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How Does Cognitive Therapy Prevent Relapse in Residual Depression? Evidence From a Controlled Trial

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ABSTRACT

This study examined the cognitive mediation of relapse prevention by cognitive therapy (CT) in a trial of 158 patients with residual depression. Scores based on agreement with item *content* of 5 questionnaires of depression-related cognition provided no evidence for cognitive mediation. A measure of the *form* of response to those questionnaires, the number of times patients used extreme response categories ("totally agree" and "totally disagree"), showed significant and substantial prediction of relapse, differential response to CT, and conformity to mediational criteria. CT reduced relapse through reductions in absolutist, dichotomous thinking style. CT may prevent relapse by training patients to change the way that they process depression-related material rather than by changing belief in depressive thought content.

There is encouraging evidence that cognitive therapy (CT; Beck, Rush, Shaw, & Emery, 1979) reduces relapse and recurrence in depression. Outpatients who recover following treatment of major depression by CT show less subsequent relapse or need for treatment than patients who recover with pharmacotherapy and are then withdrawn from antidepressant medication (Blackburn, Eunson, & Bishop, 1986 ; Evans et al., 1992 ; Shea et al., 1992 ; Simons, Murphy, Levine, & Wetzel, 1986). CT following recovery with pharmacotherapy can also reduce subsequent relapse and recurrence (Fava, Grandi, Zielezny, Rafanelli, & Canestrari, 1996 ; Fava, Rafanelli, Grandi, Conti, & Belluardo, 1998). In patients responding only partially to antidepressant medication, the addition of CT to clinical management and continuing antidepressant medication significantly reduced rates of relapse (Paykel et al., 1999).

The processes through which CT reduces depressive relapse are not well understood. Although a number of proposals have been made (see below), their empirical support is limited. To our knowledge, there is no convincing published evidence that any proposed cognitive mediator satisfies the criteria proposed by Hollon and colleagues (DeRubeis et al., 1990 ; Hollon, Evans, & DeRubeis, 1990). These criteria, adapted from more general mediational criteria (Baron & Kenny, 1986), require that (a) rates

of relapse following CT are significantly less than those in a comparison condition; (b) there is a significant treatment effect on the proposed cognitive mediator; (c) posttreatment measures of the cognitive variable predict subsequent relapse (independent of the prediction from posttreatment depression); (d) prediction of relapse by posttreatment measures of the cognitive mediator remains significant when the variable "treatment" is simultaneously entered into the regression; and (e) inclusion of posttreatment measures of the cognitive mediator reduces the extent to which the variable treatment predicts subsequent relapse.

The cognitive model underlying CT for depression suggested that vulnerability was related to underlying dysfunctional attitudes or assumptions (e.g., Beck, Epstein, & Harrison, 1983). From this perspective, reduction in relapse following CT is a consequence of CT reducing those dysfunctional attitudes. This hypothesis has received little empirical support (Barber & DeRubeis, 1989); when CT has produced significantly better long-term outcomes than pharmacotherapy, the two treatments often do not differ on posttreatment measures of dysfunctional attitudes such as the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978 ; see, e.g., Simons, Garfield, & Murphy, 1984). The National Institute of Mental Health Treatment of Depression Collaborative Research Program found no evidence for specific effects of CT on DAS total or perfectionism subscale scores, but there were specific effects on need for social approval subscale scores (Imber et al., 1990).

Segal, Gemar, and Williams (1999) reported that, when the DAS was administered following a dysphoric mood induction, posttreatment DAS scores were significantly less following CT than pharmacotherapy. Such scores also predicted subsequent relapse. However, these findings were based on small numbers of patients not randomized to treatment condition.

The reformulated learned helplessness model of depression (Abramson, Seligman, & Teasdale, 1978) suggested that vulnerability to depression involves a depressotypic attributional style: a tendency to attribute bad events to internal, stable, and global causes and good events to external, unstable, and specific causes. The Attributional Style Questionnaire (ASQ; Peterson et al., 1982) provides a measure of depressotypic attributional style. Hollon et al. (1990) reported that, in Evans et al.'s (1992) clinical trial comparing CT and pharmacotherapy for depression, ASQ satisfied four of the five mediational criteria described above, and they concluded that attributional style "emerges as a potential mediator of cognitive therapy's prophylactic effect on posttreatment relapse/recurrence" (Hollon et al., 1990 , p. 126). However, more recent analyses have suggested that "strictly speaking [the ASQ] does not meet statistical criteria for mediation" (S. D. Hollon, personal communication, July 5, 1999).

DAS- and ASQ-related accounts suggest that CT acts through changes in "deep" schematic cognitive structures or characteristic interpretative cognitive styles. By contrast (see also Persons, 1993), Barber and DeRubeis (1989) suggested that the key therapeutic process was not so much cognitive change as the acquisition of compensatory skills: "cognitive therapy does not reduce the tendency for depressives to generate negative thoughts in distressing situations, but rather it inculcates a set of skills that helps them deal with these thoughts when they do occur" (Barber & DeRubeis, 1989 , p. 450). We know of no published evidence evaluating this suggestion against the Baron and Kenny (1986) criteria.

Teasdale and colleagues (Teasdale, 1997a , 1997b ; Teasdale & Barnard, 1993 ; Teasdale, Segal, & Williams, 1995) have suggested a model of therapeutic change that combines aspects of the cognitive change and compensatory skills accounts. It is suggested that, in CT, patients repeatedly approach depressive symptoms and problematic situations with different, more functional, *cognitive sets* in place. The creation and storage of representations encoding such alternative sets, it is suggested, mediate the therapeutic effects of CT. For example, Teasdale (1985) suggested that the perception of depression as highly aversive and uncontrollable leads to "depression about depression," and that CT reduces depression about depression by increasing the perceived controllability and reducing the perceived

aversiveness of depression. It has also been suggested that CT involves a shift in the cognitive set with which negative thoughts are approached; rather than being approached as self-evident truths or aspects of the self, thoughts are approached as "events in the mind" that may or may not correspond to reality (Moore, 1996 ; Teasdale et al., 1995). Interpreted through such a "decentered" set, negative thoughts and feelings are less likely to lead to depression, and effects of CT could be mediated through increasing meta-cognitive awareness in this way. As yet, no published evidence has evaluated shifts in cognitive set as mediators of the relapse prevention effects of CT.

The present study examined predictions from the above accounts of cognitive mediation within a clinical trial that evaluated CT in patients who had failed to respond adequately to antidepressant medication (Paykel et al., 1999). In demonstrating a significant treatment effect of CT in reducing relapse, this trial met the first of the five mediation criteria, described above, and so provided an opportunity to examine potential cognitive mediators with respect to the other criteria. The following specific cognitive variables were examined.

- ASQ (Peterson et al., 1982): The ASQ was included in view of Hollon et al.'s (1990) findings suggesting that changes in attributional style might mediate the effects of CT in reducing relapse.

- DAS (Weissman & Beck, 1978)–Need for Social Approval subscale (Imber et al., 1990) report of modality-specific effects of CT on this subscale of the DAS suggested that it was worth examining further the potential mediating role of changes in this variable.

- Perceived uncontrollability of depression: A questionnaire measure was developed to examine whether CT works through changes on this variable, as suggested by Teasdale (1985 ; see also Teasdale & Barnard, 1993 , p. 231).

- Characterological self-blame for depression: A questionnaire measure of this variable was developed to examine whether CT acts through reducing "depression about depression" (Teasdale, 1985) by reducing attributions for depression to characterological weakness (see Teasdale & Barnard, 1993 , p. 231).

- Metacognitive awareness: A questionnaire measure of metacognitive awareness was developed to examine whether CT works through facilitating a decentered relationship to negative thoughts and feelings in which they are viewed as transient events in the mind, rather than accurate reflections of reality or aspects of self (e.g., Moore, 1996 ; Teasdale, 1997a , 1997b ; Teasdale et al., 1995).

In summary, measures related to five potential cognitive mediators of the effects of CT on relapse were included in a large treatment trial of CT for residual depression to investigate whether any of them met mediational criteria.

Method

Design

The methodology of the clinical trial has been described by Paykel et al. (1999) . At two treatment sites (Cambridge and Newcastle, England), 158 patients with recent major depression, partially remitted with treatment by antidepressant medication, were randomized to receive antidepressant medication and clinical management either alone or together with CT. In a 20-week treatment phase, all of the patients received drug continuation and clinical management, and one group received, additionally, 16 CT sessions. Over a 48-week follow-up, all of the patients received continuation and maintenance

antidepressant medication, and the CT group received two booster sessions of CT. Assessments of clinical state were made before treatment, monthly during treatment, and bimonthly during follow-up. Assessments of cognitive variables were made before treatment, after 8 weeks, and at the end of 20 weeks of treatment.

Participants

Participants were psychiatric outpatients, age 21 to 65 years, satisfying *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev. [*DSM—III—R*]; American Psychiatric Association, 1987) criteria for major depression within the last 18 months but not in the last 2 months, with residual symptoms scoring at least 8 on the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and 9 on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Patients were excluded if there was a history of bipolar disorder, cyclothymia, schizoaffective disorder, drug or alcohol dependence, persistent antisocial behavior or repeated self-harm, *DSM—III—R* dysthymia with an onset before age 20 years, borderline personality, learning disability, organic brain damage, or any other primary Axis I disorder at the time of the index illness. Also excluded were patients currently receiving formal psychotherapy or who had previously received CT for more than five sessions.

Patients had to be taking antidepressant medication for at least the previous 8 weeks, with 4 or more weeks at a daily dose at least equivalent to 125 mg amitryptiline, and higher levels unless there were definite current adverse effects or patient refusal to increase dose. In fact, most participants were receiving much larger doses for longer periods than this.

Measures HRSD.

The 17-item HRSD (Hamilton, 1960) is a widely used interview-based measure of severity of depressive symptomatology with acceptable reliability and validity (reviewed by Rabkin & Klein, 1987).

BDI.

The BDI (Beck et al., 1961) is a widely used 21-item self-report measure of the severity of depressive symptomatology with acceptable psychometric properties (reviewed by Rabkin & Klein, 1987).

Relapse.

Two alternative relapse criteria were used: (a) *DSM—III—R* criteria for major depressive disorder (American Psychiatric Association, 1987) for at least 1 month (2 weeks longer than *DSM—III—R* criteria require) and, at two successive face-to-face assessments at least 1 week apart, patients must have met both the severity criteria for major depression and scored 17 or more on HRSD; or (b) during follow-up, residual symptoms persisted between two successive ratings 2 months apart, reaching HRSD scores of at least 13 on both occasions, and distress or dysfunction for which the witholding of additional active treatment was no longer justified.

ASQ.

The ASQ (Peterson et al., 1982) presents scenarios with six positive and six negative outcomes.

Participants indicate the causes of the outcomes and rate those causes on three 7-point scales: External/Internal, Unstable/Stable, and Specific/Global. Following Hollon et al. (1990) , depressotypic attributional style was measured by summing scores separately for positive and negative items and subtracting the negative composite from the positive composite. More negative scores on the overall composite indicate more depressotypic attributional style.

DAS–Need for Social Approval subscale (DAS—Approval).

DAS (Weissman & Beck, 1978) Form A has 40 statements to which participants respond on 7-point scales (*totally agree* to *totally disagree*). The Need for Social Approval subscale (DAS—Approval) emerged from factor analyses (Imber et al., 1990 ; details were kindly provided by Paul A. Pilkonis) and has 11 items related to dependence of happiness and personal worth on approval from others. Higher scores on DAS—Approval reflect more dysfunctional attitudes. Imber et al. (1990) reported Cronbach's alpha of .82 for this subscale.

Perceived Uncontrollability of Depression (UNCONTROL).

On this 10-item scale, developed for this study, patients indicate how much they perceive that they have no control over depression (see the Appendix). The scale uses the 7-point DAS response format. Cronbach's alphas in two community samples of 109 and 92 members of a self-help organization for people with depression (kindly made available through the Depression Alliance, London, England) were, respectively, .94 and .93. Higher scores on UNCONTROL reflect greater perceived uncontrollability of depression.

Characterological Self-Blame for Depression (BLAME).

On this 10-item scale, developed for this study, patients indicate how much they blame their depression on their personal inadequacy (see the Appendix), using the 7-point DAS response format. Cronbach's alphas in community samples of 121 and 95 members of a self-help organization for people with depression (kindly made available through Depression Alliance) were, respectively, .93 and .92. Higher scores reflect greater self-blame.

Metacognitive Awareness Questionnaire (MAQ).

On this 9-item scale, developed for this study, patients reported how much they saw that their negative thoughts and feelings when depressed might not reflect actual realities (see the Appendix). The scale uses the same 7-point response format as the DAS. Cronbach's alpha for the 20-week MAQ administration to 139 patients in this study was .71. Higher MAQ scores reflect greater metacognitive awareness.

Assessments

Interview assessments of depression and clinical state were made by experienced research psychiatrists, at baseline, every 4 weeks to 20 weeks, and every 8 weeks thereafter. Cognitive measures were administered at baseline, 8 weeks, and 20 weeks by graduate psychologists. Assessors were unaware of treatment group, and patients were asked to conceal their treatment assignment.

Treatment Clinical management and drug continuation (all patients).

Patients were seen every 4 weeks during the treatment phase and every 8 weeks during follow-up.

Interviews were based on the clinical management described by Elkin et al. (1989) and lasted approximately 30 min. Symptoms were rated, limited support was provided, and drugs were prescribed. Specific CT techniques were prohibited.

CT.

Patients had 16 sessions of CT during the 20-week treatment period, plus two booster sessions during follow-up. CT was similar to that used in Beck et al. (1979) , with modifications described in a manual (see Scott, 1998). Recent elaborations of CT (Beck & Freeman, 1994) were permitted, including techniques for engagement in CT and schema-focused approaches to core unconditional beliefs. Therapists had diplomas in CT and at least 4 years' CT experience.

Audiotapes of randomly selected sessions were rated on the Cognitive Therapy Rating Scale (CTRS; Young & Beck, 1990) by an independent rater to ensure fidelity and competence. Median CTRS score was 54, with no rating below 39, the accepted threshold level, and no significant differences between therapists. Randomly selected psychiatrists' clinical management sessions rated on the CTRS yielded ratings high on nonspecific items, such as professionalism and empathy, but low on CT strategies; median score was 19 with no tape rated >24.

Results

Baseline Characteristics of Treatment Groups

Table 1 shows that CT and control groups were similar on initial variables.

Correlates of New Cognitive Measures

None of the new cognitive measures (UNCONTROL, BLAME, and MAQ) correlated with HRSD (all r s < ±.1). BLAME correlated .70 (df = 144, p = .000) with DAS and −.54 (df = 128, p = .000) with ASQ, suggesting that this variable reflected a dysfunctional attitude associated with a stable, global, and internal attributional style. Correlations of UNCONTROL and MAQ with BDI, DAS, and ASQ were all significant (largest p = .006) and fell in the range ±.38 in the expected directions.

Clinical Outcome: Relapse and Symptom Ratings

As reported by Paykel et al. (1999) , comparison of time to relapse in CT and control groups showed a significant (p = .02) increase in survival (nonrelapse) in the CT group. CT reduced risk of relapse by approximately 40% of the hazard in the control group; for example, in an intent-to-treat analysis, cumulative relapse rates at 68 weeks for control and CT groups were, respectively, 47% and 29%.

As previously reported (Paykel et al., 1999), 20-week HRSD and BDI scores showed no significant differences between CT and control groups. Adjusted 20-week means in control and CT groups for HRSD were, respectively, 9.40 and 8.58, and for BDI, 16.06 and 13.46. Although, compared with controls, CT significantly reduced relapse, it did not significantly reduce posttreatment depression scores.

Mediational Analysis: Analytic Strategy

Application of Baron and Kenny's (1986) mediational criteria by Hollon and colleagues (1990) , described above, was to an acute treatment trial in which patients were recruited in episode, were

treated, were assessed posttreatment, and were then followed up to determine whether or not they relapsed.

The situation in the present study was different. Because patients started the trial in partial recovery, they could relapse during the main treatment phase: 21 patients had onsets of relapse before the Week 20 posttreatment assessment. When these patients were eliminated from regression analyses, the difference in survival (nonrelapse) between CT and control groups was no longer significant: Cox proportional hazard regression analyses showed nonsignificant effects of treatment on relapse, whether the binary variable *treatment* (CT vs. control) was entered on its own (Wald = 0.62, $p = .43$, hazard ratio = .75) or with 20 week HRSD score (Wald = 0.45, $p = .50$, hazard ratio = .79). (For all Wald statistics, $df = 1$.) That is, there was no longer a significant treatment effect on relapse to explain. For this reason, we used Week 8 as the posttreatment assessment included in mediational analyses. Week 8 assessments were less than halfway through the main treatment phase and reflected effects of the initial phase, rather than complete course, of CT.

After eliminating patients with relapse onset before 8 weeks or those not conforming to treatment protocol (e.g., <4 sessions CT), a sample of 140 remained. For this sample, a Cox regression, entering treatment and Week 8 HRSD score, showed significant relapse prevention for CT (Wald = 5.02, $p = .025$, hazard ratio = .52); there was a significant effect of CT on relapse to explain.

Using the intermediate 8-week assessment point in mediational analyses required slight modifications to the mediational criteria proposed by Hollon and colleagues. Our analytic strategy retained the same underlying principles:

- Identify cognitive variables significantly predicting relapse, independent of depression, from both Week 0 and Week 8 assessments. (Week 0 measures were included as a further test of the predictive power of variables.)
- Identify which of the cognitive variables identified in No. 1 differ significantly between the two treatment groups on Week 8 measures.
- Identify which of the variables identified in No. 2 significantly predict relapse from Week 8 measures when treatment is also in the regression.
- Identify which of the variables identified in No. 3 reduce prediction of relapse from treatment condition when their Week 8 values are in the regression. Cognitive variables satisfying all steps are candidate cognitive mediators.

Identification of Cognitive Predictors of Relapse

Cox proportional hazard regression analyses examined prediction of time to relapse from Week 0 and Week 8 values of each cognitive variable for the variable both on its own and together with concurrent BDI. Analyses excluded 10 patients who failed to conform to treatment protocol. For Week 8 analyses, a further 8 patients who experienced onset of relapse prior to the 8-week assessment were eliminated, leaving a sample of 140.

High BDI scores, alone, were predictive of early relapse: Week 0, Wald = 6.82, $p = .0090$; Week 8, Wald = 24.06, $p = .0000$. Table 2 shows prediction of relapse from cognitive variables. None of the cognitive variables significantly predicted relapse, independent of depression, at both Week 0 and Week 8.

Exploratory analyses of ASQ.

The most unexpected finding in Table 2 was that Week 0 ASQ scores reflecting high depressotypic attributional style predicted less, rather than more, risk of relapse. To explore this finding, we examined original ASQ paper copies from a group of relapsing and a group of nonrelapsing patients. This examination suggested relapse was associated with use of extreme scores (1 or 7), and this association was true for both extremely undepressotypic and extremely depressotypic patterns of attribution.

To explore this possibility systematically, we created scores, separately for good and bad outcomes, reflecting how often an individual gave scores of 1 on each of the dimensions of internality, stability, and globality (i.e., endorsement of, respectively, "totally due to other people or circumstances," "will never again be present," and "influences just this particular situation") and how often an individual gave scores of 7 on each of these dimensions (i.e., endorsement of, respectively, "totally due to me," "will always be present," and "influences all situations in my life"). After logarithmic transformations to normalize, 7 of these 12 variables significantly predicted relapse, [1] in each case more extreme responses predicting earlier relapse: Good 7 Internal (i.e., how often an individual used a score of 7 on the Internality scale for items with good outcomes): Wald = 6.96, *p* .0083; Good 7 Stable, Wald = 6.55, *p* = 0.11; Good 7 Global, Wald = 9.45, *p* = .0021; Bad 7 Internal, Wald = 10.38, *p* = .0013; Bad 7 Global, Wald = 8.73, *p* = .0031; Bad 1 Stable, Wald = 14.66, *p* = .0001; and Bad 1 Global, Wald = 7.30, *p* = .0069. All predictions were significant with Week 0 BDI in the regression.

Strikingly, in these data, extreme undepressotypic responding predicted early relapse as much, or more, than extreme depressotypic responding. For example, the most significant prediction (Wald = 14.66, *p* = .0001) was from responses indicating that causes of bad outcomes "will never again be present."

Intercorrelations between (log-transformed) variables reflecting total number of 1 scores for bad outcomes, 1 scores for good outcomes, 7 scores for bad outcome, and 7 scores for good outcomes were all positive and significant, suggesting a general tendency within an individual to respond extremely.

Testing the extreme response hypothesis.

The exploratory ASQ analyses suggested that extreme responses to depression-relevant material were predictive of early relapse. As a planned test of this hypothesis, the total number of extreme (1 or 7) responses given by each patient to the remaining four cognitive variables (DAS—Approval, UNCONTROL, BLAME, and MAQ) was calculated; all of these variables used the same 7-point response format ranging from "totally agree" to "totally disagree" (ASQ was not included in the composite score used for hypothesis testing, as it had been used in the generation of the hypothesis to be tested). After logarithmic transformation to normalize, this sum-of-extreme-responses variable significantly predicted time to relapse both at 0 weeks (Wald = 11.07, *p* = .0009) and at 8 weeks (Wald = 11.18, *p* = .0008). These predictions remained significant when concurrent BDI scores were also entered: Week 0, Wald = 8.36, *p* = .0038; Week 8, Wald = 8.62, *p* = .0033. In all cases, more extreme responses predicted earlier relapse.

Relapse in patients showing any extreme score was more than 2.5 times the rate in patients with no extreme scores. On Week 0 measures, patients with no extreme scores showed 17% (5/30) relapse, whereas those with any extreme score showed 44% (51/116), χ^2 (1, *N* = 146) = 7.51, *p* = .0061; on Week 8 measures, patients with no extreme scores showed 15% (6/39) relapse, whereas those with any extreme score showed 42% (37/89), χ^2 (1, *N* = 128) = 8.34, *p* = .0039.

Principal-components analysis.

The above results suggested a relatively general tendency to think in extreme, absolute ("black-and-white") terms across several different depression-related contents for both functional and dysfunctional responses. To test this suggestion, we entered into principal-component analyses, measures reflecting extreme positive and extreme negative responding on, respectively, UNCONTROL, BLAME, DAS—Approval, and MAQ. (Extreme positive responses = number of "totally agree" responses to functional items + number of "totally disagree" responses to dysfunctional items; extreme negative responses = number of "totally agree" responses to dysfunctional items + number of "totally disagree" responses to functional items.) Extracting unrotated factors with eigenvalues > 1, these analyses yielded very similar two-factor solutions (see Table 3) for both Week 0 and Week 8 variables. A first factor accounted for 40% (Week 0) and 38% (Week 8) of variance. All eight measures loaded positively on this factor, consistent with it being a general factor of extreme responding. A second factor accounted for 23% (Week 0) and 24% (Week 8) of variance; at both times, all four positive extreme measures loaded positively, whereas all four negative extreme scores loaded negatively, consistent with this factor reflecting a contrast between positive versus negative extremity. [2]

Factor 1 and Factor 2 scores were entered separately into Cox regression analyses. At Week 0 (Wald = 9.15, $p = .0025$) and Week 8 (Wald = 7.01, $p = .0081$), Factor 1 scores significantly predicted relapse, higher scores predicting earlier relapse. By contrast, Factor 2 scores failed to predict at either Week 0 (Wald = 0.55, $p = .46$) or Week 8 (Wald = 0.33, $p = .57$). In summary, principal-component analyses supported the existence of a relatively general tendency to respond in an absolutist dichotomous style that was predictive of early relapse.

Summary of cognitive predictor analysis.

A measure (sum of extreme responses) reflecting the tendency to respond extremely in an absolutist black-and-white thinking style to depression-related material significantly predicted time to relapse from both Week 0 and Week 8, when entered on its own or together with concurrent BDI. The extent of prediction was substantial, with more extreme responding predicting earlier relapse.

Effects of CT on Posttreatment Measures of Cognitive Predictor

Treatment effects on sum of extreme responses were examined using Week 8 measures. Patients with onset of relapse prior to the 8-week assessment were eliminated from these analyses. Median Week 8 scores for sum of extreme responses were 1.00 ($n = 65$, interquartile range = 5.00) for CT and 6.00 ($n = 63$, interquartile range = 13.00) for controls. Analysis of covariance on log-transformed Week 8 sum of extreme responses, with log-transformed Week 0 sum of extreme responses as covariate, yielded a significant effect of treatment group, $F(1, 124) = 9.83$, $p = .002$, which remained significant with Week 8 BDI as a further covariate, $F(1, 123) = 9.58$, $p = .002$ [3] (in fact, there was no significant treatment effect on Week 8 measures of depression for either BDI or HRSD).

Prediction of Relapse From Posttreatment (8 Week) Measures of Sum of Extreme Responses With Treatment Also Entered Into the Regression

When we entered (log-transformed) Week 8 sum of extreme responses and the categorical variable, treatment (CT vs. control), together into a Cox proportional hazards regression, sum of extreme responses continued significantly to predict time to relapse (Wald = 8.83, $p = .0030$). With Week 8 BDI added as well, sum of extreme responses still predicted significantly (Wald = 7.26, $p = .0070$). Within the logic of the analytic strategy used, these findings suggest that the relation between sum of extreme responses and time to relapse is more than just the epiphenomenal consequence of each of these variables being affected independently by treatment condition (Hollon et al., 1990 , p. 126).

Prediction of Relapse From Treatment With Posttreatment (8 Week) Measures of Sum of Extreme Responses Also Entered Into the Regression

The categorical variable, treatment (CT vs. control), significantly predicted time to relapse when entered into a Cox regression together with Week 8 HRSD score (Wald = 5.02, $p = .025$; CT significantly increased survival). When logarithmically transformed Week 8 sum of extreme responses was also entered, the effect of treatment was no longer significant (Wald = 1.92, $p = .17$), whereas the effect of sum of extreme responses still was (Wald = 8.57, $p = .0034$). However, the reduction in prediction from treatment was not statistically significant ($t < 1$), reflecting the fact that, initially, this prediction was not strong. Accepting the limitation of this nonsignificant finding, this pattern of results is otherwise consistent with effects of CT on relapse being mediated, at least partly, through its effects on Week 8 sum of extreme responses.

Summary of Mediational Analysis

A measure reflecting an absolutist, dichotomous, black-and-white style of processing depression-related material satisfied four out of five criteria as a potential cognitive mediator of the observed effects of CT in preventing relapse/recurrence. This suggests that changes in this style mediate at least some of those prophylactic effects or are close markers of related changes in unmeasured variables that are the actual mediating mechanism.

Next to Extreme Scores

Sum of extreme responses reflected use of the categories "totally agree" and "totally disagree." The very extreme nature of these categories suggested that this measure reflected an absolutist black-and-white style of thinking ("the tendency to place all experiences in one of two opposite categories," Beck et al., 1979 , p. 14). To test the hypothesis that the extremity of the response categories contributing to sum of extreme responses was the essential feature giving this variable mediational status, we created new variables by summing the number of times "next to extreme scores" (i.e., "agree very much" and "disagree very much") were used in DAS—Approval, UNCONTROL, BLAME, and MAQ. Sum of next to extremes did not predict relapse from either Week 0 (Wald = 0.46, $p = .50$) or Week 8 (Wald = 0.18, $p = .67$). Further, CT and control groups did not differ on sum of next to extremes at Week 8 (CT mean = 10.68, $SD = 6.33$; control mean = 9.80, $SD = 6.21$), t (133) = .81, $p = .42$. In contrast to sum of extreme responses, sum of next to extremes did not conform to mediational criteria.

Comparison With Nondepressed Controls

Scores on DAS—Approval, UNCONTROL, and BLAME (but not MAQ) were available from 27 control participants with no history of past or current treatment for depression who were currently not depressed (Center for Epidemiologic Studies Depression Scale [Radloff, 1977] scores < 16), matched for age (mean age = 43.8 years, $SD = 15.6$) and gender distribution (13 female) with the patient sample. For this sample, "totally agree" and "totally disagree" responses for, respectively, functional and dysfunctional items across the three questionnaires were counted and compared with corresponding data from the patients. After log transformation to normalize, analysis of variance with group (patients vs. controls) as a between-subjects variable and response type (agreement with functional vs. agreement with dysfunctional vs. disagreement with functional vs. disagreement with dysfunctional) as a within subject variable showed that, overall, controls actually gave more extreme responses than did patients (respectively, M s = 0.80 and 0.54), F (1, 171) = 8.96, $p = .003$, but this main effect was qualified by a Group × Response Type interaction, F (3, 513) = 61.92, $p = .000$. Controls gave more extremes than did patients for agreement with functional items (respectively, M s = 0.27 and 0.066), t (171) = 5.38, p

= .000, and for disagreement with dysfunctional items (respectively, *M* s = 1.10 and 0.35), *t* (171) = 9.03, *p* = .000. Conversely, patients gave more extremes than did controls for agreement with dysfunctional items (respectively, *M* s = 0.44 and 0.10), *t* (171) = 3.89, *p* = .000, and for disagreement with functional items (respectively, *M* s = 0.22 and 0.12, *t* (171) = 2.00, *p* < .05.

Discussion

When scored in terms of agreement with item *content,* none of the five questionnaire measures of depression-related cognition in the present study met criteria as mediators of the effects of CT in preventing relapse. By contrast, a measure of the *form* of response to those questionnaires–the number of times patients used the most extreme response categories ("totally agree" and "totally disagree")–showed significant and substantial prediction of relapse, differential response to CT, and conformity to mediational criteria.

Before discussing our positive findings, we note that the negative findings from scores based on questionnaire item content are consistent with the failure of such measures as cognitive mediators in previous studies, as reviewed in the introduction. Limitations of such measures have been widely noted (e.g., Barber & DeRubeis, 1989 ; Hollon, 1992 ; Persons, 1993 ; Teasdale, 1997a ; Whisman, 1993).

Turning to the positive findings, the suggestion that relapse prevention was mediated through changes in an absolutistic, dichotomous thinking style is consistent with the importance of this cognitive error in the cognitive model of depression: "The meanings that flood [depressed people's] consciousness are likely to be extreme, negative, categorical, absolute and judgmental. The emotional response, thus, tends to be negative and extreme" (Beck et al., 1979 , p. 14). Empirical evidence supports the significance of this thinking style. For example, Litinsky and Haslam (1998) found that, compared with nonsuicidal patients, suicidal patients showed more narrowly defined dichotomous thinking involving polarized possibilities, but the groups did not differ on weaker forms of dichotomous thinking involving nonexclusive or nonbinary alternatives.

How are the present findings to be explained? The aim of CT is usually seen as reducing belief in depressive thoughts or dysfunctional assumptions (i.e., changes in thought *content*). From this perspective, changes in the *form* of processing depressive information (e.g., shifting from an "automatic" mode of processing to a more reflective, "controlled" mode) are seen as secondary to the goal of changing belief in thoughts. Our data suggest an alternative view: These shifts of cognitive mode, rather than being merely the means to the end of changing belief, may actually be the primary mechanism through which the relapse prevention effects of CT are achieved.

To expand on this suggestion, we consider, first, what extreme responding in the present study is, specifically, measuring, and, second, how the effects of CT on this measure are related to relapse prevention.

Developmentally, all-or-none thinking is normative in very early to middle childhood (Harter, 1999 , p. 36). As children mature, all-or-none thinking is largely superseded by less extreme, moderated, thinking styles. This progression can be understood within the kind of two-stage models of social cognition (e.g., Bargh, 1994) proposed to account for stereotyping (e.g., Devine, 1989) and social inference (e.g., Gilbert, 1989). According to such models, much adult cognition involves an initial, relatively automatic, processing stage that depends on rapid access to precomputed schematic representations. The cognitive products of this initial "quick-and-dirty" stage are subject to reappraisal in a secondary, nonautomatic stage that depends on the availability of controlled processing resources and is likely to yield more moderate thinking patterns. Uncorrected schematic thinking might be viewed as cognitive

impulsivity.

Applied to the present findings, these considerations suggest that, in vulnerable individuals who are already at least mildly depressed, extreme responses ("totally agree" or "totally disagree") to depression-related questionnaire items are the "tip of the iceberg" reflecting underlying activity of mood-dependent, developmentally early, depressogenic schematic processing, uncorrected by subsequent reappraisal. Such schematic processing, it is suggested, is an integral part of self-perpetuating patterns of negative thinking (e.g., Teasdale, 1997b ; Teasdale & Barnard, 1993 ; Teasdale et al., 1995) that mediate relapse to depression. Within this account, it is, specifically, very extreme scoring–involving dichotomous, all-or-none thinking–that marks the activity of uncorrected, developmentally early, dysfunctional, thinking patterns; less extreme responses (e.g., "agree very much") are assumed to reflect more mature processing involving some degree of reappraisal.

The above account explains the predictive relationship between sum of extreme scores and subsequent relapse observed in the present study: Dichotomous thinking with respect to depression-related material was a marker of the ease of activation of relapse-engendering processing. The suggestion that such extreme responding is a marker of rapid, automatic processing is consistent with previously reported negative correlations between attitude extremity and response latency (e.g., Bargh, Chaiken, Govender, & Pratto, 1992). Sheppard (2000) recently demonstrated the same relationship for extreme scoring on the DAS. The failure of next-to-extreme scores to predict relapse or show treatment effects is consistent with such scores marking more mature, less automatic processing.

Turning to the mediation of relapse prevention, we suggest that the process of CT involves repeated experiences in which patients are led to disengage from self-perpetuating, depressogenic processing configurations, dominated by automatic access to precomputed dysfunctional schematic representations. Such disengagement depends on the use of volitional controlled processing strategies, as, for example, when identifying and "answering" negative automatic thoughts. Repeated experiences of interrupting habitual, schema-driven, depressogenic processing routines and replacing this mode of processing by an intentional, controlled processing mode, make it more likely that when patients subsequently encounter depression-related material, the controlled processing mode will preempt more habitual automatic processing routines. In this way, the chances of reinstating depressogenic thinking patterns at times of potential relapse are reduced, and relapse is prevented. Assuming that, in the present study, extreme responding was an indicator of a dysfunctional, relatively automatic, processing mode, this account explains our findings that CT reduced sum of extreme responses and risk of relapse, and that reduction of relapse was mediated by processes marked by reduction in extreme responding to depression-related material.

The above account is strikingly similar to previous suggestions by Ingram and Hollon (1986) :

fostering a switch out of the automatic mode to a more deliberate controlled mode is a critically important effect of cognitive therapy. ... Cognitive therapy relies heavily on helping individuals switch to a controlled mode of processing that is metacognitive in nature and focuses on depression-related cognition. ... The long-term effectiveness of cognitive therapy may lie in teaching patients to initiate this process in the face of future stress. (p. 272)

Our proposals are also reminiscent of Barber and DeRubeis's (1989) suggestion that CT works by teaching patients to reappraise, or have "second thoughts," in relation to depressive cognition. Barber and DeRubeis described this process in terms of the deployment of compensatory skills. Thinking of this process more generally in terms of shifting cognitive modes, as in our account, emphasizes a central mechanism common to a range of interventions rather than the specifics of particular compensatory skills.

If, in the present study, extreme responding was an indicator of a dysfunctional, relatively automatic, processing mode, the question arises: How clinically significant were the effects of CT in reducing this mode of processing? Kendall, Marrs-Garcia, Nath, and Sheldrick (1999) described use of normative comparisons as a method to evaluate the clinical significance of treatment effects. In the present context, this procedure is not straightforwardly applicable with respect to the effects of CT on extreme responding. Comparative data from the nondepressed control sample suggested that extreme responding was only higher in the patient group for negative responses (agreement with dysfunctional items and disagreement with functional items), with the control group actually showing more extremes for positive responses (agreement with functional items and disagreement with dysfunctional items). Such data suggest that the interpretation of extreme positive responses in patients with depression is not straightforward. Predictive data suggested that such responses were "pathological" in that they predicted relapse just as much as extreme negative responses. However, comparison with nondepressed controls suggested that extreme positive responses are not pathological in the nondepressed controls in that they were more common in them than in patients. The significance of extreme positive responses to depression-related material appears to be different in the two groups. Actually, this is not surprising; for a patient who has been continuously depressed for a year, who has failed to respond to antidepressant medication, and who still experiences substantial residual depression, to answer "totally agree" to an item such as "I have a sense of control over depression" seems to have a different meaning than the same response from a nondepressed control participant. Extreme positive responding in depressed patients may reflect dysfunctional, defensive thinking, as predictive of relapse as more negative extreme responding.

It should be noted, with respect to the need to replicate the present findings, that the sample we studied, patients with residual depressive chronicity who were maintained on antidepressant medication, was different from other CT trials. Those with a chronic condition might show extreme thinking ("always" or "never") especially frequently. For similar populations, we would expect the sum of extreme responses to act as a marker of the dysfunctional processing style we have described and to conform to the mediating pattern shown in our study. However, it may not act in the same way for other types of patients.

Our findings suggest that CT may prevent relapse by allowing patients to disengage from an habitual, dysfunctional, cognitive mode at times of potential relapse. Relatedly, our findings suggest that interventions that focus on changing patients' relationship to their dysfunctional thoughts and feelings, rather than attempting to modify thought content or belief, would be useful (Teasdale, 1999 ; Teasdale et al., 1995). Mindfulness-based CT systematically trains patients to operate in an intentional rather than automatic cognitive mode, to be more aware of unwanted thoughts and feelings, and to relate to them as "events in the mind" without necessarily attempting to modify their content. This program is effective in reducing relapse in recurrent depression (Teasdale et al., 2000). The success of such programs suggests that future developments in CT might profitably include procedures explicitly designed to change the mode in which dysfunctional cognitions are processed as well as, or instead of, procedures focused on changing thought content.

APPENDIX A

Items of Perceived Uncontrollability of Depression Questionnaire (UNCONTROL), Characterological Self-Blame for Depression Questionnaire (BLAME), and Metacognitive Awareness Questionnaire (MAQ)

Items with asterisks indicate reverse-keyed items.

UNCONTROL

- * I have a sense of control over depression.
- * When I'm depressed, there are things I can do to change how I feel.
- * There *is* something I can do about depression.
- * I know what to do to make myself feel less depressed.
- * When I'm depressed, by changing the way I think I can change the way I feel.
- I have no control over my depression.
- When I'm depressed, I can do nothing except wait for it to go.
- I feel hopeless about ever mastering depression.
- When I'm depressed, there is nothing I can do to change the way I feel.
- I feel hopeless about dealing with depression.

BLAME

- It's my own fault that I get depressed.
- I get depressed because there is something basically wrong with me as a person.
- I am to blame for getting depressed.
- I get depressed because I am not as good as other people.
- My depression is a sign of my inadequacy.
- I get depressed because I am a failure.
- The main cause of my depression is me.
- I have no one to blame but myself for being depressed.
- I get depressed because I am weak.
- My depression will not change until I change as a person.

MAQ

- If something has upset me, I try to put my judgments on hold for a while.
- When I get low, my feelings show things in their true light.
- When I get low, I remind myself that I may be seeing things as more negative than they really are.

- * I trust my own way of seeing things when I feel depressed.
- If I am feeling low, I know my thoughts and feelings are not necessarily realistic.
- * When I am down, I can see things as they really are.
- When I am depressed, I am aware that there could be other ways of viewing the situation.
- * When I am depressed, I am sure that things really are as bad as they seem.
- I can't trust my judgments about myself when I feel down.

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1

As these are exploratory, rather than planned, tests, significance levels are best viewed as approximate guides. Application of the Bonferroni correction for multiple (12) tests indicates that only 4 of the 7 tests reached a conventional level of significance.

2

The signs of factor loadings were the same for all four variables, but the magnitude of loadings appeared somewhat different for MAQ.

3

At Week 20, for this same group of patients (i.e., excluding patients who had relapsed prior to Week 8), the sum of extreme scores was still less in CT (*Mdn* = 2.00, first quartile score = 0, third quartile score = 6.75) than in controls (*Mdn* = 4.0, first quartile score = 0, third quartile score = 14), but this difference was no longer significant on a two-tailed *t* test on logarithmically transformed scores, *t* (130) = 1.71, *p* = .091.

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Table 1. Baseline Characteristics of Sample

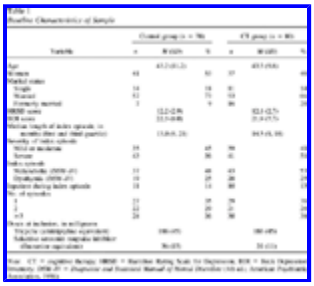

Table 2. Prediction of Relapse From Week 0 and Week 8 Cognitive Measures (Excluding Patients With Relapse Onset Prior to Week 8)

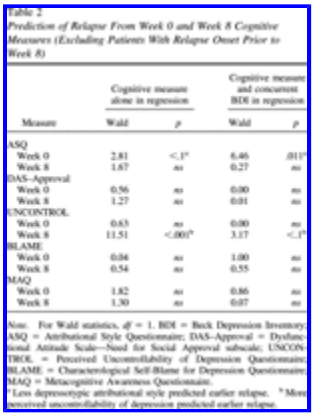

Table 3. Factor Loadings of Positive and Negative Extreme Scores from DAS—Approval, UNCONTROL, BLAME, and MAQ