

Cognitive Processes in Cognitive Therapy: Evaluation of the Mechanisms of Change in the Treatment of Depression

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A central theoretical principle guiding cognitive therapy is that mediation by cognitive processes is linked to the successful treatment of depression. The most recent review of the literature on this question is over a decade old and was suggestive of cognitive mediation for cognitive therapy, but was not conclusive. Since this review, a number of studies have been published that address cognitive mediation. The mediation hypothesis can be broadly defined as encompassing two related questions: cognitive mediation framed as “are cognitive changes associated with therapeutic improvement,” and cognitive specificity from the perspective of “are changes in cognition specific to cognitive therapy?” This latter question is particularly important when cognitive therapy is compared to pharmacotherapy. This article reviews the current literature associated with these questions. Our review indicates that the current body of research generally supports cognitive mediation, but is considerably more mixed for cognitive specificity. However, some evidence suggests that cognitive changes associated with pharmacotherapy are more superficial than those associated with cognitive therapy.

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Beck's theory of depression is arguably the most influential model ever developed on the causes, course, and treatment of depression. Significant elements of the theory were originally proposed in 1963, with a more comprehensive framework appearing in Beck's seminal 1967 book. Since the 1967 volume, substantial elaboration of the model has been suggested by Beck, as well as other theorists (Ingram, Miranda, & Segal, 1998). In its contemporary form, the theory encompasses both descriptive elements of depression, as well as statements about causal factors (Haaga, Dyck, & Ernst, 1991). For example, Beck suggests that depressogenic schemas lie at the root of depression, and that these schemas form part of a diathesis–stress relationship; such schemas are proposed to be latent until evoked by stressful circumstances (Beck, 1967; Ingram et al., 1998; Segal & Ingram, 1994), but once evoked they lead to negative perceptual biases as well as negative automatic thinking that cast a negative interpretation on events. This cognitive reactivity turns “normal” sad mood states into a downward spiral of negativity and eventually a depressive disorder (Ingram et al., 1998). Together, these elements form the core causal features of the model.

Perhaps the most important element of Beck's model is the development of a therapeutic paradigm, cognitive therapy (or cognitive–behavioral therapy), for the treatment of depression.¹ Given the theoretical structure of the model, cognitive therapy comprises a set of procedures intended to alter the function of the depressive schema, as well as its content and structure (Beck, Rush, Shaw, & Emery, 1979). As such, the model makes clear assumptions about the processes responsible for therapeutic change. In particular,

cognitive therapy assumes that change in cognitive schemas is sufficient to bring about recovery from the disorder. Cognitive therapy not only makes use of a number of cognitive methods to effect this change, but also assumes that even explicitly behavioral treatment components, either directly or indirectly, facilitate changing cognitive structures.

Support for many elements of Beck's model is considerable. For example, the descriptive aspects of depression noted by the model have been confirmed by numerous studies (Haaga et al., 1991). The diathesis-stress proposals of the model have also been borne out by an accumulating body of data. In particular, proposals regarding the emergence of a negative schema under stress have begun to receive consistent and considerable support (e.g., Scher, Ingram, & Segal, 2005; Segal & Ingram, 1994). Recent data have also been consistent with proposals regarding the causal processes in depression that are linked to the emergence of negative schemas (e.g., Segal, Gemar, & Williams, 1999; Segal et al., 2006).

There is also considerable evidence substantiating the efficacy of cognitive therapy. In a number of trials assessing the treatment of depression, cognitive therapy has been found to be efficacious in comparison to no treatment, as well as in comparison to other active treatments, and also fares well in comparison to psychopharmacological approaches (Hollon, 2006). Data also suggest the long-term efficacy of cognitive therapy in the prevention of future episodes of depression (Hollon, Stewart, & Strunk, 2006). Although the focus in this article is on the treatment of depression, for which cognitive therapy was originally developed, it is worth noting that cognitive therapy has shown considerable promise in the treatment of generalized anxiety disorder, panic disorder, and social phobia. These data account for the fact that cognitive therapy has been designated as an empirically supported treatment for several disorders (see Chambless & Ollendick, 2001).

MECHANISMS OF CHANGE IN COGNITIVE THERAPY:

NOT ALL COGNITIONS ARE CREATED EQUAL

Although cognitive therapy is clearly efficacious in the treatment of depression, less clear is the evidence concerning the cognitive mediation that is at the conceptual core of cognitive therapy. The model assumes that changes in key cognitive processes are critical for recovery from the disorder as well as for improving the deficits (e.g., social skills problems) that frequently accompany depression.

More specifically, cognitive mediation proposals assume that the depressogenic schema is changed in fundamental ways. Hollon, Evans, and DeRubeis (1990) note that this idea reflects an *accommodation* model of change: "the operative schema is modified in a profound way" (p. 121). Other models of change are possible, however (Ingram & Hollon, 1986; Whisman, 1993). For instance, rather than profoundly change the nature of depressogenic cognitive structures, these structures may remain intact but become deactivated over the course of treatment (Ingram & Hollon, 1986), a possibility that Hollon et al. (1990) refer to as the *activation-deactivation* model. In this case, the deactivation of cognitive schemas may be functionally linked to the reduction of depressive symptoms, but the schemas themselves are unchanged. Another alternative is that depressogenic structures are largely unchanged, but that cognitive therapy leads to the development of *compensatory* schemas that incorporate skills for dealing with stressful situations that ordinarily would lead to depression (Hollon et al., 1990). Recent adaptations of cognitive therapy incorporating mindfulness training (Segal, Williams, & Teasdale, 2002) are based in part on such assumptions.

In evaluating these competing models of cognitive change, it is worth noting that variability in therapist skills may play a substantial role in determining the manner in which cognitive therapy works. Although we are unaware of any empirical data that bear directly on this issue in cognitive therapy, it seems reasonable to propose that experienced and skilled cognitive therapists may be more adept at changing the schemas in profound ways. Hence, for these therapists cognitive therapy may indeed function in the way that the original model proposes; by changing the structure of depressive schemas. On the other hand, cognitive therapy may work for less experienced or skilled therapists because the schema has been deactivated rather than changed in some important fashion. In a sense then, all of these models may be accurate inasmuch as cognitive therapy depends on the ability of the therapist.

Although the different models of change focus on the schema, it is important to note the variety of cognitive constructs that are potentially available for change. In general, cognitive depression models propose that the negative self-schema is the core structure in depression, but that schemas are also associated with cognitive variables that span a number of domains. Such domains include, but are not limited to, automatic thoughts, dysfunctional

attitudes, negative attributional patterns, and cognitive distortions. These and similar constructs are the focus of virtually all studies of cognitive mediation in cognitive therapy for at least two reasons. First, such variables are important in their own right. Second, because schema change cannot be directly observed, these variables therefore serve as indicators of possible changes in the functioning of schemas. However, it should be noted that despite the fact that changes in these variables are consistent with ideas about structural change in the schema, such changes are also consistent with alternative models of change in cognitive therapy (e.g., the deactivation of the schema). Thus, directly documenting structural change, and distinguishing one change model from another, is difficult if not impossible.

The diversity of variables that have been examined in studies of cognitive therapy also highlights distinctions between “deep” and “surface” cognitions. Arguably, the deepest cognitive variable is the schema. Beck et al. (1979) note that such schemas are composed of long-held core beliefs about the self that may not be accessible. Alternatively, a variable such as automatic thoughts reflects the subconscious monologue in which depressed individuals engage, and is thus thought to reflect a more surface-level cognitive variable. However, this is not to suggest that even surface cognitions do not have causal relevance, at least depending on how causality is defined. For example, Beck et al. (1979) propose that automatic thoughts reflect a mediator of change in cognitive therapy, thus suggesting the causal relevance of this construct. Relatedly but more broadly, however, most definitions of causality focus exclusively on onset. Although this is an important aspect of causality, a more complete definition encompasses a causal cycle in which onset variables are an important dimension, but so too are the factors that cause depression to be maintained (Ingram et al., 1998). In this more broadly defined definition of causality, variables such as automatic thoughts may very well play a causal role in the maintenance of the depressed state. Moreover, the causal relevance of these constructs may also be seen in considerations of therapist ability and its link to change in cognitive therapy. In particular, therapists who are less experienced and less skilled may be more prone to focus on surface rather than deep cognitions, which might lead to improvement through deactivation rather than through changes in the nature of the schema

itself. Theoretical discussions of schema activation processes in depression have noted how such processes may occur (e.g., Scher et al., 2005; Segal & Ingram, 1994).

PREVIOUS REVIEWS OF COGNITIVE MEDIATION IN COGNITIVE THERAPY AND THE COMPONENTS OF COGNITIVE MEDIATION

Data on mediation in cognitive therapy have been accumulating for some time, although the last review of relevant data was published over a decade ago. In particular, Whisman (1993) reviewed the available literature and concluded that the data showed promise for the cognitive mediation hypothesis. However, Whisman also noted that support was tentative, varied from cognitive measure to cognitive measure, and was hampered by both conceptual and methodological limitations in extant studies. Given these issues and the 14 years since that review appeared, it is noteworthy that substantial additional research has examined the ideas that underlie assumptions about the mechanisms of change in cognitive therapy. For example, whereas Whisman (1993) was able to report on nine studies examining these ideas, there are now over two dozen studies reporting data relevant to the cognitive mediation hypothesis. Given the considerable additional data now available, our purpose is to review research aimed at evaluating cognitive mediation in cognitive therapy.

Whisman noted two fundamental assumptions underlying the proposed mechanisms of cognitive therapy. First is the idea that cognitive change must covary with symptomatic reduction; hence, cognitive changes putatively produced by cognitive therapy lead to changes in depression. Second, the hypothesis suggests that cognitive change is specific to cognitive interventions. Questions about specificity reflect whether cognitive changes are unique to cognitive therapy because of specific procedures (i.e., a focus on the modification of cognition) or are due to nonspecific variables that are part of all therapies. However, this latter criterion is problematic when cognitive therapy is compared to other psychosocial treatments in that contemporary treatments for depression may affect cognitive variables. That is, it is plausible and perhaps quite likely that psychosocial treatments achieve their effects by altering patients' perceptions of themselves and their environment in ways that promote more effective interactions and less depressive distress. Moreover, some “noncognitive” treatments intentionally target cognition. Procedures reported by Jacobson et al. (1996) reflect a

case in point. In particular, they report a study on the efficacy of behavioral activation. However, in addition to targeting behaviors, behavioral activation also specifically targeted a fundamental cognitive variable: rumination. Such an approach is consistent with behavioral procedures employed in cognitive therapy; the goal is to change behavior as a way of changing cognition (Beck et al., 1979). Few if any psychosocial treatment probably can claim to have no effect on thinking patterns in depression.

One therapeutic procedure that most likely can lay claim to not directly affecting cognition is medication; theoretical proposals specifying that medication achieves its effect through altering cognitions have yet to be suggested. Hence, the most appropriate test of cognitive specificity stems from cognitive therapy–pharmacological comparisons. However, it is important to note that although this is the most appropriate comparison for cognitive therapy, it should not be expected that no cognitive changes are associated with pharmacological interventions; some cognitive change can be a consequence of symptomatic reduction, but should not play a causal role in symptom reduction (Hollon, DeRubeis, & Evans, 1987). Rather, the issue becomes a matter of comparing the magnitude of cognitive change seen in cognitive therapy with cognitive changes that might be associated with medication. How large should this difference be?

Mediation criteria articulated by DeRubeis et al. (1990) may answer this question from a practical standpoint. In particular, they note four criteria by which to judge mediation in cognitive therapy: (a) cognitive therapy reduces depressive symptoms more than does the alternative treatment; (b) cognitive therapy produces greater changes in a cognitive variable than does the alternative treatment; (c) change in the cognitive variable covaries with symptom change; and (d) inclusion of the cognitive variable as a covariate reduces the treatment effect on symptoms change (p. 862).

Given the efficacy of other treatments in treating depression that have been demonstrated (e.g., Elkin et al., 1989), it appears somewhat less than practical to require that for cognition to be a mediator, cognitive therapy must be more effective than alternative treatments. Additionally, little treatment research is able to covary cognitive variables. However, the idea that cognitive change should covary with symptom reduction, and that cognitive therapy should produce greater cognitive changes than alternatives,

is not only central to questions about the mechanisms of cognitive therapy, but also in the case of requiring reliably larger changes than medication, provides a practical answer about how to test specificity questions.

THE CURRENT BODY OF LITERATURE

These two aspects of mediation, association with therapeutic change and cognitive specificity, serve as the focus of our review. We start with a review of research examining the association between cognitive changes and therapeutic improvement. Most of this research assesses group outcomes at the conclusion of treatment, but some research examines within-session associations between cognition and symptomatic change. Next, we examine research relevant to the specificity aspect of cognitive mediation, in particular research that compares changes in cognitions between cognitive therapy and noncognitive interventions. Although the most appropriate specificity comparisons are with pharmacological interventions, which also form the bulk of the available research, we also examine comparisons with other psychosocial treatments. A summary of the results of this review is presented in Table 1.

Literature Search

The studies included in this review were identified by a computerized literature search using PsycINFO and by examining the reference sections in relevant articles. The search was conducted on articles published through December 2004. The computerized search used the following key terms separately and in combination: cognitive therapy, cognitive behavior therapy, cognitive–behavioral therapy, and depression. Because an important aspect of this review focused on the influence of cognitive therapy on cognitions, we also searched for studies that included the following measures of cognition: Attributional Style Questionnaire, Automatic Thoughts Questionnaire, Beck Hopelessness Scale, Cognitive Errors Questionnaire, Dysfunctional Attitudes Scale, Personal Beliefs Inventory, and Sociotropy–Autonomy Scale. Each measure has been used extensively in both therapy and psychopathology research, and all are considered to have adequate psychometric properties.

Criteria for Inclusion

In order to be included in this review, a study had to meet the following criteria: (a) examine the effectiveness

Table 1. Studies of cognitive change in cognitive therapy

Study	Patient type	Treatment	n	Measures	Results
Bieling et al. (2004)	Depressed outpatients	CT	149	BDI, SAS, HS	Full & partial responders < fear of criticism & rejection, > preference for affiliation compared w/nonresponders
Oei & Sullivan (1999)	Outpatients	CT	67	ATQ, BDI, DAS, HS	Responders decrease on ATQ > nonresponders; all subjects decreased on DAS & HS
Tang & DeRubeis (1999)	Depressed outpatients	CBT	61	PCCS, BDI	Substantial cognitive change in session preceded sudden gains in depressive symptom improvement
Tang et al. (2005)	Depressed outpatients	Partial CBT	44	PCCS, BDI	Replicated Tang & DeRubeis (1999)
Westra et al. (2002)	Outpatients	Full CBT	50		
Beevers & Miller (2004)	Depressed post-inpatients	CT	48	BDC, DAS, HS-Burns	Reductions on BDC, DAS, & HS-Burns
		PT, PT+FT, CO, PT+FT+CT	T = 100	BDI, CBQ, DAS-PS, HS, MSSJ	All treatments reduced depression & depressive thinking w/no differences between groups
Beutler et al. (1987)	Depressed outpatients	PT	12	BDI, HRSD, CEQ	CT < other groups on BDI & HRSD
		PL	15		No changes on CEQ w/any treatment
		CT+PL	16		
		CO	13		
Blackburn & Bishop (1983)	Depressed hospital & general practice outpatients	CT	22	BDI, HRSD, HS, SDM	Hospital: CO > CT > PT change on BDI, HRSD, HS, SDM
		PT	20		General practice: CT & CO > PT change on BDI, HRSD, HS, SDM
		CO	22		
Bowers (1990)	Outpatients	PT	10	BDI, HRSD, ATQ	RT+PT & CO < PT on BDI & ATQ; no group differences on HRSD.
		RT+PT	10		
		CO	10		
Imber et al. (1990)	Depressed outpatients	CT	37	DAS	CT > PT, IPT change on DAS-NSAS; CT ~ PL on DAS-NSAS; PL had greatest variance & mean was comparable to other non-CT groups
		PT	36		
		IPT	47		
		PL	34		
McKnight et al. (1992)	Depressed female outpatients	CT	22	BDI, DAACL, PBI	CT > PT change on PBI
		PT	21		
McNamara & Horan (1986)	Depressed university outpatients	CT	T = 40	ATQ, cognitive & behavioral measure, judge's rating	CT & CBT > BT & WL change on all cognitive & behavioral measures
		BT			
		CBT			
		WL			
Rehm et al. (1987)	Depressed female outpatients	Self-control + beh target	35	DAS, self-control, positive & negative self-statement measures	All treatments reduced depressive symptoms & depressive cognitions on all measures
		self-control + cog target	35		
		combined tx	34		
Rush et al. (1981)	Depressed outpatients	CT	18	BDI, HRSD, SCL-90, HS	CT & PT reduced depressive symptoms & thinking; improvements on HS, views of self & mood preceded changes in vegetative & motivational symptoms for CT
		PT	17		
Simons et al. (1984)	Depressed outpatients	CT	14	ATQ, DAS, CRT-IDC	CT & PT reduced DAS & ATQ scores; changes on DAS & ATQ ~ changes on BDI & HRSD
		PT	14		
Stravynski et al. (1994)	Depressed outpatients	CT	T = 18	ATQ, HS, DAS	No treatment differences in depressive symptoms or thoughts at termination or follow-up
		CO			
Teasdale et al. (2001)	Partially remitted depressed outpatients	CO	80	DAS-NSAS, ASQ, MAQ, UN-CONTROL, BLAME	No treatment differences in depressive symptoms or thoughts; CO > change on form of response for all cognitive measures-extreme responses; CT reduced relapse rates
		PT	78		

Table 1. *Continued*

Study	Patient type	Treatment	n	Measures	Results
Whisman et al. (1991)	Depressed inpatients	PT	17	ATQ, HS, CBQ, DAS	PT & CO reduced depression & ATQ at termination, 6, & 12 months; no change & no group differences on CBQ at termination; CT > PT change on CBQ at 6 & 12 months; no change on DAS at termination; CT > PT on DAS at 6 months; CT > PT on HS.
		CO	14		
Zettle & Rains (1989)	Outpatients	CT	10	BDI, HRSD, ATQ, DAS	All treatments reduced BDI; Distancing > CT & Partial CT change on BDI; CT & partial CT > Distancing change on DAS.
Barber & DeRubeis (2001)	Depressed outpatients	Partial CT	10		
		Distancing	11		
DeRubeis et al. (1990)	Depressed outpatients	CT	27	BDI, ASQ, DAS, SCS, WOR	Depression & cognition improved on all measures; change on DAS & WOR ~ change in depression. Improvement on all measures for all groups; change on ASQ, DAS, & HS predicted improvement in depression only for CT.
		PT	16	ASQ, ATQ, DAS, HS	
		CO	32		
Fava et al. (1994)	Depressed outpatients	CO	16	BDI, CQ, DAS, HRSD	Change on BDI & HRSD ~ change on DAS & CQ; changes on DAS & CQ didn't predict degree of improvement on BDI & HRSD.
		PT	115		
Jacobson et al. (1996)	Depressed outpatients	BA	57	ATQ, BDI	All treatments reduced depression, ATQ, & EASQ; early changes on EASQ predicted change in depression only for BA.
		BA + thought modification	44	EASQ, HRSD	
		Full CT	50		
Kuyken (2004)	Outpatients	CT	122	HS	Early changes on HS predicted depression improvement at termination beyond initial depression severity.
Kwon & Oei (2003)	Outpatients	CT	35	BDI, DAS, ATQ	Early decrease on ATQ associated decrease on BDI & DAS.
Persons & Burns (1985)	Depressed dysthymic depressed-bipolar	CT	17	ATQ	Mood & scores on ATQ changed w/in session; changes on ATQ made independent contributions to changes in mood.
Safran et al. (1987)	Outpatients	Working on beliefs vs. problem solving	19	Measures of problem resolution	Mood & problem resolution improved within session; greater problem resolution associated w/greater cognitive shift; cognitive shift didn't predict changes in mood.
Segal et al. (1999)		PT	29	DAS & mood priming	After successful treatment, only PT subjects showed elevated DAS scores while sad; elevated DAS scores in response to sad mood predicted relapse years later.
		CT	25		
Seligman et al. (1988)	Unipolar & bipolar outpatients	CT	51	BDI, HRSD, ASQ, HRSR	ASQ change correlated w/change on BDI & HRSD; ASQ change predicted BDI & HRSD scores 1 year later.
Teasdale & Fennell (1982)	Depressed outpatients	Exploring thoughts vs. modification	5		Thought modification produced greater belief change & was associated w/greater improvements in mood.
Wilson et al. (1983)	Depressed community members	CT	8	BDI, HRSD, IBT, cognition & pleasant events schedule	Cognitions schedule & IBT improved w/CT & BT; CT > BT on positive cognitive scores at mid-treatment.
		BT	8		
		WL	9		

Notes: Treatments: BA = behavioral activation, BT = Behavioral Therapy, CBT = cognitive-behavioral therapy, CO = combination of CT and PT, CT = Cognitive Therapy, FT = Family Therapy, IPT = interpersonal therapy, PL = placebo, PT = pharmacotherapy, RT = relaxation therapy, WL = waitlist control. Measures: ASQ = Attributional Style Questionnaire, ATQ = Automatic Thoughts Questionnaire, BDC = Burns Depression Checklist, BDI = Beck Depression Inventory, BLAME = Characterological Self-Blame for Depression, CBQ = Cognitive Bias Questionnaire, CEQ = Cognitive Errors Questionnaire, CQ = Cognitions Questionnaire, CRT-IDC = Cognitive Response Test-Irrational depressed category, DAACL = Lubin Depression Adjective Checklist, DAS = Dysfunctional Attitudes Scale, DAS-NSAS = Dysfunctional Attitudes Scale-Need for Social Approval Scale, DAS-PS = Dysfunctional Attitudes Scale-Perfectionism Scale, EASQ = Expanded Attributional Style Questionnaire, HRSD = Hamilton Rating Scale for Depression, HS = Hopelessness Scale, MAQ = Metacognitive Awareness Questionnaire, MSSSI = Modified Scale for Suicidal Ideation, PCCS = Patient Cognitive Change Scale, SAS = Sociotropy-Autonomy Scale, SCL-90 = Symptoms Checklist, SCS = Self-Control Schedule, SDM = Semantic Differential Measure, UNCONTROL = Perceived Uncontrollability of Depression, WOR = Ways of Responding questionnaire.

of some form of cognitive therapy in treating depressive symptoms; (b) include at least one of the previously noted measures of cognition; (c) be written in English; and (d) be published in a peer-reviewed journal. Based on these criteria, a total of 31 studies were identified (Table 1). The studies included 1,841 participants. The majority ($n = 1,631$) of the participants were outpatients, but studies also included inpatients and community members. The studies included both single treatment designs ($k = 8$) and multiple treatment comparison designs ($k = 21$).

COGNITIVE THERAPY AND COGNITIVE PROCESSES IN THE TREATMENT OF DEPRESSION

A number of studies have shown that cognitive therapy appears to result in changes in negative cognitions, and that these changes covary with improvement in depressive symptomatology. For example, Zettle and Rains (1989) examined changes in depressive symptoms and cognitions in depressed individuals after being treated with complete cognitive therapy, partial cognitive therapy, or comprehensive distancing that asks patients to “distance” themselves from their thoughts but not to modify the content of those thoughts. For all groups, depressive symptoms were reduced significantly over the course of treatment, with the cognitive distancing group showing a significantly greater reduction in depressive symptoms than either the complete cognitive therapy or partial cognitive therapy groups. Despite the reduction in depressive symptoms, the participants showed no changes in automatic thoughts over the course of treatment. In contrast, dysfunctional attitudes, as assessed by the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978), decreased significantly over the course of treatment in the complete cognitive therapy and partial cognitive therapy groups, while changes in dysfunctional attitudes for the comprehensive distancing group were nonsignificant.

In a study of depressed individuals undergoing 12 weeks of cognitive therapy, Barber and DeRubeis (2001) found that depressive symptoms improved modestly over the 12-week period and that patients showed significant cognitive improvement as measured by the Attributional Style Questionnaire (ASQ; Seligman et al., 1979), the DAS, and a thought-listing measure called the Ways of Responding Questionnaire (WOR; Barber & DeRubeis, 1992). Moreover, change on the DAS and WOR covaried with change in depression. A year later, Westra, Dozois,

and Boardman (2002) investigated depressive symptoms and cognitions in individuals undergoing 10 weeks of group cognitive therapy. Over the course of treatment, they reported significant reductions in depressive symptoms, in DAS scores, and on a five-item hopelessness scale. In addition, higher levels of initial hopelessness predicted treatment dropout and also distinguished responders from nonresponders.

Oei and Sullivan (1999) assessed changes in cognitions for 67 patients who had undergone 12 weeks of group cognitive therapy. Patients who recovered showed a greater rate of reduction in automatic thoughts than patients who did not recover. Additionally, during the second six weeks of the treatment program, recovered patients showed a greater change in dysfunctional attitudes than did the nonrecovered patients. In a subsequent study that showed similar results, Kwon and Oei (2003) examined scores on the DAS and the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980) in 35 patients undergoing 12 weeks of group cognitive therapy. Using structural equation modeling (SEM) analyses, they found that therapy was associated with a reduction in automatic thoughts early in treatment, which was in turn linked to both a reduction in depressive symptoms and a reduction in dysfunctional attitudes. During the middle portion of treatment, cognitive therapy was associated with a reduction in dysfunctional attitudes that preceded a reduction in automatic thoughts and that was tied to a reduction in depressive symptoms. These results are consistent with proposals that the initial phases of cognitive therapy involve monitoring and challenging automatic thoughts, but that as cognitive therapy continues the focus of treatment turns to identifying and modifying underlying cognitive structures (Beck et al., 1979).

As measured by the Hopelessness Scale (HS; Beck & Steer, 1988), Kuyken (2004) investigated the influence of changes in hopelessness on depression in a sample of 122 depressed outpatients. Results indicated that early changes in hopelessness (i.e., within the first four weeks) predicted improvement in depression at termination above and beyond the initial severity of hopelessness. Moreover, responders to cognitive therapy could be distinguished from nonresponders based on initial changes in HS scores.

Bieling, Beck, and Brown (2004) examined scores on the Sociotropy-Autonomy Scale (SAS; Bieling, Beck, & Brown, 2000) in 149 depressed outpatients undergoing

cognitive therapy. In addition to measuring sociotropy and autonomy, the SAS assesses fear of criticism and rejection, preference for affiliation, independent goal attainment, and sensitivity to others' control. Analyses showed that full and partial responders evinced changes in fear of criticism and rejection and preference for affiliation, whereas these attitudes were unchanged for the nonresponders.

Within-Session Mediation

The studies in the preceding section assessed cognitive mediation over the course of a full trial of cognitive therapy. Other approaches to examining mediation hypotheses are possible: for example, the assessment of mediation within cognitive therapy on a session-by-session basis. Two studies have employed this approach. In one, Teasdale and Fennell (1982) compared the effects of identifying dysfunctional thoughts versus modifying depressive thoughts in a group of five individuals undergoing cognitive therapy. They found that techniques in cognitive therapy used to modify thoughts produced a greater change in beliefs than techniques simply used to explore thoughts. Moreover, changes in beliefs were associated with greater within-session reductions in depressed mood. However, Safran et al. (1987) were unable to replicate these findings using a problem resolution approach; greater problem resolution was associated with cognitive shifts, but cognitive shifts did not predict significant changes in depressive or anxious symptoms.

Using a somewhat different approach, some studies have examined cognitive mediation between sessions. For example, Tang and DeRubeis (1999) found evidence of sudden gains in many participants, which is a sudden and substantial improvement in depression symptoms in one between-session interval. These sudden gains tended to immediately follow critical sessions in which substantial cognitive changes occurred. Thus, their findings suggested that sudden gains in cognitive therapy are triggered by substantial cognitive change and are therefore consistent with cognitive mediation hypotheses regarding the efficacy of cognitive therapy. A subsequent study by Tang, DeRubeis, Beberman, and Pham (2005) replicated these findings.

SPECIFICITY ISSUES: TESTING THE COGNITIVE MEDIATION HYPOTHESIS WITH NONCOGNITIVE TREATMENTS

Although a good deal of research relevant to testing ideas about cognitive mediation derives from studies that focus

on cognitive therapy, these studies assess only one aspect of the cognitive mediation hypothesis. As noted, the second part of cognitive mediation focuses on specificity; that is, are cognitive changes specific to cognitive interventions? Assuming therapeutic change can be mediated by noncognitive processes, the pertinent question in this regard is whether the cognitive change associated with cognitive therapy is more substantial than cognitive changes that might be observed with noncognitive treatments. The majority of such studies compare pharmacological treatments with cognitive therapy, although a small number have compared noncognitive psychosocial treatments to cognitive therapy. We review these latter studies first.

Specificity: Comparisons Between Cognitive Therapy and Noncognitive Psychotherapy

McNamara and Horan (1986) compared the cognitions of depressed patients who were assigned either to cognitive therapy, behavior therapy, cognitive therapy plus behavior therapy, or a control condition. In addition to examining cognitions with the ATQ, a measure of acquired cognitive skills and an independent judge's rating of the quality of clients' verbalized thought processes were also examined. All therapy groups showed significant improvement in depressive symptoms, and cognitive therapy demonstrated significant or marginal effects on all cognitive measures. In contrast, the behavior therapy group showed no significant changes or marginal effects on the cognitive measures.

In a dismantling study, Jacobson et al. (1996) investigated the changes in cognitions of 151 depressed outpatients undergoing three permutations of components of cognitive therapy: (a) behavioral activation alone; (b) behavioral activation and dysfunctional thought modification; and (c) the full cognitive therapy protocol, including behavioral activation, dysfunctional thought modification, and presumably core schema modification. All treatment conditions led to improvements in depressive symptoms, automatic thoughts, and attributions, but there were no differences between the groups on automatic thoughts or attributions. To further investigate the mechanisms of change, Jacobson et al. (1996) examined the temporal relationship between change in depressive symptoms and cognitive changes. They found that early changes in attributional style predicted subsequent improvement in depressive symptoms, but only for the behavioral-activation

treatment group. Cognitive models of depression can theoretically accommodate cognitive changes resulting from behavioral activation, but would, of course, also expect such changes to stem from direct attempts to alter cognition.

In an earlier and somewhat similar approach, Rehm, Kaslow, and Rabin (1987) compared the effects of three treatment approaches on cognitions and depressive symptoms of 104 women: (a) self-control treatment with a behavioral target; (b) self-control treatment with a cognitive target; and (c) a combination treatment with both behavioral and cognitive targets. All treatment conditions showed significant improvement in symptoms on all depression measures, and all three treatments also showed significant improvement in cognitions, but there were no significant cognitive change differences among the groups. Finally, in a very early study, Wilson, Goldin, and Charbonneau-Powis (1983) found that, relative to a placebo condition, both cognitive therapy and behavior therapy produced significant increases in positive cognitions and significant decreases in negative cognitions, with no significant differences between these two groups. There was also a significant decrease in irrational beliefs at post-treatment that was comparable for the active treatments, but differences between active and control treatments just failed to reach significance.

Specificity: Comparisons Between Cognitive Therapy and Pharmacotherapy

A number of studies have evaluated cognitive differences associated with cognitive therapy versus cognitive differences associated with pharmacological interventions. As noted, these studies represent the most appropriate test for the specificity hypothesis in that while “noncognitive” psychosocial interventions can be argued to effect change through cognitive mechanisms, this seems substantially less likely to be the case for pharmacotherapy.

In the first published cognitive therapy–pharmacotherapy specificity study, Rush, Kovacs, Beck, Weissenburger, and Hollon (1981) found that both cognitive therapy and medication produced significant improvement in depression severity and depressive thinking on the HS, but with no significant differences between groups on any of the measures at posttreatment. However, a cross-lagged panel analysis showed that improvements in hopelessness, views of the self, and mood tended to precede changes

in vegetative and motivational symptoms of depression for cognitive therapy between weeks 1 and 2 and weeks 2 and 3, with no consistent pattern of change found for patients who received medication.

In another early study, Blackburn and Bishop (1983) compared the efficacy of cognitive therapy, pharmacotherapy, and a combination of medication and cognitive therapy. Treatments that included at least a cognitive therapy component produced the most improvement in depressive cognition (i.e., negative views of the self, world, and environment). A study published a year later (Simons, Garfield, & Murphy, 1984) compared the effectiveness of cognitive therapy to medication on changes on several cognitive measures. Both medication and cognitive therapy produced significant improvement in dysfunctional attitudes and automatic thoughts, although they were not significantly different from one another. However, cognitive change was associated with improvement in depression for both treatment groups.

Using the Cognitive Errors Questionnaire (CEQ; Lefebvre, 1981) to assess depressive thinking (cognitive distortion), Beutler et al. (1987) compared the effectiveness of (a) medication plus support, (b) placebo plus support, (c) cognitive therapy plus placebo plus support, and (d) cognitive therapy plus medication plus support. Although participants who received cognitive therapy demonstrated a significantly greater reduction in depressive symptoms compared to participants who did not receive cognitive therapy, none of the treatments resulted in a significant reduction in cognitive distortions.

In a study reported by Bowers (1990), participants were assigned to one of three treatment groups for depression: relaxation therapy plus medication, cognitive therapy plus medication, or medication alone. Following treatment, patients in both the cognitive therapy plus medication and relaxation therapy plus medication groups showed lower ATQ scores than the medication alone group, although no differences were found on the DAS or HS. A study that same year by Imber et al. (1990) compared the efficacy of (a) cognitive therapy, (b) medication plus clinical management, (c) interpersonal therapy (IPT), and (d) placebo plus clinical management in the treatment of depression. Participants who received cognitive therapy demonstrated significantly greater improvement on the Need for Social Approval factor of the DAS at posttreatment compared to the IPT and

pharmacotherapy treatments, but cognitive therapy participants did not differ significantly from participants in the placebo control condition on this measure. However, the authors noted that the differences in means on the DAS Need for Social Approval factor between the cognitive therapy group and the placebo group were comparable to the differences found between cognitive therapy and the other active treatments. They noted that the lack of a reliable difference on the Need for Social Approval scale between cognitive therapy and placebo may in part be explained by the fact that participants in the placebo condition had the greatest variation in their performance on this measure compared to participants in other treatment conditions.

DeRubeis et al. (1990) also compared the effectiveness of cognitive therapy with pharmacotherapy in treating depression and for reducing depressive cognition. Both treatments produced significant improvement on all cognitive measures (the ATQ, DAS, HS, and ASQ), and there were no significant differences between groups for cognitive improvement. However, improvement on the ASQ, DAS, and HS predicted subsequent improvement in depression, but only for participants treated with cognitive therapy, suggesting that while both treatments are associated with cognitive change, the mechanisms by which they are associated may be different.

In a particularly thorough study, Whisman, Miller, Norman, and Keitner (1991) examined whether adding cognitive therapy to a standard medication regimen produced increased improvement in depression and changed cognition. Both groups demonstrated significant decreases in automatic thoughts and depressive symptoms. Dysfunctional attitudes were not significantly changed at the posttreatment assessment; they did improve for the cognitive therapy group at six months, but not for the medication group. However, DAS scores increased for the cognitive therapy group and thus at the 12-month assessment no difference was found between this group and the medication group. Finally, hopelessness decreased for both groups, but at each interval period hopelessness was significantly improved for the cognitive therapy group in comparison to the medication group.

McKnight, Nelson-Gray, and Barnhill (1992) compared the effectiveness of cognitive therapy to pharmacotherapy in the treatment of depression. Rather than using the DAS, dysfunctional thoughts were measured with the

Personal Beliefs Inventory (PBI; Muñoz & Lewinsohn, 1976). Both treatments were equally effective in reducing depression symptomatology. Cognitive therapy and pharmacotherapy significantly reduced posttreatment dysfunctional thoughts, with cognitive therapy producing a greater reduction in dysfunctional thoughts.

A study by Fava, Bless, Otto, Pava, and Rosenbaum (1994) found that successful pharmacotherapy treatment was associated with significant reductions on the DAS and the Cognitions Questionnaire (CQ; Fennell & Campbell, 1984) and also found that the degree of decrease in scores on the DAS and CQ was associated with the change in depression severity. Negative thinking and dysfunctional attitudes, however, did not predict the degree of improvement in depressive symptoms. In another study that examined the efficacy of combining pharmacotherapy with cognitive therapy, Stravynski et al. (1994) found that both cognitive therapy and a combination of cognitive therapy and medication were effective in treating depression, but did not find significant differences in clinical outcome or depressive thinking (measured with the ATQ, HS, and DAS) between treatment conditions at posttreatment or at a follow-up assessment.

In a more recent study, Teasdale et al. (2001) compared the efficacy of a treatment that combined cognitive therapy, medication, and clinical management to a treatment that combined medication and clinical management. Teasdale et al. found no significant differences between the cognitive therapy group and the control group on the depression severity measures or on depressive thinking measures. However, a measure of the form of response to all of the cognitive questionnaires, specifically the number of times participants endorsed extreme response categories, showed a differential response to cognitive therapy. In particular, cognitive therapy decreased the sum of extreme responses on questionnaires whereas pharmacotherapy did not. Moreover, when participants who relapsed before eight weeks and those who were not compliant with treatment were excluded, cognitive therapy significantly decreased relapse in comparison to pharmacotherapy and was associated with changes in absolutistic/dichotomous thinking style.

Beevers and Miller (2004) compared the effectiveness of (a) medication, (b) medication plus family therapy, (c) medication plus cognitive-behavioral therapy, and (d) medication plus cognitive-behavioral therapy plus

family therapy on depression severity and depressive thinking. All of the treatment conditions resulted in clinically significant improvement in depression severity, surface-level cognitions, and core cognitive functioning. However, all treatments were equally effective at reducing depressive thinking on the Cognitive Bias Questionnaire (CBQ; Krantz & Hammen, 1979), the DAS-Perfectionism, the HS, and the ATQ.

Mood-Linked Longitudinal Research

Two particularly innovative studies by Segal et al. (1999, 2006) bear on the issue of cognitive mediation, although from a different perspective than the treatment studies previously reviewed. Segal et al. (1999) examined the effect of a temporary sad mood on thinking in participants who had been successfully treated for depression with either medication or cognitive therapy. Such sad mood inductions are frequently used to test the reactivity of depressive thinking patterns in individuals who are vulnerable to depression (Ingram et al., 1998; Segal & Ingram, 1994). They found that when study participants were in a normal mood, there was little evidence of dysfunctional attitudes, as measured with the DAS, and that this was equally true for participants treated with cognitive therapy and participants treated with pharmacotherapy. In a follow-up study, Segal et al. (2006) found very similar results; that is, there were no differences in DAS scores between those treated with cognitive therapy versus pharmacotherapy. However, in both studies, inducing a sad mood was associated with increased dysfunctional attitudes for the pharmacotherapy group but not for the cognitive therapy group. Thus, patients successfully treated with pharmacotherapy for their depression showed significantly greater cognitive reactivity relative to participants successfully treated with cognitive therapy. Furthermore, in both studies this cognitive reactivity (regardless of the type of treatment) was predictive of depression relapse several years later; patients who did not respond with dysfunctional attitudes following negative mood priming were significantly less likely to relapse compared to patients who endorsed dysfunctional attitudes following the sad mood induction. Such findings are consistent with cognitive mediation inasmuch as the likelihood of relapse appeared to be related to how successfully dysfunctional attitudes were modified in treatment. In summary, cognitively treated patients thus showed less

reactivity, and patients who presumably experienced the most cognitive changes were the least likely to relapse.

SUMMARY AND CONCLUSIONS

Despite the wealth of data available in the years since the 1993 review, in some respects the state of affairs is not strikingly dissimilar to that noted by Whisman. In particular, conclusions from extant studies are complicated by vastly different research methodologies, a variety of different cognitive measures, and the varied specific research questions each study addresses. Nevertheless, some progress has been made in the intervening years toward answering the two related questions that underlie assumptions about the mechanisms responsible for the success of cognitive therapy: in particular, does the modification of cognitions appear to mediate therapeutic change, and is cognitive change specific to cognitive interventions?

Does Cognition Mediate Change in Cognitive Therapy?

If the question of cognitive mediation is framed as “do changes in cognition as a function of cognitive therapy predict changes in depression?” then the bulk of available results generally answer in the affirmative. For example, studies have largely confirmed that cognitive improvement precedes and, when tested, predicts improvement in depressive symptoms. (e.g., DeRubeis et al., 1990; Kuyken, 2004; Kwon & Oei, 2003; Rush et al., 1981; Tang & DeRubeis, 1999; Teasdale & Fennell, 1982). It is worth noting that other studies have investigated the cognitive mediation hypothesis for cognitive therapy with anxiety disorders and have provided support for cognitive mediation of symptom improvement (Kendall & Treadwell, 1996, in press). Hence, with reasonably few exceptions, cognitive therapy does appear to be associated with, and predict, positive changes in cognition and clinical improvement. Pharmacotherapy sometimes demonstrates symptomatic improvement that is associated with cognitive change, but cognitive change does not appear to predict symptomatic improvement with medication (e.g., Fava et al., 1994).

Are Cognitive Changes Specific to Cognitive Therapy?

As noted, the prediction of change in cognitive therapy is only one part of the question raised by the proposed mechanisms of cognitive therapy. A complete answer to the question must also address whether cognitive changes

are specific to cognitive interventions. As we have discussed, studies that bear on this question typically compare post-treatment cognitions after successful cognitive therapy (i.e., treatment that results in significant improvement in depressive symptoms) to posttreatment cognitions after other successful treatments. This specificity hypothesis is supported when the cognitive change associated with cognitive therapy is significantly greater than cognitive change that is associated with comparison treatments; although some change may be seen with other treatments, the degree of cognitive change associated with cognitive therapy should be significantly larger.

Comparison treatments can be psychosocial in nature or, much more commonly, pharmacological. This distinction is important inasmuch as psychosocially produced changes in cognition are more consistent with cognitive proposals than are pharmacologically produced changes in thinking. Specifically, and in a manner similar to cognitive therapy, it is quite plausible that psychosocial treatments achieve their effects by altering patients' perceptions of themselves and their environment in ways that promote more effective interactions and less depressive distress. Drawing the same conclusion about the mechanisms of change in pharmacological treatments is considerably more difficult. As we have noted, although it cannot be ruled out that pharmacological agents achieve efficacy by altering cognitions, compelling theoretical proposals specifying such effects have yet to be suggested.

The most appropriate tests of the specificity of cognitive therapy are thus seen in studies comparing cognitive therapy and pharmacological treatment. In our review, the results of cognitive specificity studies are more mixed than the body of research showing a link between cognitive changes and symptom reduction in cognitive therapy. As defined by significant differences in cognition between cognitive therapy and pharmacological treatments, our review finds five studies suggestive of specificity in cognitive–pharmacological comparisons (Blackburn & Bishop, 1983; Imber et al., 1990; McKnight et al., 1992; Teasdale et al., 2001; Whisman et al., 1991) and eight that are not (Beevers & Miller, 2004; Beutler et al., 1987; Bowers, 1990; Rush et al., 1981; Segal et al., 1999, 2006; Simons et al., 1984; Stravynski et al., 1994). Moreover, study findings did not depend on type of measure used to assess cognition (i.e., significant group differences were not a function of cognitive measures used in the study).

Given the lack of uniform findings, or the somewhat larger number of nonsupportive studies, can the specificity hypothesis then be disconfirmed? A myriad of possibilities suggest why the answer should be no. For example, in all but the most tightly controlled studies, it is possible that cognitive therapy may not have been delivered in an optimum fashion, or perhaps patients in treatment receiving pharmacotherapy may also have received some psychosocial treatment. Aside from these speculative possibilities, however, careful consideration of the data at hand suggests that it would be inappropriate to conclude that this hypothesis can be disconfirmed. In fact, two lines of evidence lend support to the idea of specificity. First, the clear majority of nonsupportive studies showed larger reductions on cognitive measures in the cognitive therapy group than in the pharmacotherapy group. Although not statistically significant, differences were in the direction predicted by the specificity hypothesis. More importantly, however, examination of sample sizes reveals that nonsupportive studies had on average substantially smaller samples than supportive studies. For example, mean sample sizes for supportive studies ($M = 34.62$) were larger by roughly half compared to nonsupportive studies ($M = 22.14$). It may thus be that although these studies were adequately powered to find treatment effects, they were less so to find significant cognitive differences.

Second, it is important to consider the intriguing line of evidence found in the data reported by Segal et al. (1999, 2006). In both studies, Segal et al. found no significant differences at posttreatment on DAS scores between patients treated with cognitive therapy and patients treated with pharmacotherapy. However, they found that, as a group, depressed participants treated with pharmacotherapy were cognitively reactive to depressed mood, whereas the patient group treated with cognitive therapy was much less so. Additionally and more critically, cognitive reactivity was associated with a higher probability of relapse. Thus, although successful pharmacotherapy for depression was associated with reductions in negative cognitions in some cases, these negative cognitions also easily resurfaced. It may therefore be that the cognitive changes seen in pharmacotherapy are superficial compared to the changes produced by cognitive therapy; cognitive therapy, by design, attempts to produce change in “deep” cognitions that are less likely to resurface in the manner seen in pharmacologically treated patients.

Moreover, to the extent that negative cognitions play some causal role in depression onset, the fact that cognitive reactivity was predictive of depression relapse several years later also lends support to the specificity component of the mediation hypothesis. That is, if cognitions play a role in the onset of depression, then those with only superficial cognitive changes linked to an intervention should be more likely to relapse. This line of thinking is augmented by the data showing that cognitive therapy has considerably greater efficacy than pharmacotherapy in preventing future episodes of depression (Hollon et al., 2006). Thus, deeper cognitive change may help to explain at least one mechanism by which cognitive therapy demonstrates superior efficacy to pharmacotherapy for reducing subsequent relapse risk, and is consistent with the cognitive specificity hypothesis (i.e., the absence of cognitive reactivity is specific to cognitive therapy).

Despite these important qualifications on studies that, at first glance, run counter to specificity proposals, we nevertheless cannot conclude that the hypothesis is confirmed at this point. Yet, as we have noted the evidence also clearly does not suggest that this hypothesis can be disconfirmed. Most conservatively, it seems reasonable to conclude that the available data suggest that the proposition that cognitive therapy produces changes that are specifically and causally linked to symptomatic reduction remains quite promising. The data show that cognitive changes do appear to predict symptomatic changes, adequately powered studies show evidence of specificity, and there is some evidence to suggest cognitions that are changed by pharmacotherapy may be only superficially changed.

In the nearly 40 years since cognitive therapy was developed (i.e., Beck, 1967) and ideas about its effects on cognition in treatment were proposed, it is perhaps surprising that an unambiguously clear answer regarding specificity in comparison to medication is not yet apparent. It is important to note in this regard, however, that much of the research that is relevant to questions of cognitive mediation typically addresses these questions as secondary to a focus on treatment efficacy questions. Comparatively, few studies have specifically sought to test the specificity hypotheses with adequate power and a range of cognitive measures that address broad and meaningful cognitive constructs.

More generally, although it is important that cognitive therapy demonstrate specificity, the specificity question

itself may imply a false dichotomy. For example, even if similar and deep cognitive changes for cognitive therapy and pharmacotherapy are assumed, the actual mechanisms of change may be different (Hollon et al., 1987). A possibility, consistent with the data of Segal et al., (1999, 2006) is that pharmacological treatment reduces depression, and in so doing, negative cognitions diminish. These cognitive changes then are consequences of effective treatment, but in this form of treatment they do not play a causal role in recovery. The change in cognitions in cognitive therapy, on the other hand, may be the critical factor leading to reductions in depression. On the surface then, cognitive therapy and pharmacotherapy may look similar on some cognitive outcomes, but the mechanisms of symptom change and cognitive change may nevertheless be quite different. This perspective is consistent with ideas that cognitions and emotion represent reciprocal aspects of the same process, and that intervention at one point in the system (e.g., cognition or dysfunctional emotion via pharmacotherapy) will affect other points in the system (Hollon et al., 1987).

CONCLUDING COMMENTS

The idea of cognitive mediation in cognitive therapy remains quite promising; data show that changes in cognition predict symptomatic changes, and a plausible case can be made for specificity. Yet, it is important to note that DeRubeis et al. (1990) proposed several criteria for a cognitive mediator in cognitive therapy. We are unaware of any individual studies that are able to meet all these criteria, and even as a body of research, studies have not yet been able to address these criteria in ways that permit clear-cut answers. It has been 14 years since the last review of this body of literature; perhaps the next 14 years will see data that do address all of these criteria in methodologically sound ways.

It is also worth noting that some statistical methods, not previously widely in use, may help to fashion more clear answers to questions about cognitive mediators. For example, SEM may be helpful when comparing cognitive outcomes for various treatments for depression. SEM possesses several advantages in comparison with the techniques that have typically been used by studies comparing cognitive outcomes for different treatments for depression (e.g., ordinary least squares). These include the ability to compare the adequacy of each theorized

model (e.g., cognitive change leads to improved mood or improved mood leads to cognitive change) for explaining the data, and the ability to compare models in order to determine the model that best fits with the data. The use of SEM techniques would enable an interpretation of results that extends beyond changes in cognitions preceding (or predicting) changes in mood for cognitive therapy to demonstrating a causal model for depression recovery with different treatments.

Recruitment of larger sample sizes and the use of statistical methods such as SEM, along with a broader array of cognitive measures, should improve researchers' ability to answer questions about the mechanisms of cognitive therapy. Although mediation in cognitive therapy has been the focus of much interest, such statistical methods might also be applied to other approaches in regard to the mechanisms that they propose to be at the heart of therapeutic change. When considered along with data on cognitive mediators, such data may also help point the way toward the factors that are common to therapeutic change across different psychotherapeutic approaches (e.g., Norcross & Goldfried, 2005). Cognitive factors almost certainly will play an important role in common factors that are responsible for positive changes.

NOTE

1. The terms *cognitive therapy* and *cognitive-behavioral therapy* as used here are considered interchangeable, although it is important to acknowledge that different investigators may use these terms to denote different procedures with different theoretical assumptions. Even though we consider these interchangeable, we use the term *cognitive therapy* throughout this article. Additionally, a number of different therapies can be considered cognitive in nature (e.g., problem-solving therapy, rational-emotive therapy). We limit our discussion to the version of cognitive therapy articulated by Beck.

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