How Does Cognitive Therapy Work? Cognitive Change and Symptom Change in Cognitive Therapy and Pharmacotherapy for Depression

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The effects of changes in depression-relevant cognition were examined in relation to subsequent change in depressive symptoms for outpatients with major depressive disorder randomly assigned to cognitive therapy (CT; n = 32) versus those assigned to pharmacotherapy only (NoCT; n = 32). Depression severity scores were obtained at the beginning, middle, and end of the 12-week treatment period, as were scores on 4 measures of cognition: Attributional Styles Questionnaire (ASQ), Automatic Thoughts Questionnaire (ATQ), Dysfunctional Attitudes Scale (DAS), and the Hopelessness Scale (HS). Change from pretreatment to midtreatment on the ASQ, DAS, and HS predicted change in depression from midtreatment to posttreatment in the CT group, but not in the NoCT group. It is concluded that cognitive phenomena play mediational roles in cognitive therapy. However, data do not support their status as sufficient mediators.

There is now substantial evidence that cognitive therapy (Beck, Rush, Shaw, & Emery, 1979) is an effective intervention for the acute treatment of nonbipolar depressed outpatients (Blackburn, Bishop, Glen, Whalley, & Christie, 1981; Elkin et al., 1989; Hollon et al., 1990; Murphy, Simons, Wetzel, & Lustman, 1984; Rush, Beck, Kovacs, & Hollon, 1977). Yet we have little evidence about how symptom reduction is mediated in cognitive therapy. It is reasonable to assume that cognitive therapy reduces symptoms by changing cognitive processes, but evidence for cognitive mediation has been hard to come by, perhaps because of the difficulties inherent in such a quest (see Hollon, DeRubeis, & Evans, 1987).

To paraphrase Baron and Kenny (1986), evidence for cognitive mediation of cognitive therapy's symptom reduction effects would come from the following set of relations:

1. Cognitive therapy reduces depressive symptoms more than does the alternative treatment (i.e., a treatment effect on symptom change). Only if this first test is met can one go on to the succeeding ones.

2. Cognitive therapy produces greater changes in a cognitive variable than does the alternative treatment (i.e., a treatment effect on cognitive change).

3. Change in the cognitive variable covaries with symptom change, even when the variable "treatment" is held constant statistically.

4. Inclusion of the cognitive variable as a covariate reduces the treatment effect on symptom change. (In complete mediation the relation is reduced to zero.)

Unfortunately, most of the designs used in clinical trials of cognitive therapy for depression have not included a substantially less powerful treatment (e.g., a pill- or attention-placebo). Hence, Condition 1 has not been satisfied, and thus Baron and Kenny's recommendations cannot be followed.

Instead, investigators have tried other ways of discerning mediation. For example, Simons, Garfield, and Murphy (1984) looked for differential effects of cognitive therapy versus nortriptyline pharmacotherapy on cognitive measures. They found, however, that cognitive change occurred as much in pharmacotherapy as in cognitive therapy, and they concluded that "cognitive (phenomenal) behavior more as symptoms of depression than as causes" (p. 49). But, as we have argued elsewhere (Hollon et al., 1987), because they also found equivalent symptom reduction for cognitive therapy and pharmacotherapy, such evidence is equally compatible with the proposition...
that these cognitive variables were mediators in one treatment and consequences of change in depression in the other. Indeed, with the benefit of hindsight, it can be seen that Simons et al.'s analyses would have uncovered a cognitive mediator only if changes in that cognitive variable did not also occur as a consequence of symptom change. They could not have ruled out this alternative even by examining the relation between change in depression from Time 1 to Time 2 and change in cognition from Time 2 to Time 3. This is because one cannot be confident that the proper time lag has been selected for such an analysis.

Another attempt to uncover mediation was reported by Rush, Kovacs, Beck, Weissenburger, and Hollon (1981). They analyzed data from the early phases of treatment in the Rush et al. (1977) comparison of cognitive therapy versus imipramine pharmacotherapy. They found no differences in the magnitude of cognitive change or symptom change between the two treatments. They did find, using cross-lagged panel correlations, that in cognitive therapy, cognitive measures predicted later status on mood and vegetative symptom measures, but not vice versa. Curiously, in the pharmacotherapy group, they did not find this (or any other) pattern. They concluded that the "data offer indirect support for the cognitive theory of depression" (p. 227). But the data did not support the simple claim that cognitive changes precede (and thus perhaps cause) mood and vegetative changes. For if such a direct causal chain exists, it should have been evident in the pharmacotherapy group as well. What Rush et al. (1981) did expose, however, was a clue that might lead to the specification of treatment mediation processes. In particular, what may have found was a "moderator" variable in the prediction of mood change from cognitive change. The moderator variable was treatment (cognitive therapy vs. pharmacotherapy), in that the variable "treatment" affected the strength of the prediction from cognition to subsequent depression. They did not discuss their results in these terms, and the implication of such a moderator effect, which we shall discuss below, may not be obvious. But their findings do call, at least, for an attempt to replicate the moderator relationship.

To this end we report an examination of temporal patterns of change in cognition and depressive symptomatology. We do so in the context of a randomized trial (Hollon et al., 1990) in which patients either received cognitive therapy (with or without imipramine) or did not receive cognitive therapy (but did receive imipramine pharmacotherapy). Several measures of depression-relevant cognition and symptomatology were obtained before and after treatment and at the midpoint, allowing for analyses of the effects of early changes in cognition on subsequent changes in depressive symptoms.

Method

Subjects

A comprehensive description of the subjects and procedures can be found in Hollon et al. (1990). Subjects had been referred to the study from the clinics of the St. Paul-Ramsey Medical Center and from the nearby Ramsey County Mental Health Center, St. Paul, Minnesota. On the basis of a Schedule for Affective Disorders and Schizophrenia —Lifetime (SADS-L) based interview (Endicott & Spitzer, 1978), 112 nonpsychotic, nonbipolar adult outpatients were screened into the study. Subjects were required to meet Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) for major depressive disorder and other inclusion criteria. A check on diagnostic accuracy was conducted by William M. Grove, an acknowledged expert on the RDC, who screened videotapes of the interviews. He disagreed with the diagnosis made on only four (4%) of the patients originally entered into the study. These four cases were dropped and are not included in the subsequent analyses.

Patients admitted to the study were randomly assigned to one of four active treatments. Each of the treatments was to last for 12 weeks. Cognitive therapy alone, imipramine pharmacotherapy alone, and the combination of cognitive therapy with imipramine were three of the packages. The fourth cell was imipramine for 12 weeks plus maintenance imipramine in the succeeding year.

Both types of treatment (pharmacotherapy and cognitive therapy) were conducted at both sites from which referrals were obtained. Once patients were assigned to treatment condition, they were assigned to a therapist within that treatment condition on the basis of therapist availability, irrespective of the site from which the referral came. Thus, the project functioned as a single-site study.

The sample was 80% female and 91% White (9% were Black). The mean age was 33 (range = 18–62 years), and 32% of the sample was married or cohabiting. Fourteen percent of the subjects had completed college, and another 34% had attended college; 20% of the sample had not completed high school.

Treatments

Cognitive therapy. The rationale and procedures of cognitive therapy are described in detail elsewhere (Beck et al., 1979). In our study, patients in the cognitive therapy or combined conditions were to meet with their cognitive therapist between 16 and 20 times over the 12-week treatment period. Sessions were 45 to 60 min in length. The plan was for two sessions to occur in each of the first 4 weeks, followed by one or two sessions per week in Weeks 5 through 8, followed by one session in each of the last 4 weeks.

Imipramine pharmacotherapy. Patients assigned to imipramine treatment, whether in one of the imipramine alone cells or in the combined cell, were to attend weekly sessions with their pharmacotherapists. These sessions were from 15 to 60 min, typically 30 min. They focused on pharmacotherapy management and medication compliance, adjusting dosage and dosage schedules, dealing with side effects, etc.) and clinical management (reviewing the patient's functioning, giving brief supportive counseling and limited advice, etc.). Pharmacotherapists were enjoined from conducting formal psychotherapy, including cognitive therapy. Ratings of therapist behavior, reported elsewhere (DeRubeis, Hollon, & Evans, 1989), revealed that the pharmacotherapists indeed refrained from conducting psychotherapy in these sessions.

The pharmacotherapists followed a flexible dosage schedule that was to result in a dose of 200–300 mg per day by the 3rd week of the
treatment. Plasma imipramine/desmethyliimipramine levels were monitored at midtreatment and posttreatment or if a patient experienced nonresponse or intolerance of side effects.

In this article, because we are concerned with the role of cognitive variables, we collapse the two groups which received cognitive therapy into one. We refer to this group of 32 treatment completers as CT. And because we are concerned only with the active treatment period, the two imipramine-only conditions will also be collapsed into one; the 32 completers in these cells constitute the NoCT group.

Therapists

Cognitive therapists. All four cognitive therapists were experienced practitioners of psychotherapy before their participation in this study. Three were ACSW clinical social workers; a fourth was a PhD clinical psychologist. Therapists had from 8 to 20 years of psychotherapy experience before study participation. None of the cognitive therapists had experience with Beck's cognitive therapy for depression before the intensive training conducted by Steven D. Hollon, Robert J. DeRubeis, and Mark D. Evans. The nature and structure of the training and supervisory programs are described in Hollon et al. (1990).

Pharmacotherapists. Five board-certified psychiatrists served as pharmacotherapists. All were experienced in the use of tricyclic antidepressants, both in clinical practice and in controlled research settings. Consequently, no formal training before the present study was deemed necessary. However, weekly meetings of the pharmacotherapists were held under the supervision of Vicente B. Tuason to discuss ongoing cases.

Measures

In this report we describe only the measures of cognition and depression. All measures reported in this article were obtained before, during, and after treatment (midtreatment and posttreatment evaluations were conducted after 6 and 12 weeks of treatment, respectively). We focus in this paper on cognitive change that occurred over the first half of treatment, in an attempt to replicate Rush et al. (1981). Because in our data set end-of-treatment cognitive scores are most relevant to the prediction and understanding of posttreatment protection against relapse, we present those data in a separate article that deals with relapse (Evans, Hollon, & DeRubeis, 1989). See Hollon et al. (1990) for a more complete schedule and a comprehensive listing of measures used in the study.

Cognitive measures. Four cognitive measures were obtained: the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980), the Hopelessness Scale (HS; Beck, Weissman, Lester, & Trexler, 1974), the Dysfunctional Attitudes Scale, Form A (DAS; Weissman & Beck, 1978), and the Attributional Styles Questionnaire (ASQ; Seligman, Abramson, Semmel, & van Baaren, 1979).

The ATQ is a 30-item, self-report questionnaire that assesses the frequency with which patients experience 30 depressogenic self-statements (e.g., "I'm no good," "I'll never make it"). It has been shown to distinguish clinically depressed from nondepressed psychiatric patients and from normal control subjects (Hollon, Kendall, & Lumry, 1986). In the present study the ATQ was used as an index of "surface" cognitive phenomena (Beck et al., 1979). As such, it was not expected to behave differently between CT and NoCT, but rather as a cognitive concomitant (or aspect) of depressive symptoms. Each item is scored on a 1-5 scale, so that total scores can range from 30 to 150. Nonpatients tend to score in the 40-60 range, whereas depressed patients tend to score in the 90-130 range.

The HS is a 20-item, true/false, self-report measure intended to tap the degree of the respondent's general pessimism. High scorers on this scale endorse such items as "Things just won't work out the way I want them to." The HS has been shown to distinguish clinically depressed from nondepressed psychiatric patients (Brown & Beck, 1989). Total scores can range from 0 to 20. Nonpatients tend to score in the 0-4 range, whereas depressed patients tend to obtain scores of 8 or above. Early-in-treatment HS was found to predict subsequent levels of depressive symptoms in cognitive therapy but not in pharmacotherapy in the Rush et al. (1981) study. It is the only cognitive measure from that study that was also used in the present investigation.

The DAS is a series of 40 attitudinal statements that were written to represent depressotypic "underlying assumptions" (Beck et al., 1979). Examples are "I cannot be happy unless most people I know admire me," "If I fail at my work, then I am a failure as a person." Patients check the degree to which they endorse the statements on a 7-point Likert scale. The DAS has been shown to distinguish clinically depressed from nondepressed psychiatric patients and from normal control subjects (Hollon et al., 1986). Total scores can range from 40 to 280. Nonpatients tend to score in the 80-120 range, whereas depressed patients tend to score in the 140-200 range. Changes in attitudes represented on the DAS are seen as important to the process of cognitive therapy (see Beck, 1984a; Kovacs & Beck, 1979).

On the ASQ, patients are asked to imagine life events and to assign causes for those life events, six of which are positive and six negative. Patients then rate the degree to which each event is internal, stable, and global. The ASQ yields several indices that are highly correlated with each other. We will report on only one of these indices—the composite of internality, stability, and globality for positive events minus negative events—as its properties are the most consistent in the literature on the ASQ (Peterson & Seligman, 1984). The ASQ, too, has been found to distinguish clinically depressed from nondepressed psychiatric patients and from normal control subjects (Eaves & Rush, 1984; Hamilton & Abramson, 1983). For this report, in order to simplify presentation, we have rekeyed the ASQ composite score so that higher scores reflect more depressotypic attributional styles. We did this by subtracting the composite score from zero. Thus, total scores can range from -18 to +18. Nonpatients tend to score in the -5 to +1 range, whereas depressed patients tend to score in the -3 to +3 range. Seligman et al. (1988) reported a high correlation of change in ASQ with change in depression in a sample of patients treated with cognitive therapy. We had hypothesized that the ASQ would behave differently in CT versus NoCT, because an important aspect of cognitive therapy

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2 Moreover, results obtained in the cognitive therapy alone condition and the cognitive therapy plus imipramine condition were very similar. We present the results with these groups collapsed so as to increase the power of statistical tests and to simplify the presentation of the data.

3 We did not obtain group differences in end-of-treatment outcome; thus our data failed to meet Condition I (see page 862) for testing for mediators of change. However, we did obtain a differential effect of treatment on posttreatment relapse incidence (CT patients fared better in follow-up than did NoCT patients). Consequently, we examined the end-of-treatment cognitive measures as possible mediators of posttreatment relapse prevention (see Evans et al., 1989). In brief, we found that the ASQ (and to some extent the DAS) changed differentially in CT vs. NoCT patients who improved in therapy. That is, the attributions of symptomatically improved CT patients were significantly better at the end of treatment than those of symptomatically improved NoCT patients. Thus, we found the "specificity" that others have not been able to find. But since these findings were obtained at the end of treatment, they could provide only the weakest form of evidence that change in attributions mediated change in acute symptoms. We did, however, find that such differential change on the ASQ may explain the differential relapse rate we observed in our study.
Table 1
Cognitive Scores at Pretreatment and Midtreatment for Each Treatment Group

<table>
<thead>
<tr>
<th>Cognitive measure</th>
<th>Cognitive therapy</th>
<th>No cognitive therapy</th>
<th>Between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(M^*)</td>
<td>(SD)</td>
<td>(M^*)</td>
</tr>
<tr>
<td>ASQ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>0.93</td>
<td>2.81</td>
<td>0.22</td>
</tr>
<tr>
<td>Midtreatment</td>
<td>-0.65***</td>
<td>2.64</td>
<td>-1.08**</td>
</tr>
<tr>
<td>ATQ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>100.0</td>
<td>23.8</td>
<td>105.5</td>
</tr>
<tr>
<td>Midtreatment</td>
<td>62.0****</td>
<td>25.9</td>
<td>6.28***</td>
</tr>
<tr>
<td>DAS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>156.8</td>
<td>32.1</td>
<td>147.6</td>
</tr>
<tr>
<td>Midtreatment</td>
<td>131.0***</td>
<td>33.1</td>
<td>137.4**</td>
</tr>
<tr>
<td>HS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>11.9</td>
<td>4.6</td>
<td>11.1</td>
</tr>
<tr>
<td>Midtreatment</td>
<td>7.4****</td>
<td>5.8</td>
<td>8.0***</td>
</tr>
</tbody>
</table>

Note. ASQ = Attributional Styles Questionnaire; ATQ = Automatic Thoughts Questionnaire; DAS = Dysfunctional Attitudes Scale; HS = Hopelessness Scale.

* \(p < .10\). ** \(p < .05\). *** \(p < .01\). **** \(p < .001\). All tests are two-tailed.

is to focus patients on the explanations they give for events, so that they may reconsider them.

Depression measure. A composite depression measure was constructed at each assessment point using three measures of depression. Two of the three were clinician-rated scales: the Hamilton Rating Scale for Depression (Hamilton, 1960) and the Raskin Depression Scale (Raskin, Schulerbrandt, Reatig, & McKeon, 1970); the other was a self-report measure, the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). At each of the three measurement points (pretreatment, midtreatment, and posttreatment), the respective measures were each standardized such that the mean was set to zero and the standard deviation to one. These three scores were then averaged so that each patient had a composite depression score at each point.

Summary of Outcome Findings

The Hollon et al. (1990) comparative outcome findings and their implications for the questions addressed in this article can be summarized as follows: Symptom depression remitted significantly and substantially in all treatment groups during the acute (12-week) treatment period. The combined treatment evidenced some advantage over the two single modalities among completers, including a nonsignificant trend relative to pharmacotherapy alone. However, none of the between-groups comparisons reached conventional levels of significance. Of most relevance to this article is the fact that the difference in depression outcome between CT and NoCT patients was small and nonsignificant. The near equivalence in depression outcome for the treatments allows for a relatively unconfounded examination of the temporal relations of changes in cognition and change in depression.

Results

Pretreatment Scores and Early Change on Cognitive Measures

Table 1 gives descriptive statistics at pretreatment and midtreatment for the four cognitive measures, by group (CT vs. NoCT). Pretreatment means were equivalent between the groups on all four measures (all \(F < 1\)). Significant improvement from pretreatment to midtreatment was exhibited by both groups on all four cognitive measures, as assessed by paired \(t\) tests within each group (see Table 1). Thus, cognitive therapy did not uniquely produce improvement on any of the cognitive measures at midtreatment. We also tested for the differential effect of treatment on pretreatment to midtreatment change in the cognitive variables (see Table 1), using analyses of covariance (ANCOVAs) with the midtreatment cognitive score as the dependent variable and pretreatment score as the covariate. There was a nonsignificant trend for greater improvement on the DAS by the CT group relative to the NoCT group. No between-groups difference in improvement was found at midtreatment on any of the other cognitive measures (ASQ, ATQ, and HS; all \(F < 1\)).

Intercorrelations of Early Change on Depression and Cognitive Variables

In order to examine the interrelations of the measures of change during the first half of treatment, we first computed pretreatment to midtreatment residual change scores for the depression composite measure as well as for each of the cognitive variables (see Cohen & Cohen, 1979, pp. 379–382). Table 2 is a correlation matrix of these variables. Changes in the cognitive measures were moderately related to one another, indicating some overlap between the variables. But there was suffi-
Table 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>DEP</th>
<th>ASQ</th>
<th>ATQ</th>
<th>DAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ASQ</td>
<td>.23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. ATQ</td>
<td>.76***</td>
<td>.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. DAS</td>
<td>.51***</td>
<td>.24</td>
<td>.43*</td>
<td></td>
</tr>
<tr>
<td>4. HS</td>
<td>.59***</td>
<td>.47**</td>
<td>.42*</td>
<td>.56***</td>
</tr>
</tbody>
</table>

Note: DEP = Depression severity composite; ASQ = Attributional Style Questionnaire (keyed negative relative to the other measures); ATQ = Automatic Thoughts Questionnaire; DAS = Dysfunctional Attitudes Scale; HS = Hopelessness Scale. Coefficients are Pearson correlations between residualized change scores.

*p < .05. **p < .01. ***p < .001. All tests are two-tailed and adjusted by the Bonferroni method.

Predicting Late Depression Change From Early Cognitive Change

In order to examine the relation of early change on the cognitive variables to subsequent change in depression, we first computed scores to represent residual change in depression from midtreatment to posttreatment. Then, for each of the cognitive measures we calculated a correlation of residualized early change with residualized late depression change. We then tested each of these correlations against the hypothesis of no relation. As is shown in Table 3, early ASQ, DAS, and HS change each significantly predicted subsequent depression change in CT. The significance of these relations held when we applied the Bonferroni correction. In NoCT, none of these predictive relations were observed. The ATQ was not a significant predictor of subsequent change in either group.

Table 3 also displays the results of analyses recommended by Baron and Kenny (1986) to test for a differential relation between two variables as a function of a third (moderator) variable. These analyses are similar to tests of between-groups differences in correlations, but, as Baron and Kenny have pointed out, these analyses avoid the deficiencies inherent in such tests. Specifically, they do not allow between-groups differences in range or measurement error to influence the test of between-groups differences in the strength of the relation between the two variables.

To do these analyses we cast four hierarchical regression equations of the following form:

\[
\text{rcDEP} = \text{rcCOGearly \ GROUP (rcCOGearly \ \times \ GROUP)}
\]

where rcDEP is residual change in depression from midtreatment to posttreatment; rcCOGearly is the pretreatment to midtreatment residual change score on a cognitive measure; and GROUP is a categorical variable (CT vs. NoCT). The final term in each model, the interaction, represents the between-groups difference in the prediction of depression change from cognitive change (i.e., the difference between CT and NoCT in the slope of the regression line). As is shown on the right side of Table 3, the interaction term reached significance for both the ASQ and the DAS. On the HS the difference was not significant. The ATQ, as expected, yielded no between-groups difference in slope. Thus, early ASQ improvement and early DAS improvement were differentially predictive of subsequent improvement in depressive symptoms, depending on treatment group. This conclusion held only for the DAS when alpha was adjusted by the Bonferroni method.

Thus, changes in the ASQ and DAS that occurred in the first 6 weeks of treatment each significantly predicted subsequent change in depression in CT. Moreover, these relations were found to be stronger in the CT group than in the NoCT group. The HS yielded a similar pattern, but differential prediction between the two groups (the test of moderator status) was not significant. As expected, the ATQ yielded no specific or differential predictions of subsequent change in depression.

Predicting Late Cognitive Change From Early Depression Change

We also examined the data for temporal effects in the direction opposite to the effects just described. In analyses patterned after those above, we found no evidence for the differential prediction of subsequent change in depression from early depression change. The p values for the interaction term were greater than .15 on all four measures, indicating that there were no significant between-groups differences in predictions of late cognitive change from early depression change. Moreover, none of the within-group correlations between residualized early depression change and residualized late cognitive change were greater than .25 (all ps greater than .20).

To sum up the results, midtreatment to posttreatment change in depression was predicted by prior cognitive change in the CT group, but not in the NoCT group. Early depression change did not predict subsequent cognitive change in either group.

Discussion

The search for mediation of symptom reduction in cognitive therapy (or any intervention) for depression has yet to yield satisfying answers. Such an undertaking is extremely difficult, filled with logical pitfalls and logistical constraints. Not the least problematic is the likelihood that depressive symptoms and the mechanisms of effective treatments are integrally intertwined, or reciprocally caused (see Beck, 1984a, 1984b; Simons, 1984). Thus, using a cross-sectional strategy in the context of equivalently powerful treatments, as has been done in most of the outcome studies of cognitive therapy for depression includ-
Table 3
Prediction of Late Change in Depression From Early Cognitive Change for Each Treatment Group

<table>
<thead>
<tr>
<th>Cognitive measure</th>
<th>Treatment group</th>
<th>Group × Cognitive Change interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cognitive therapy</td>
<td>No cognitive therapy</td>
</tr>
<tr>
<td>ASQ</td>
<td>.54**</td>
<td>-.07</td>
</tr>
<tr>
<td>ATQ</td>
<td>.08</td>
<td>.20</td>
</tr>
<tr>
<td>DAS</td>
<td>.51*</td>
<td>-.25</td>
</tr>
<tr>
<td>HS</td>
<td>.55**</td>
<td>.08</td>
</tr>
</tbody>
</table>

Note. ASQ = Attributional Style Questionnaire (keyed negative relative to the other measures); ATQ = Automatic Thoughts Questionnaire; DAS = Dysfunctional Attitudes Scale; HS = Hopelessness Scale. Dash indicates p > .50.

* Coefficients are Pearson correlations between residualized early cognitive change and subsequent residualized depression change.

b Tests are for slope differences between cognitive therapy and no cognitive therapy.

*p < .05; **p < .01. All tests are two-tailed and adjusted by the Bonferroni method.

ing Hollon et al's (1990), it may be impossible to distinguish cause from effect. But delineating causal mechanisms of cognitive therapy is as important as it is perplexing, both for theoretical and practical reasons. And although it is difficult to document that different effective treatments (such as cognitive therapy vs. pharmacotherapy) work through different means, it seems likely that they do at some level, so the challenge lies in isolating the proper construct(s) to measure and selecting appropriate research methods.

Our findings suggest that a set of cognitive constructs, as measured by the ASQ, DAS and HS, do play a meditational role in cognitive therapy. However, the meditational role they play does not appear to be a causally sufficient one, because the relation of change on these variables to subsequent symptom change was not also found in pharmacotherapy. The question then becomes: What is it about the process of therapy that makes such cognitive change so necessary to further improvement in cognitive therapy, yet unrelated to further improvement in pharmacotherapy? We offer one hypothesis below.

Perhaps success in cognitive therapy depends on cognitive change in the following way: In order to benefit from cognitive therapy, the patient must engage in day-to-day life the methods and strategies learned in therapy. In order to do so fruitfully, the patient must already have experienced some changes in relevant thinking and attitudes. He or she must be more hopeful (as indicated by change on the HS), he or she must have begun to rethink dysfunctional attitudes (i.e., change on the DAS), and he or she must be able to generate and entertain more sanguine reactions to events (change on the ASQ). Insofar as these changes have begun, the patient (a) will engage what he or she is learning in therapy (e.g., to define problems carefully, to challenge inferences) and (b) will be successful in these efforts. If (a) and (b) occur, the patient's symptoms will tend to improve.

This explanation differs from the view that changes in the cognitive constructs we measured are sufficient to mediate change in depressive symptoms. Indeed, such an account is supported neither by our findings nor by those of Rush et al. (1981). In neither study did changes in cognition bear a relation to subsequent changes in symptoms among pharmacotherapy patients, in spite of the fact that substantial—and significant—early cognitive change occurred in pharmacotherapy in both studies. Thus it appears that cognitive change in pharmacotherapy does not breed further change, as it does in cognitive therapy. If we are correct, this is because in pharmacotherapy, changes in the patients' cognitions are not accompanied by the learning and use of cognitive and behavioral strategies. We might speculate, in contrast, that for pharmacotherapy the psychological (cognitive) state requisite for improvement is the belief that taking the pills is important.

The hypothesis we are proposing about change in cognitive therapy, then, is that symptom change results from an interaction of changes in thinking and attitudes with the active engagement of the therapy (i.e., the application of its mood-correcting methods). Engagement of the therapy includes, but may not be limited to, efforts directly suggested by the therapist (i.e., in-session efforts and homework). Consistent with this hypothesis are findings from other studies of the process of cognitive therapy. In a naturalistic study of therapist behavior and depression outcome, DeRubeis and Feeley (1980) found a relation between symptomatic improvement and the degree to which patients had been encouraged to engage in specific mood-correcting strategies. And there is evidence that practice in the form of homework is of specific benefit as well (Fennell & Teasdale, 1987; Neimeyer, Twentyman, & Prezant, 1985; Persons, Burns, & Perloff, 1988).

Barlow, O'Brien, and Last (1984) and Michelson, Mavissakalian, Marchione, Dancu, and Greenwald (1986) have offered an explanation for the efficacy of exposure treatment for agoraphobia that also relies on the patient's active engagement of the therapeutic methods. Both groups have reported that the amount of practice the patient engages in during the course of therapy is related to benefits achieved from exposure therapy.

We should stress that the hypothesis we offer is speculative, and not, in its specific form, supported by the present findings. But we should also emphasize that the pattern it attempts to explain is quite substantial, and it replicates the pattern reported by Rush et al. (1981).

Findings from outcome studies have shown that cognitive therapy often leads to rapid symptom reduction. This means that in order to detect the forces that move symptom change, measures of these phenomena will have to be obtained early and, preferably, often.

Also, if a study aims to detect mediation in the way that Baron and Kenny (1986) recommend, the design must include a minimal treatment, or at least a less powerful treatment condition that results in less change in the syndrome (see Baron & Kenny's first requirement on p. 862 of this article). This less powerful condition could be a less intensive version of the full treatment package, such as sessions every other week in comparison with twice-weekly sessions. Or it could be a part of the package in a "dismantling" study.

Alternatively, the investigator can hope to capitalize on naturally occurring variation in the quality or adherence of the therapy given to patients. Even aside from the problem of third-variable causality, however, this last strategy is a gamble. Robert J.
DeRubeis has been involved in two such efforts. In analyses of therapist behavior from the current study, we did not obtain a significant effect of therapist behavior on outcome (DeRubeis, Hollon, Evans, Garvey, et al., 1989), perhaps because of range restriction on our measures of therapist behavior. In the other study (DeRubeis & Feeley, 1990), variation in therapist behavior was related to outcome. Unfortunately, in the latter study, assessments of cognitive constructs to detect the mediation of this effect were not in place.

Finally, we offer a comment about what to measure. In accordance with the hypothesis suggested, we propose that in future studies, in addition to measures such as the ASQ, DAS, and HS, the following constructs be examined as mediators: (a) the acquisition of problem solving and “compensatory” skills the patient can apply in response to upsetting events (see Barber & DeRubeis, 1989, 1990) and (b) the frequency with which the patient applies those skills in daily encounters during the course of therapy.

References


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