999) found that the addition of CBT to treatment-as-usual (clinical management and antidepressant medication), while reducing residual symptoms, did not significantly improve acute symptoms compared to treatment-as-usual, although it did significantly decrease relapse. Although Fava and colleagues found that CBT did significantly reduce residual symptoms compared to clinical management in patients successfully treated with antidepressant medication, the generalisability of these findings to more severe residual depression is unknown, given that (a) antidepressant medication was discontinued in both groups, leaving the clinical management group with no active treatment, (b) the sample size was small, and (c) the initial level of residual symptoms was low (Fava et al., 1994, 1996, 1998). These trials suggest the value of CBT interventions for residual symptoms, while indicating that there is significant scope for improvements in therapeutic efficacy.

One potential way to improve the efficacy of CBT for residual depression is to adapt CBT to specifically address core residual symptoms. The current study reports the preliminary test of a modified form of CBT for depression intended to improve the treatment of residual depression by targeting depressive rumination (Nolen–Hoeksema, 1991)(rumination-focused CBT, RFCBT). Rumination is hypothesised to play an important role in residual depression because it: (a) is a common residual symptom, remaining elevated after both partial and full remission from depression (Riso et al., 2003; Roberts, Gilboa, & Gotlib, 1998); (b) is associated with less responsiveness to both antidepressant and cognitive-behavioural interventions (Ciesla & Roberts, 2002; Schmaling, Dimidjian, Katon, & Sullivan, 2002); and (c) prospectively predicts the onset, severity, and duration of depression (e.g., Just & Alloy, 1997; Kuehner & Weber, 1999; Nolen–Hoeksema, 2000; Spasojevic & Alloy, 2001). Thus, the aim of the current study was to use a single case series to establish preliminary evidence of whether RFCBT has the potential to be an efficacious therapy for difficult-to-treat residual depression.

Although still grounded within the core principles and techniques of CBT for depression (Beck, Rush, Shaw, & Emery, 1979, e.g., collaborative empiricism, Socratic questioning), RFCBT involves several additional, novel elements. First, it incorporates the functional–analytic and contextual approach developed in the behavioural activation treatment that resulted from a component analysis of CBT (BA; Addis & Martell, 2004; Jacobson et al., 1996; Jacobson, Martell, & Dimidjian, 2001; Martell, Addis, & Jacobson, 2001). BA approaches have been integrated into RFCBT because BA includes an explicit focus on reducing rumination from a functional–analytical perspective (Addis & Martell, 2004; Martell et al., 2001) and has been found to be an effective intervention for depression (Dimidjian et al., 2006). Within BA and RFCBT, rumination is conceptualised as a form of avoidance, and functional analysis is used to facilitate the reduction of this avoidance and to replace it with more helpful approach behaviours. Second, the approaches used within RFCBT are derived from recent experimental research which suggests that there are distinct styles of rumination, with distinct functional properties and consequences: a helpful style characterised by concrete, process-focused and specific thinking versus an unhelpful, maladaptive style characterised by abstract, evaluative thinking (Treynor, Gonzalez, & Nolen–Hoeksema, 2003; Watkins, 2004; Watkins & Baracaia, 2002; Watkins & Moulds, 2005; Watkins & Teasdale, 2001, 2004).

Thus, in practise, RFBCT uses functional analysis to help patients realise that their rumination about negative self-experience can be helpful or unhelpful and then to coach them in how to shift into the most

effective style of thinking. Functional analysis focuses on the variability of: (a) rumination (e.g., differences between helpful and unhelpful thinking about problems); (b) associated behaviours (e.g., procrastination) and (c) counter-rumination behaviours such as effective engagement in tasks. This detailed analysis of context and function is then used to help patients: (a) recognise warning signs for rumination; (b) develop alternative strategies and contingency plans (e.g., relaxation, assertiveness); (c) alter environmental and behavioural contingencies maintaining rumination (e.g., shifting the balance from routine chores and obligations towards self-fulfilling activities). Further, RFCBT uses experiential/imagery exercises and behavioural experiments designed to facilitate a shift into more helpful thinking style. Patients use directed imagery to vividly recreate previous states when a more helpful thinking style was active, such as memories of being completely absorbed in an activity (e.g., "flow" or "peak" experiences) or experiences of being compassionate to themselves or others. Such exercises provide a direct counter to rumination and can be used within contingency plans. These adaptations mean that RFCBT differs from standard CBT for depression, which focuses on modifying the content of thoughts, by having a greater emphasis on directly modifying the process of thinking.

Method

Participants

Fourteen patients (9 women) who were referred to outpatient treatment clinics for residual depression based in London ($n = 6$) and Mid-Devon ($n = 8$) were consecutively enrolled in the 12-session individually based RFCBT. To be considered for the case series, patients needed to meet criteria for medication-refractory residual depression as defined by Paykel et al. (1999), namely: (a) meeting DSM-IV criteria (American Psychiatric Association, 1994) for major depression within the last 18 months but not in the last 2 months, (b) residual symptoms reaching at least 8 on the 17-item Hamilton Depression Rating Scale (HRSD; Hamilton, 1960; Williams, 1998) and 9 on the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996), (c) taking antidepressant medication at a therapeutic dose as recommended by the British National Formulary and/or equivalent to 125 mg of amitriptyline for at least 8 weeks continuously during the current episode and within the last 2 months. Residual symptoms had lasted between 3 and 24 months and the mean number of previous episodes of major depression (including the current partially remitted episode) was 5.79. All participants gave signed written informed consent to participate in the open case series.

Exclusion criteria were a history of bipolar disorder, psychosis, current drug or alcohol dependence, learning disability, organic brain damage, physical impairment that would interfere with the ability to attend therapy, and concurrent psychotherapy. We did not have any exclusion criteria with respect to co-morbid anxiety disorders or Axis II diagnoses. Current co-morbid disorders at initial assessment included social phobia (50%, $n = 7$), generalised anxiety disorder (GAD, 43%, $n = 6$), panic disorder (50%, $n = 7$), agoraphobia without panic (21%, $n = 3$), obsessive–compulsive disorder ($n = 1$), bulimia nervosa ($n = 1$), post-traumatic stress disorder ($n = 1$), health anxiety ($n = 1$), dysthymia ($n = 1$), avoidant personality disorder ($n = 2$), depressive personality disorder ($n = 1$), and obsessive–compulsive personality disorder ($n = 1$). Three patients had a previous history of alcohol and/or substance dependence. Antidepressant medication taken included fluoxetine at 20 mg daily (36%, $n = 5$), venlafaxine (29%, $n = 4$; dose range 75–225 mg daily), citalopram 40 mg daily (14%, $n = 2$), paroxetine 20 mg ($n = 1$), dothiepin ($n = 1$), and lofepramine ($n = 1$).

All patients were Caucasian. The patients' ages ranged from 26 to 57 ($M = 42.86$, SD = 11.84). Four patients were unemployed, five patients were working full-time, and five working part-time. All the patients who were unemployed or only working part-time reported that this was related to difficulties caused by their depression. Seven patients were married, three were divorced and four were currently single.

Consistent with the proposed relationship between residual depression and elevated depressive rumination, the participants had a mean rumination score at baseline of 59.4, which is consistent with the levels of rumination found in chronic current depression (Riso et al., 2003) and greater than 2 SD above the mean level of rumination reported in non-depressed populations (Roberts et al., 1998). Despite not meeting criteria for current major depression, the high levels of depressive symptoms reported are consistent with the chronic, recurrent, complex and co-morbid nature of the participant's clinical presentations.

Design

As well as an overall statistical examination of average treatment effect of RFCBT on symptom measures across patients from pre-treatment to post-treatment, we also used a multiple-baseline design, with the length of baseline prior to treatment ranging from 1–17 weeks. A measure of depressive symptoms (the BDI-II) was administered each week of the baseline and each week of the course of therapy. The added benefit of the multiple-baseline design is that should we observe that symptoms of depression reduce when and only when the treatment programme is underway in each participant, despite the differences in individual presentation and length of baseline, this observation would reduce the likelihood that other factors (e.g., history, maturation, statistical regression, spontaneous remission) were responsible for symptom improvement (Hayes, 1981; Kazdin, 2003).

Assessments and materials

All patients underwent extensive pre-treatment and post-treatment assessments of depressive symptoms and diagnostic status. The pre-treatment assessment was the start of the baseline recording period, and the post-treatment assessment was arranged, where possible, within 4 weeks of therapy completion.[1] Assessments were not blind: the assessor knew that patients were receiving therapy. The assessments consisted of the following measures:

The Structured Clinical Interview for DSM-IV (SCID; Spitzer, Williams, Gibbon, & First, 1996)

The SCID for Axis I and Axis II diagnoses was administered at each assessment by an experienced clinician or a trained research worker for both current and past diagnoses. The SCID was used to ensure that participants met the study criteria and to examine whether diagnostic status changed across the course of therapy.

Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960; Williams, 1998)

The 17-item HRSD was administered in an interview format at each assessment.

Beck Depression Inventory (BDI-II; Beck et al., 1996).

The BDI-II is a 21-item self-report instrument developed to measure severity of depression in adults and adolescents. Higher scores represent greater depression severity (range 0–63), and minimal, mild, moderate and severe symptom severity ranges have been specified. The BDI-II was administered (where possible) every week of the treatment and each week for a varying baseline period before the start of treatment.

The Ruminative Response Scale of the Response Styles Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991)

The RRS consists of 22 items that assess ruminative responses to sad and depressed mood. Participants rate the frequency that they use ruminative strategies, and higher scores connote higher levels of rumination (range 22–88). The RRS assesses the less helpful style of rumination, with a number of items focusing on abstract evaluations of the self (e.g., Think "Why do I always react this way?", Treynor et al., 2003), and with elevated scores on the RRS predicting worse outcomes. The RRS was administered pre-treatment and post-treatment in order to assess whether the course of RFCBT influenced levels of rumination.

RFCBT

RFCBT consisted of up to 12 sessions lasting 60 min, scheduled weekly or fortnightly, where possible. The treatment protocol was approved by an institutional review board. Treatment was provided by 5 doctoral level

[1]For 11 patients (78%), the post-treatment assessment was within 4 weeks of completing therapy; for the remaining 3 patients, we were not able to complete the post-therapy assessment until 4–6 months after therapy—for these patients, the final BDI score was used as the end of therapy symptom measure and both current and retrospective diagnostic status and HRSDs were calculated.

clinical psychologists and 1 psychiatrist, all of whom had received at least 12 months prior supervision in CBT, and received RFCBT supervision every 2 weeks. Sessions were audio-taped to facilitate supervision and monitor adherence.

Results

Response to treatment was determined with the Wilcoxon paired rank sum test to compare participant's symptoms before and after treatment, using an intention to treat analysis. Within-group effect sizes (Cohen's *d*) were calculated for each Wilcoxon test. Treatment response was defined as $\geqslant 50\%$ decrease in baseline HRSD scale. Remission was defined as symptom levels below 8 on the HRSD at termination and below 9 on the BDI for 4 consecutive weeks. Table 1 reports the means and SD on the outcome measures at each assessment point, and the within-group effect sizes.

Treatment was well tolerated. All patients received at least 10 sessions of therapy, with the exception of one patient who discontinued at session 5, reporting significant symptomatic improvement. Patients attended a mean number of 11.2 sessions (SD = 1.9).

The RFCBT treatment programme was associated with a significant reduction in depressive symptoms on both the BDI and HRSD (both p's $<.001$). At termination, 71% of patients met criteria for treatment response and 50% met full remission criteria. There were large effect sizes for declines in depressive symptoms, reflecting a mean reduction on the BDI-II of 20.36 points and on the HRSD of 9.00 points, both Cohen's $d>2$, (see Table 1). There was also a significant mean reduction in levels of rumination, $p<.001$.

Furthermore, there was a reduction in co-morbid diagnoses at the end of therapy. Current co-morbid Axis I disorders at post-therapy assessment were social phobia (14%, $n=2$), GAD (7%, $n=1$), panic disorder (7%, $n=1$), agoraphobia (7%, $n=3$), and dysthymia (7%, $n=1$). That is, in the course of the RFCBT treatment, five patients no longer met diagnostic criteria for social phobia, five patients no longer met diagnostic criteria for GAD, six patients no longer met diagnostic criteria for panic disorder and one patient no longer met criteria for health anxiety. In addition, one patient, with multiple co-morbidity pre-treatment, no longer met diagnostic criteria for obsessive–compulsive disorder, bulimia nervosa and post-traumatic stress disorder, all in partial remission, following RFCBT. Overall there was a 71% reduction in co-morbid Axis I diagnoses from baseline to post-intervention (i.e., from 28 co-morbid diagnoses to 8). Given the single post-therapy assessment, it was not possible to reach any firm conclusions about the impact of RFCBT on Axis II diagnoses, although there were reductions in reported personality disorders from four at baseline to one post-treatment, consistent with the improvement in personality disorders found with symptomatic improvements in depression following antidepressant treatment (Fava et al., 2002).

In order to evaluate whether the treatment impacted on outcomes in the experimental single case design, the multiple-baseline data are visually inspected with reference to criteria relating to changes in the magnitude (i.e., mean, level) and rate (i.e., trend, slope, latency) of the symptom scores across treatment phases (baseline to therapy) (Hayes, 1981; Kazdin, 2003). Meeting these criteria typically requires marked treatment effects, of

Table 1
Scores at baseline and at end of RFCBT for 14 Participants with residual depression

Measure	Score				Analysis		
	Baseline		Post-treatment		Effect size[a]	Wilcoxon z	p[b]
	Mean	SD	Mean	SD			
HRSD	14.07	3.67	5.07	4.94	2.07	−3.18	0.001
BDI	29.36	6.92	9.00	8.98	2.54	−3.18	0.001
RSQ	59.43	5.96	44.21	12.90	1.51	−2.75	0.006

Note. HRSD, Hamilton Rating Scale for Depression; BDI, Beck Depression Inventory-II; RSQ, rumination scale of Response Style Questionnaire.

[a]Within-group effect size (Cohen's *d*). Large effect sizes were defined as $\geqslant 0.80$.

[b]From Wilcoxon paired rank sum tests.

at least as much stringency as tests for statistical significance. Data representing the BDI-II scores for each week across the individual patients over the baseline and treatment periods are presented in Fig. 1. Because patients were encouraged to report their depressive symptoms each week, even if the therapy sessions occurred less often than weekly, the figure depicts both week-by-week level of depressive symptoms and which weeks actually included a therapy session. Given the frequent sampling of symptoms required for this design coupled with the relatively high level of depressive symptoms in the sample, there were a number of non-completions of the BDI-II during the baseline phase.

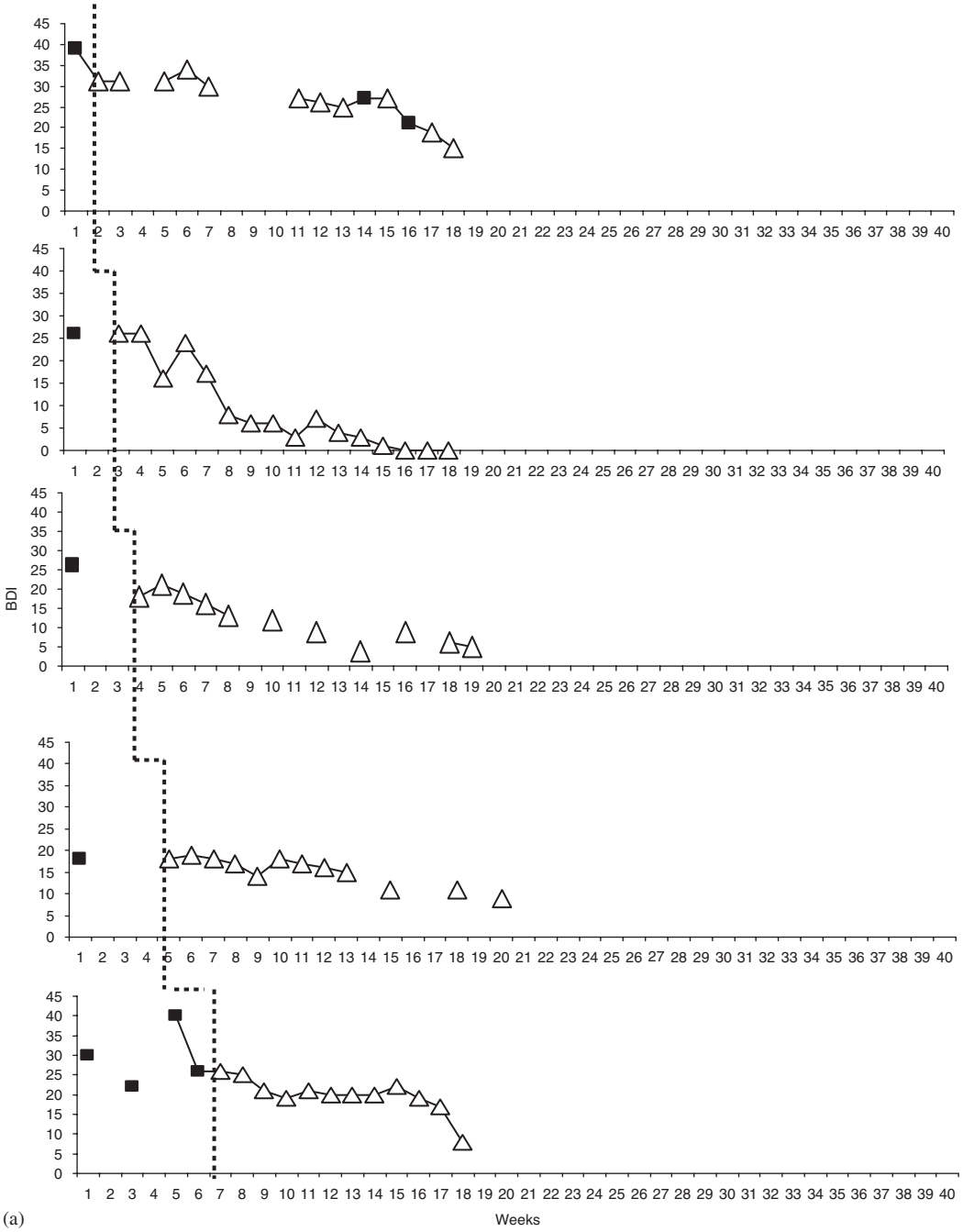

Fig. 1. Multiple-baseline design across 14 patients for depressive symptoms per week. In all graphs, the points marked with triangles are treatment sessions and the points marked with squares are other weeks where BDI was completed.

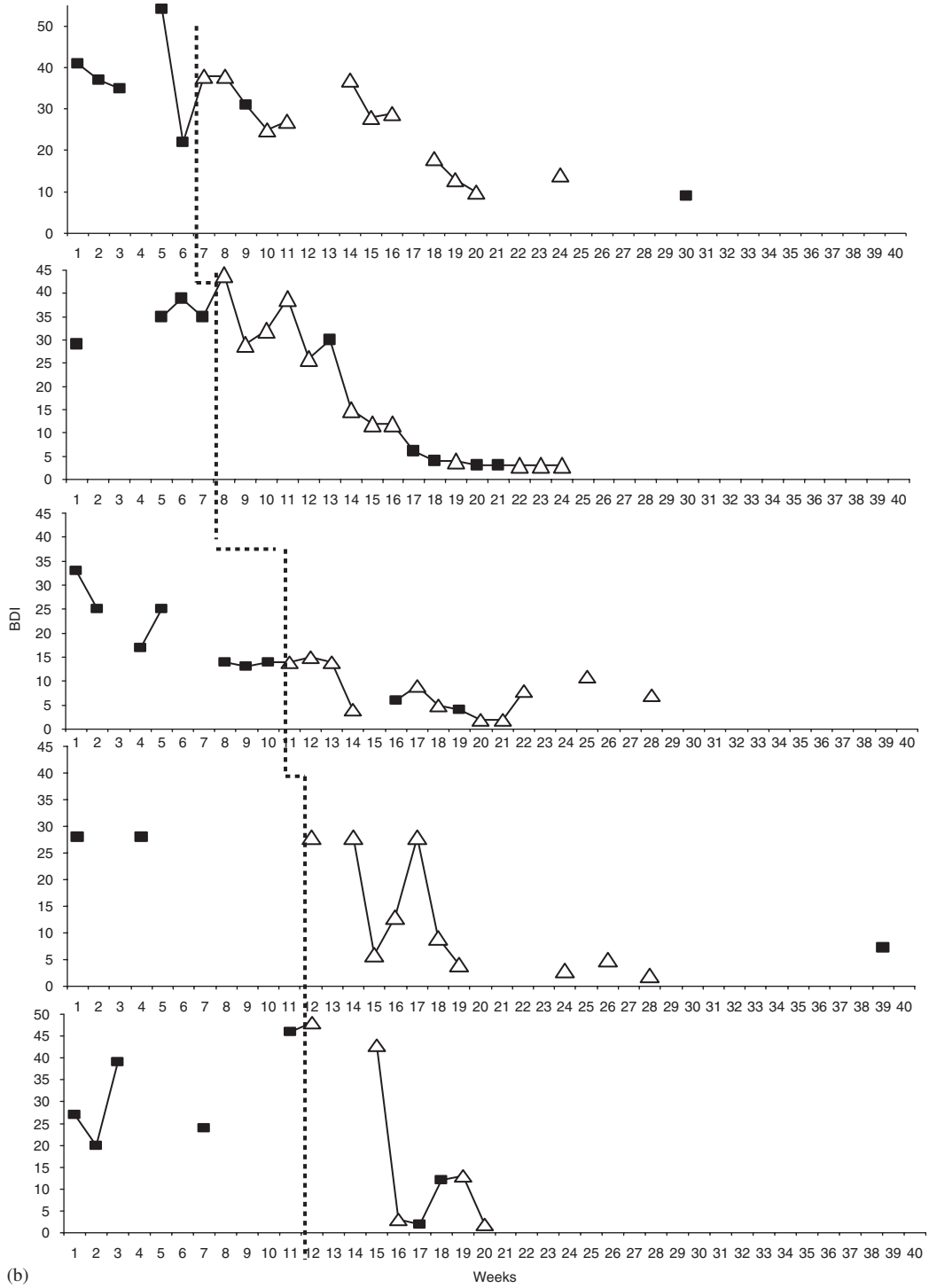

Fig. 1. (*Continued*)

Visual inspection of the multiple-baseline series (see Fig. 1a–c), with baselines ranging from 1 to 17 weeks, suggests that the observed changes were reliable and consistent, and probably due to the introduction of RFCBT rather than to other factors. Across the majority of baseline periods, the level of depressive symptoms

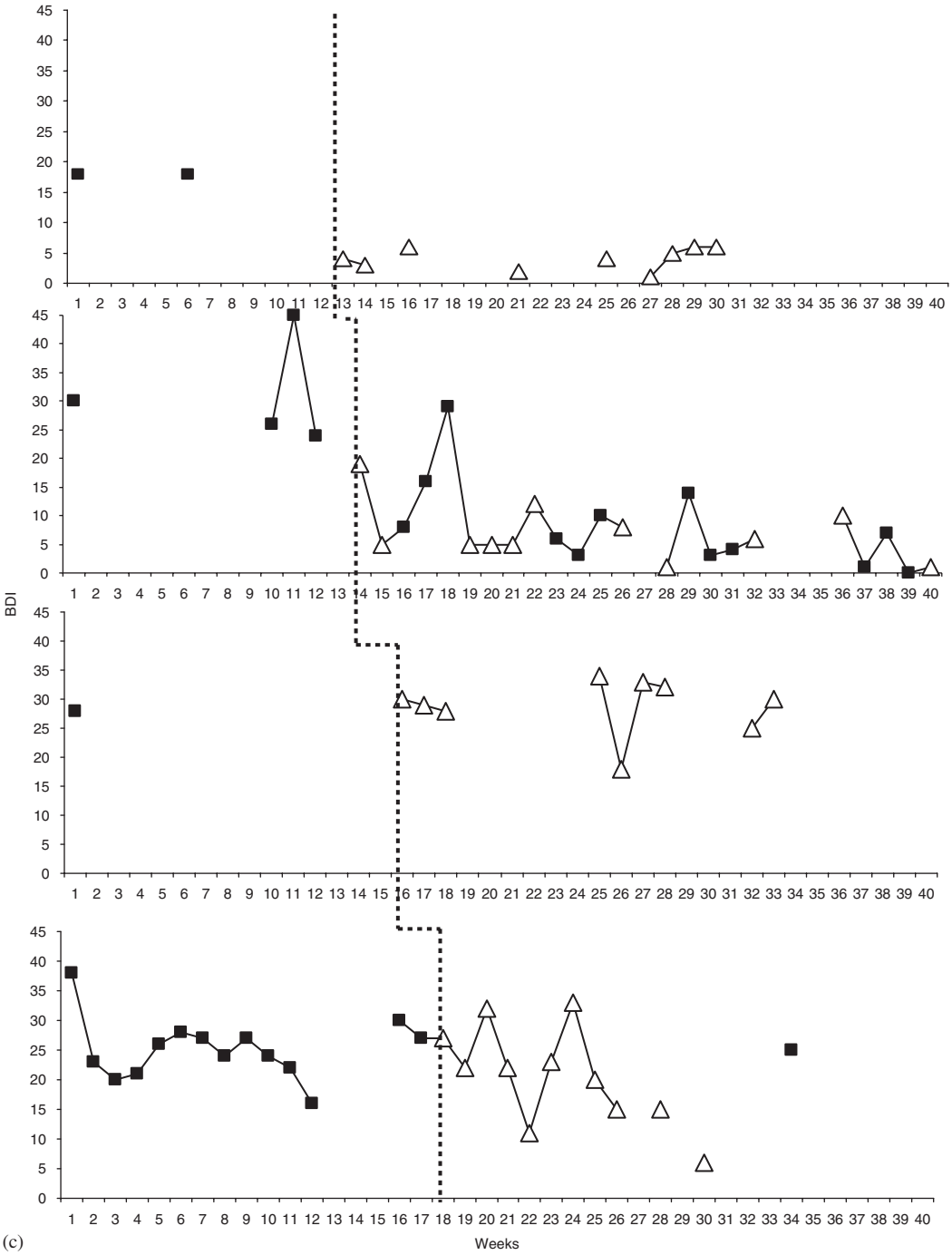

Fig. 1. (*Continued*)

was stable or increasing, and only tended to decrease within 2–6 weeks of treatment commencing, showing a clear decrease in the BDI-II level after treatment was initiated. It is notable that this pattern of reductions in BDI-II scores after the initiation of RFCBT occurred across all but one of the individual patients, despite a varying length of baseline. The latency of treatment effects was consistent with the general observation that CBT starts to show treatment effects after 2–6 weeks and with the first 2–4 sessions of RFCBT being focused on assessment rather than direct intervention.

Discussion

The aim of this study was to act as a preliminary single case series exploration of whether RFCBT may be a potentially efficacious treatment for hard-to-treat residual depression. Despite the patients having relatively high levels of residual symptoms, histories of chronic and recurrent depression and extensive co-morbidity, patients in the clinical case series improved significantly across the course of therapy, with RFCBT showing excellent retention and high response and remission rates. While combined pharmacotherapy and psychological treatments are widely recommended, the additional gains from this approach are often modest. Our findings are therefore very encouraging as they suggest that focusing on one aspect of residual depression—rumination—may yield generalised improvement across depressive symptoms, consistent with the hypothesis that rumination plays a causal role in maintaining residual depression.

While we have to be cautious when comparing between studies, the outcomes for depressive symptoms found for 12 sessions of RFCBT (effect size 2–2.5, remission rates 50%) compare favourably with those found for 20 sessions of CBT by Paykel et al. (1999) (effect size approximately 1; remission rates 25%). Allowing for the more rigorous nature of the Paykel et al. (1999) randomised controlled trial, we are tentative in drawing any firm conclusions from these comparisons, other than that they suggest that RFCBT is worthy of further investigation.

Importantly, RFCBT was found to significantly reduce self-reported rates of maladaptive rumination, consistent with the proposed focus of the treatment. The change in rumination across the course of treatment moved the range of rumination scores reported from that found in currently depressed patients to the range of rumination found in never-depressed participants (for never-depressed groups, Riso et al., 2003 reported $M = 39.3$, $SD = 10.3$; Roberts et al., 1998 reported $M = 38.3$, $SD = 9.7$).[2] Thus, it appears that RFCBT successfully reduced the elevated levels of rumination found in remitted depressed patients, down to more normative levels of rumination. Previous studies have failed to find a reduction in rumination down to normal non-clinical levels in recovered depressed patients (Roberts et al., 1998), even following CBT interventions (Schmaling et al., 2002). Thus, the current study provides the first preliminary data that rumination can be reduced to normal levels by targeting rumination within the context of a BA/CBT intervention.

We note also that RFCBT was effective in reducing a range of co-morbidity, predominantly anxiety disorders, despite not explicitly focusing on these difficulties in treatment. This reduction across co-morbid disorders is consistent with recent suggestions that rumination may be a transdiagnostic process, that is, a cognitive process that plays a causal role in the development of psychopathology across a range of diagnoses (Harvey, Watkins, Mansell, & Shafran, 2004).

The several limitations to this open study include a small group size, the lack of a comparison/control group, and the lack of rater blindness. Without a control group, the observed improvements cannot be unequivocally attributed to RFCBT. However, we note (a) that the pattern of results found on the multiple-baseline design reduces the possibility that a number of other factors were responsible (e.g. maturation, timing, history) and (b) that spontaneous improvement after a prolonged course of antidepressant medication is unlikely in patients with residual depression (Cornwall & Scott, 1997).

In conclusion, these preliminary data indicate that RFCBT may offer benefit for treating a treatment-refractory group in depression. Given that CBT prevented future relapse despite not significantly reducing acute residual symptoms relative to treatment-as-usual, the hope is that if the effects of RFCBT on acute symptoms observed in the current study are replicated under controlled conditions, then RFCBT should produce even larger relapse prevention effects. To this end, we are conducting a pilot randomised controlled trial of RFCBT for residual depression, using the treatment manual from this study, to more rigorously investigate the efficacy of RFCBT as an adjunct to treatment-as-usual.

[2]We also examined the effects of RFCBT on the brooding and reflection subscales of the RRS (Treynor et al., 2003) (although for 7 cases we could only score 3 out of 5 brooding items because we used a different version of the RRS from that analysed by Treynor et al., 2003). There was a significant reduction in brooding, $Z = -2.99$, $p = .0003$ (pre-intervention $M = 15.57$, $SD = 2.88$; post-intervention, $M = 9.29$, $SD = 3.90$) but no significant reduction in reflection, $Z = -1.48$, $p = 0.137$ (pre-intervention $M = 12.29$, $SD = 3.68$; post-intervention, $M = 11.14$, $SD = 3.72$). These results indicate (a) that RFCBT successfully reduced rumination when assessed on a measure less contaminated with depressive symptoms and (b) that RFCBT selectively reduced only the depressogenic subtype of rumination.

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