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THE SEARCH FOR MODE-SPECIFIC EFFECTS OF COGNITIVE AND OTHER THERAPIES: A METHODOLOGICAL SUGGESTION

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Abstract

The hypothesis that the unique mechanism ofaction ofcognitive therapy (CT) for depression involves change in underlying cognitions (schema) has not been supported by empirical studies; instead, many therapies seem to produce cognitive change. Likewise, evidence fails to support the hypothesis, drawn from the cognitive theory, that change in underlying cognitions protects patients from relapse. We argue that methodological problems may explain these empirical disconfirmations ofthe cogni-tive theory. In particular, the failure to activate latent underlying cogni-tions before assessing them prevents investigators from adequately testing mode-specific hypotheses about the mechanism of action of CT. A similar difficulty may plague tests ofmode-specific hypotheses about the mechanisms of action of other therapies.

Empirical evidence frequently fails to support the statements made by Beck's cognitive theory (see Beck, Rush, Shaw, & Emery, 1979) about the etiological role of underlying cognitions in mediating both therapeutic change during cognitive ther-apy (CT) and symptomatic relapse in recovered patients. After reviewing the theory's predictions and the status of the empirical evidence, we propose a solution to this problem that has methodological implications for future studies of the mechanism of action of CT. Because the problem we address involves difficulties arising in the assessment oflatent cognitive structures, and because many theories of psychotherapeutic change postulate changes in latent cognitive structures, we believe our suggestions are applicable to many psychotherapies, not just CT.

THE COGNITIVE THEORY

The cognitive theory proposes that the central mechanism of action of cognitive therapy is cognitive change (Beck et al., 1979). Two types of cognitive change are expected to occur: change in "automatic thoughts," and change in underlying cognitions. "Automatic thoughts," as described in the model, are readily available ideas about ongoing experience. For example, when, to obtain the patient's "automatic thoughts" about a situation, the therapist usually asks directly, "What thoughts are you having that are causing you to feel so upset about that situation?" the therapist is asking for the patient's "automatic thoughts."

Underlying cognitions, also termed dysfunctional attitudes, or schemas, are the organized structures of past reactions and experiences that form a cohesive body of knowledge that guide subsequent perceptions (Beck, Freeman, & Associates, 1990). 1 According to the theory, the underlying beliefs, or schemas, are not readily available for recall; Beck describes them as "latent" (and they might even be described as "unconscious"). Dysfunctional attitudes must be activated in order to play any role in a person's current functioning and emotional experiences. For example, Beck proposes that individuals who hold beliefs like, "I must be loved or I am worthless," are vulnerable to depressive symptoms if they experience a life event, such as a rejection or loss, that causes them to feel unloved by someone important to them.

Thus, in general terms, the cognitive theory predicts that underlying dysfunc-tional attitudes or schemas are activated by life events to produce symptoms and automatic thoughts. The theory also predicts that cognitive therapy is effective because it teaches patients to reduce distortions in their automatic thoughts and dysfunctional attitudes, thereby obtaining both symptom relief and protection from relapse.

In contrast, non-cognitive therapies are viewed by the cognitive theory as operating via other, non-cognitive, mechanisms. Other therapies, such as pharmacotherapy, are viewed as operating by improving mood and physiological functioning but, according to the cognitive theory, would not be expected to operate via cognitive mechanisms. According to this view, patients treated with pharmacotherapy would be expected, at the end of therapy, to retain the dysfunctional attitudes that activated their depression, and therefore to be vulnerable to relapse should those dysfunctional attitudes be activated again. Empirical findings, however, contradict the cognitive theory's predictions about the role of the underlying cognitions in the mechanism of action of the therapy and in relapse protection. We review the evidence in each area.

EMPIRICAL EVIDENCE: MECHANISMS OF ACTION OF COGNITIVE THERAPY

Contrary to the theory, cognitive therapy has not been shown to be superior to other therapies in producing changes in dysfunctional attitudes. Table 1 presents the results of seven studies addressing this issue. As can be seen, the studies unanimously fail to find that cognitive therapy is superior to non-cognitive therapies in changing underlying dysfunctional attitudes. In all cases, change in dysfunctional attitudes was similar across treatments. Similarly, Reda, Carpiniello, Secchiaroli, and Blanco (1985) reported substantial changes in dysfunctional attitudes in patients treated only with pharmacotherapy.

Results of the Collaborative Study are somewhat more complex than depicted in Table 1. Patients treated with cognitive therapy did not show more change in dysfunctional attitudes (as measured with the Dysfunctional Attitude Scale; DAS) than patients treated with interpersonal therapy, pharmacotherapy, or placebo. However, there *were* some differences between treatment modalities when scores on the DAS Need for Social Approval subscale were examined. Patients treated with cognitive therapy did change more on this measure than patients treated with interpersonal therapy or pharmacotherapy-but they did not change more than patients treated with placebo.

Table 1 presents one mixed finding. In DeRubeis and associates' (1990) study, changes in dysfunctional attitudes and in attributions predicted change in depres-sion in patients who received cognitive therapy but not in patients who received another type of treatment. Despite this one mixed finding, the overall results to date do not support the hypothesis that dysfunctional attitudes respond to cognitive therapy but not to other treatments for depression.

EMPIRICAL EVIDENCE: MECHANISMS OF RELAPSE PREVENTION

The theory's predictions that cognitive therapy produces changes in dysfunctional attitudes and that dysfunctional attitudes fuel relapse lead to the prediction that patients treated with cognitive therapy ought to relapse less frequently than those treated with non-cognitive therapies. Several controlled outcome studies have re-ported this result (Blackburn, Eunson, & Bishop, 1986; Evans et al., 1992; Simons, Murphy, Levine, & Wetzel, 1986).

However, this result is not a strong test of the cognitive theory, as this pattern of findings is also consistent with other proposed mechanisms of action of the therapy the view that the therapy teaches coping skills, for example (Barber & De-Rubeis, 1989). If, as Barber and DeRubeis (1989) proposed, patients learn coping skills in CT, then they can use them at the first sign of relapse and thus protect themselves from a full-blown recurrence.

To get closer to the mechanism of action of the therapy, several investigators have examined relationships between dysfunctional attitudes at posttreatment and subsequent relapse or recurrence. Unfortunately, studies of this question do not provide much support for the proposed mechanism of action of the therapy. In the NIMH Treatment of Depression Collaborative study, dysfunctional attitudes at termination did not predict clinical status at 6-, 12-, or 18-month follow-up for either the total sample of treatment completers or the smaller sample of treatment responders (Leber, Beckham, & Watkins, 1990). A survival analysis of time to relapse also failed to find an effect for dysfunctional attitudes at termination. Similarly, Seligman, Castellon, Cacciola, Schulman, Luborsky, Ollove, and Downing (1988) reported that when initial depression was controlled, negative attributional style did not predict depression severity at one-year follow-up.

Two studies report mixed results. Evans and colleagues (1992) found that one measure of cognitive vulnerability to depression (attributions) predicted relapse, but another measure (dysfunctional attitudes) did not. Rush, Weissenburger, and Eaves (1986) reported that one of three measures of depressive symptoms obtained 6 months following recovery from depression was a function of posttreatment dysfunctional attitudes but not negative attributions.

One clear positive (consistent with the cognitive theory) finding was reported by Simons and colleagues (1986), who found that patients ending treatment with higher levels of dysfunctional attitudes were more likely to relapse during the year following treatment than patients ending treatment with lower levels of dysfunctional attitudes, even when level of termination depression (BDI) was controlled. Overall, however, studies of the relationship between posttreatment dysfunctional attitudes and relapse do not provide much support for the cognitive theory.

Table 1. Comparative Studies of Mechanisms of Action of Cognitive Therapy for Depression

Investigators	*N*	Change in Dysfunctional Attitudes during Treatment
Zeiss et al., 1981	66	CT = BT = social skills
Blackbum & Bishop,	64	CT not consistently superior to
1983 Simons et al., 1984	28	antidepressants CBT = antidepressants
de Jong et al., 1986 Rehm	30	CBT = BT
et al., 1987 DeRubeis et	104	CT= BT= CBT
al., 1990 Imber et al.,		CBT = antidepressants
1990		CBT = antidepressants = IPT = placebo

Note.--CT = cognitive therapy; CBT = cognitive-behavior therapy; BT = behavior therapy; IPT = interpersonal therapy.

ACCOUNTING FOR EMPIRICAL EVIDENCE CONTRADICTING THE COGNITIVE THEORY

Several investigators have suggested that the failure to find mode-specific effects of cognitive (and other) therapies reflects the fact that therapeutic efficacy derives from common factors inherent in all therapies, such as a sense of hope and optimism, a convincing treatment rationale, and/or an important relationship with the therapist (Frank, 1982; Waterhouse & Strupp, 1984; Zeiss, Lewinsohn, & Munoz, 1979). Yet another view has been offered by Horowitz and colleagues (1983), namely, that all effective therapies produce changes in all mechanisms--that is, for example, both cognitive therapy and pharmacotherapy produce both cognitive and biological change. Butler and Strupp (1986) argue that repeated failure to demonstrate mode-specific therapeutic effects suggest that the search for mode-specific effects in psychotherapy is a misguided enterprise.

Others have coped with the failure to demonstrate mode-specific effects by developing alternative accounts of the mechanisms of action of cognitive therapy. Barber and DeRubeis (1989) recently proposed that cognitive therapy is effective because it teaches compensatory skills. Baron, Baron, Barber, and Nolen-Hoeksema (1990) suggested that cognitive therapy consists of training in rational thinking strategies.

Yet another way of accounting for the available evidence was offered by Hollon, DeRubeis, and Evans (1987), who argue that the evidence that cognitive change occurs in non-cognitive therapies does not necessarily contradict the theory that cognitions are the mechanism of change in CT, but not in other therapies. They point out that cognitions may cause change in CT but be the consequence of change in other therapies. An explanation of this sort may explain the recent finding that obsessive-compulsive patients treated with behavior therapy showed physiological changes equal to those seen in obsessive-compulsive patients successfully treated with pharmacotherapy (Baxter et al., 1992). Cognitive or biological changes may be the consequences of another mode of change.

We propose a different account of the fact that the available evidence con-tradicts the cognitive theory. We suggest that the failure to demonstrate mode-specific effects of cognitive therapy (and perhaps other psychotherapies) is due to inadequate assessment of the proposed mechanisms of the therapy. In particular, we suggest that investigators are assessing underlying cognitions that are "latent" without ascertaining that the cognitions have been activated and are accessible and reportable by the subject being studied. We believe that, unless the underlying cognitions have been activated, either by the individual's ongoing spontaneous life events or negative mood states, or by a purposeful procedure such as a mood induction, the researcher will not be able to assess these cognitions accurately.

We hypothesize that dysfunctional underlying cognitions are only accessible and readily reportable after they have been activated, or primed; we call this the "activation hypothesis."2 Following a description of the activation hypothesis and a brief overview of some of the evidence supporting it, we show how it might account for the evidence contradicting the cognitive theory that we just presented. We also suggest, based on the activation hypothesis, some methodological changes in the design of studies of mode-specific effects of cognitive therapy and other therapies.

THE ACTIVATION HYPOTHESIS

The activation hypothesis states that an individual who holds underlying dysfunctional attitudes that make him/her vulnerable to depression will not report those cognitions unless they have been activated. This proposal is not original. This idea is the heart of the diathesis-stress view of depression described in several prominent cognitive theories (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978; Beck, 1976; Brown & Harris, 1978; Oatley & Bolton, 1985). In all these theories, underlying vulnerabilities to depression are "latent" until activated by life events, negative mood state, or other factors.

In Beck's theory, life events are viewed as the most important type of activating mechanism (Beck et al., 1979). Others have suggested that negative mood might also serve as an activating, or priming, stimulus for dysfunctional attitudes (In-gram,1984; Miranda & Persons, 1988; Riskind & Rholes, 1984; Segal, 1988; Segal & Shaw, 1986; Teasdale, 1988).

EVIDENCE SUPPORTING THE ACTIVATION HYPOTHESIS

Smith, Ingram, and Brehm (1983) studied the role of life events in activating cognitive vulnerabilities in an experimental setting. They demonstrated that socially anxious college students showed cognitive deficits on a depth-of-processing task that tapped the individual's specific vulnerability (concern about evaluation by others). As predicted by the activation hypothesis, the cognitive deficit occurred only for socially anxious students, and only when these students were in a social evaluative situation that "activated" the underlying cognitive vulnerability (concern about evaluation by others). Miranda (1992) demonstrated that nondepressed subjects showed elevated scores on two measures of dysfunctional thinking if they were vulnerable to depression (had a past history of DSM-III major depression) and had experienced recent stressful life events, which presumably activated the latent cognitive vulnerability. In contrast, individuals who had no past history of depression, no recent stressful life events, or only one of these factors, did not report dysfunctional attitudes. Again, the dysfunctional thinking was measurable only under circumstances where latent cognitions were activated by life events.

These two studies show that life events appear to "activate" or "prime" cogni-tive vulnerability factors. Ingram (1990a) recently proposed that self-focused atten-tion can activate schemas as well.

More recently, investigators have shown that negative mood also activates underlying dysfunctional cognitions in vulnerable individuals. Miranda and Persons (1988) and Miranda, Persons, and Byers (1990), showed that dysfunctional attitudes changed as mood changed. Miranda and Persons (1988) asked 43 nondepressed women to rate their mood and complete a measure of dysfunctional attitudes (the Dysfunctional Attitude Scale; DAS; Weissman, 1979) before and after a Velten-type mood induction.

Reported dysfunctional attitudes varied as a function of mood for those subjects with a history of depression DSM-III-R major depression. Miranda and associates (1990) asked depressed psychiatric patients to report their mood and dysfunctional attitudes twice during a 24-hour period: when their moods were best and worst. As predicted, dysfunctional thinking varied with mood; it was most severe when mood was most depressed.

These two studies, as well as a third (Miranda, Persons, & Gross, 1994) also showed that reporting of dysfunctional attitudes is mood-state dependent for in-dividuals who are vulnerable to depression but that this relationship does not hold for nonvulnerablc individuals. Miranda and Persons (I 988) and Miranda and colleagues (1990) compared individuals vulnerable to depression with individuals not vulnerable to depression. Vulnerability to depression was determined by selecting subjects who reported a previous episode of depression. For those vulnerable to depression, dysfunctional attitudes were a function of current mood: the more negative the individuals' mood, the higher the DAS score. Mood and DAS were unrelated for nonvulnerable individuals. Miranda and colleagues (1994) assessed mood and dysfunctional attitudes in women vulnerable to depression and in non-vulnerables before and after a film negative mood induction. They showed that mood and dysfunctional attitudes were positively related (the more negative the mood, the more dysfunctional the thinking) for vulnerables but not for nonvulner-ables. (In fact, unexpectedly, mood and dysfunctional attitudes were negatively related-the more negative the mood, the less negative the thinking-for non-vulnerables.)

In a similar study, Ingram (1990b) compared nondepressed college students who had received a diagnosis of major depression within the last three years with students who had never been depressed. He showed that vulnerable subjects (those with a past depression) were excessively distracted by negative words they were told to ignore on a dichotic listening task, but that this deficit only appeared in subjects who had received a negative mood induction. The nonvulnerable subjects did not show the deficit regardless of their mood state.

In another study of the activating effects of mood on cognition, Teasdale and Dent (1987) showed (on one of two measures of "self-schema") that vulnerable women have a more negative self-schemas than nonvulnerable women following, but not preceding, a musical mood induction. To assess self-schema, Teasdale and Dent (1987) used two tasks. First, subjects read negative and positive trait adjectives and indicated which described themselves (this is the "rating" task); a few moments later, in an incidental recall task, subjects recalled the adjectives they had endorsed (this is the "recall" task). To control for the fact that vulnerables tended to have a sadder initial mood state than nonvulnerables, the investigators discarded subjects to create matched groups. When this was done, vulnerables and nonvulnerables did not differ on either self-schema measure before the sadness induction. As predicted, the vulnerables and nonvulnerables did differ following the sadness induction (on the "recall" but not on the "rating" task).

Several of these studies could be viewed as having activated cognitive disorgani-zation (e.g., tracking deficits on a dichotic listening task) rather than having activated schemas. The results of Smith and associates (1984) and Ingram (1990b) might be viewed in this way. However, several of the other studies, particularly those by Miranda and colleagues, as well as the one by Teasdale and Dent (1987) do attempt directly to measure the schemas and dysfunctional attitudes described by Beck in this cognitive theory as playing the role of cognitive vulnerability factors for depression.

Thus, several studies have shown that evidence of dysfunctional thinking in vulnerable individuals appears when activating stimuli (mood or life events) have primed otherwise latent underlying cognitions. This line of thinking is consistent with attempts by personality psychologists (cf. Mischel, 1973) to resolve the person versus situation debate suggesting that behavior is a product of situation-person interactions. In the studies reviewed, dysfunctional thinking was a product of the situation (stress/ negative mood) and person (vulnerable/nonvulnerable) interac-tion.

USING THE ACTWATION HYPOTHESIS TO ACCOUNT FOR EVIDENCE CONTRADICTING THE COGNITIVE THEORY

We argue that the activation hypothesis can account for negative findings in studies of mechanisms of action of cognitive therapy and in studies of relapse and recurrence of depression following cognitive therapy. To understand our argument, remember that the usual strategy for testing the hypothesis that cognitive change is the mechanism of action of the therapy involves assessing underlying cognitions, such as dysfunctional attitudes, before and after treatment for depression. Thus, assessments occur at two time points, one when the patient is clinically depressed (in a negative mood state), and one at posttreatment, when the average patient has recovered from depression (in a more positive mood state). We hypothesize that patients in all treatment groups appear to show improvement in dysfunctional attitudes between pre- and posttreatment simply because reporting of dysfunctional attitudes (cognitions) depends on activation of the attitudes. At the beginning of treatment, patients readily report dysfunctional attitudes (because their negative mood state activates these attitudes); at the end of treatment they do not readily report dysfunctional attitudes (because they are in a positive mood), even if the dysfunctional attitudes are (latently) present. Similarly, we hypothesize that underlying cognitions are poor predictors of relapse and recurrence in many studies because they were not activated before they were assessed. Unless underlying cognitions have been activated, they are not likely to be assessed with accuracy.

Thus we suggest that use of an activation procedure before assessing underlying cognitions at posttreatment is more likely than the current research strategies to produce evidence that is consistent with, rather than contradictory to, the cognitive theory. That is, if dysfunctional attitudes in patients treated with CT and with pharmacotherapy were assessed at posttreatment following a mood induction, results might support the prediction of the cognitive theory that more change in underlying cognitions (dysfunctional attitudes, schemas) occurs in patients receiving CT than in those receiving pharmacotherapy.

What are other possible results of a comparative outcome study that uses a posttreatment activation procedure before assessing the underlying cognitions? What implications do these other possible findings have for the cognitive theory?

Suppose that a study comparing CT and pharmacotherapy, for example, showed that neither therapy produced schema change. This finding would disconfirm the predictions from the cognitive theory that the therapy's active mechanism is schema change and the therapy produces schema change. This finding would be more consistent with the Barber and DeRubeis (1989) and the Baron and associates (1990) proposals that short-term cognitive therapy teaches compensatory skills and rational thinking but does not produce underlying cognitive change. However, we note that to test these differing hypotheses about the nature of the cognitive change that occurs in cognitive therapy (Is it schema change, or change in compensatory skills, or an increase in rational thinking?), activation procedures are needed (Per-sons, 199 3).

Suppose that a study comparing CT and pharmacotherapy showed that both therapies produced schema change, and that CT did not produce significantly more schema change than pharmacotherapy. This finding would disconfirm the theory, if we make the assumption that a therapy operating via schema change mechanisms ought to produce more schema change than therapies operating via non-cognitive mechanisms (Hollon et al., 1987).

Finally, we offer another idea about why both cognitive and non-cognitive therapies, as they are currently studied, seem to produce schema change (see Table 1). In our clinical experience, patients are quite resistant to reporting dysfunctional attitudes just as they are recovering from a depressive episode, as if they are reluctant to activate material that might rekindle their symptoms. This hypothesis is consistent with the observation that in the weeks and months following recovery, dysfunctional attitudes appear to "rebound" to a level slightly higher than the immediate posttreatment level (see Reda et al., 1985; Seligman et al., 1988; both studies show this pattern in the data). This hypothesis is also consistent with Blackburn and Smyth's (1985) finding that they could induce negative mood in non-psychiatric controls but not in recovered depressed and anxious patients who were tested approximately three months after recovery from an acute episode. Unless a method for bypassing this protective mechanism can be developed, it might be necessary to wait some weeks or months after recovery from an acute episode before assessing posttreatment dysfunctional attitudes in vulnerable individuals.

IMPLICATIONS FOR STUDIES OF MODE-SPECIFIC EFFECTS OF OTHER PSYCHOTHERAPIES

We argue that accurate assessment of underlying cognitive mechanisms requires an activation procedure prior to assessment. Negative mood or stressful life events can activate these attitudes. Underlying cognitions are probably "naturally" activated in clinically depressed subjects, but if subjects are not clinically depressed, an activation procedure is advised before assessing underlying dysfunctional cognitions. We recommend using a mood induction or some other procedure to activate latent dysfunctional cognitions before assessing them at the end of treatment in com-parative outcome studies or in attempts to predict relapse and recurrence.

Although our discussion here focuses on cognitive therapy for depression, the importance of activation procedures when assessing latent structures applies to other treatments for other problems as well. The notion that latent structures must be activated before they are assessed is well established in the study of the anxiety disorders; posttreatment assessment of therapy outcome in the anxiety disorders literature routinely includes exposing the patient to the fear-evoking situation (cf. Barlow, Mavissakalian, & Schofield, 1980). Accurate assessment of other latent cognitive constructs, such as core conflictual relationship themes (Luborsky, 1977), the Plan Formulation (Weiss, Sampson, & the Mount Zion Psychotherapy Research Group, 1986), and the role relationship models configuration (Horowitz, 1989), for example, are also likely to require activation procedures. The current movement to integrate cognitive and psychodynamic therapies is currently under way, and studies of combined treatments may need to measure more than one type of latent structure; we recommend the use of activation procedures in these studies as well. In fact, accurate diagnostic assessment in past psychiatric disorders may require an activation procedure. Goodwin and Sher (1993) recently found that current negative mood increased report of lifetime symptoms of depression, presumably increasing accuracy of the symptom report by priming recall. In sum, we recommend the use of activation procedures, such as mood inductions, whenever the psychotherapy researcher is assessing "latent" and otherwise unaccessible psychological character-istics in asymptomatic individuals.

NOTES

1. We treat the terms "underlying cognitions," "underlying beliefs," "schema," and "dysfunctional attitudes" as synonymous.
2. In an earlier paper (Persons & Miranda, 1992), we called this idea the "mood-state hypothesis." We use the label "activation hypothesis" here to reflect the fact that other factors, not just mood, can serve to activate the latent schema.

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