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# Cognitive Theories of Vulnerability to Depression: Reconciling Negative Evidence

**Jacqueline B. Persons and Jeanne Miranda**
**University of California, San Francisco**

Cognitive theories of depression propose that stable beliefs predispose vulnerable individuals to depression. Empirical evidence appears to contradict the stability hypothesis; the cognitions described by the theories appear to covary with depressive symptoms. As a result of these findings, many investigators have concluded that the etiological portions of the cognitive theories are inco"ect. We propose an alternative account of the empirical evidence that is consistent with the theories. We propose that the beliefs that are vulnerability factors for depression are stable, but they are accessible only during negative mood states; we call this the mood-state hypothesis. This article describes the mood-state hypothesis, reviews evidence supporting it, and outlines its implications for the cognitive theories of depression. We also describe implications of the mood-state hypothesis for other theories of depression, including biological theories, for studies of psychotherapy process, for studies of other psychopathologies, for epidemiological studies of depression, for the prevention and treatment of depression, and for theories of cognition.

This article proposes a solution to an important problem encountered by cognitive theories of depression. Beck's cognitive theory (Beck, Rush, Shaw, & Emery, 1979), the reformulated learned helplessness theory (Abramson, Seligman, & Teasdale, 1978), and the hopelessness theory (Abramson, Met-alsky, & Alloy, 1989) propose that stable cognitive structures (dysfunctional attitudes in the case of Beck's theory and attributions in the case of the other theories) predispose vulnerable individuals to depression. A problem arises because the empirical evidence appears to show that the cognitions described by the theories are not stable; in fact, they appear to fluctuate in tandem with depressive symptoms (see review by Barnett and Gotlib, 1988). In this article we describe the mood-state hypothesis and show how it solves this problem for the theories. Specifically, we propose that the cognitive structures described by the theories are stable, but the ability to report. them is mood state dependent.

The article begins with a brief description of the cognitive theories, followed by a review of evidence that appears to refute the theories. Next, we describe the mood-state hypothesis and show how it accounts for this negative evidence in a way that is consistent with the theories. Next, we review evidence supporting the mood-state hypothesis. Finally, we describe implications of the mood-state hypothesis for cognitive and biological theories of depression, for studies of psychotherapy process, for studies of other psychopathologies, for epidemiological studies of depression, for the pre-vention and treatment of depression, and for theories of cognition.

## COGNITIVE THEORIES OF DEPRESSION

According to Beck's cognitive thoery, underlying dysfunctional attitudes are stable personality traits that are "latent" in vulnerable individuals. When these individuals experience a stressful life event that activates their belief, depressive symptoms result (Beck et al., 1979). For example, a per-son who believes "I am nothing if a person I love doesn't love me" is vulnerable to depression following the loss of an important relationship. A person who believes "If I fail at my work, then I am a failure as a person" is vulnerable to depression following a failure at work. Beck (1983) labeled these two particular types of vulnerability sociotropy ( or dependency) and autonomy.

According to the learned helplessness theory, attributional style is a stable personality trait that determines the nature, duration, and generality of depressive symptoms (Abramson et al., 1978). A "helplessness depression" results when a person who experiences a negative life event (or fails to experience a desired positive event) perceives that he or she is helpless to change the circumstances of the event and expects this helplessness to persist in the future. When this happens, the individual experiences the cognitive, motivational, and affective symptoms described by helplessness theory.

theory. If, in addition, the individual makes an internal attribution about the cause of the negative event ("I failed at medical school because I'm stupid"), then she will experience a loss of self-esteem. If she makes a stable attribution about the cause of the negative event that is, infers a cause (e.g., stupidity) that is likely to be stable over time, the depressive deficits are expected to persist over time. If she makes a global attribution (e.g., stupidity), the deficits are expected to affect many areas of her life.

In a revision of the learned helplessness theory, Abramson et al. (1989) proposed the hopelessness theory of depression. The hopelessness theory emphasizes hopelessness, not helplessness, as the proximal sufficient cause of depression. Like helplessness theory, hopelessness theory views attributional style as a stable factor that predisposes vulnerable individuals to depressive symptoms, although the role of the particular attributional dimensions is somewhat different. In hopelessness theory, the tendency to make stable and global attributions increases the likelihood of developing hopelessness symptoms, and the tendency to make global attributions (vs. specific ones) increases the severity of symptoms. Low self-esteem results from internal, stable, and global attributions, not just internal attributions, as in the helplessness theory.

This brief review does not do justice to the complexity of any of these theoretical statements. For example, all the theories propose that the na-ture of the depressed person's symptoms depend on the nature of the un-derlying characteristics (cf. Beck, 1983). However, the present discussion focuses only on the etiological portions of the theories.

## EVIDENCE AGAINST THE COGNITIVE THEORIES

Three types of evidence appear to refute the theories. First, longitu-dinal studies (except one, Dobson & Shaw, 1986) following depressives over the course of their illness show that, as depressive symptoms remit, under-lying dysfunctional beliefs and attributions "remit" as well (Dobson & Shaw, 1987; Dohr, Rush, & Bernstein, 1989; Eaves & Rush, 1984; Hamilton & Abramson, 1983; Hammen, Miklowitz, & Dyck, 1986; Klein, Harding, Taylor, & Dickstein, 1988; Persons & Rao, 1985; Rea, Carpiniello, Secchiaroli, & Blanco, 1985; Seligman et al., 1988; Silverman, Silverman, & Eardley, 1984a, 1984b; Simons, Garfield, & Murphy, 1984). The finding that dysfunctional attitudes and attributions wax and wane with the clinical state appears to refute the hypothesis that they are stable traits.

Second, comparisons of normal and recovered depressives ( except one, Eaves & Rush, 1984) find that these groups do not differ in dysfunctional attitudes or attributions (Blackburn & Smyth, 1985; Dobson & Shaw 1986; Dohr, Rush, & Bernstein, 1989; Fennell & Campbell, 1984; Hamilton & Abramson, 1983; Hollon, Kendall, & Lumry, 1986; Reda et al., 1985; Silverman et al., 1984a; 1984b; Wilkinson & Blackburn, 1981).3 The finding that remitted depressives do not differ from normals contradicts the theo-ries because unless patients received therapy designed specifically to produce changes in dysfunctional beliefs or attributions the stable cognitive vulnerability factors would be expected to be present in recently recovered depressives

Third prospective longitudinal studies evaluating whether underlying cogmt10ns predispose md1v1duals to later depressive episodes have produced mixed results. Two important studies report negative findings ( others with positive findings are discussed below). Dysfunctional beliefs and attributions did not predict to onset of depression during a 1-year study of a large community sample (Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Similarly, depressive self-schemas did not predict to subsequent depressive symptomatology in a 4-month followup of college students (Hammen, Marks, deMayo, & Mayol, 1985a, Study 1). Thus, these prospective studies did not find that stable cognitions are vulnerability factors that predict to subsequent depression. This evidence contradicts the theories because sta-ble underlying cognitions in asymptomatic populations should predict those who will develop subsequent depressions.

In response to this large body of negative evidence, many investigators have concluded that dysfunctional attitudes and attributions are not vul-nerability factors, but instead are consequences or correlates of depression, or even simply part of the depressive syndrome itself (Barnett & Gotlib, 1988; Coyne & Gotlib, 1983; Dohr et al., 1989; Hammen et al., 1985a; Hammen et al., 1986; Hollon et al., 1986; Lewinsohn et al., 1981). These writers suggest that the cognitive theories of depression accurately charac-terize the nature of thinking during depressive episodes, but do not describe the vulnerability factors that determine which individuals become depressed in the face of stressful life events. Using the terms proposed by Brewin (1985), these investigators accept the "symptom" model of the relationship between cognition and symptoms but not the "vulnerability" model.

## THE MOOD-STATE HYPOTHESIS

We propose an alternative account of the negative evidence reviewed above-an account that is consistent with the etiological portions of the cognitive theories. We propose that dysfunctional attitudes and attributions are, as stated by the theories, stable personality traits, but that an individual's ability to report them depends on current mood state. In particular, the more negative the mood, the more likely the vulnerable individual will be able to report negative cognition. We call this the mood-state hypothesis. The mood-state hypothesis is not really an original idea. We propose that dysfunctional beliefs and attributions are encoded in memory via mechanisms like those outlined in Bower's (1981) associative network

model of mood and memory. In this model, a person's ideas, beliefs, memories of events, inferences, actions, and views about the self, world, and future, are encoded in an interconnected network that also includes nodes for affective states, such as sadness, guilt, anxiety, and so on. Facts or beliefs, such as "I am nothing if a person I love doesn't love me," are rep-resented by associations connecting the concepts of self, person I love, absence of life, and worthlessness. The "spreading activation" part of the model proposes that when one node, idea, concept, cognition, or mood, for example, is activated, other nodes that are directly or indirectly linked by association with the activated one are activated as well.

According to Bower's (1981) model, an individual may hold a belief but not be aware of it unless it is activated in some way (it is unconscious). If the belief is tightly associated with a given mood state, the individual may not be able to report the belief unless the mood state to which it is linked has been activated. For example, a negative mood state might prime the nodes corresponding to the statement "I am nothing if a person I love doesn't love me" because these nodes are associatively linked to the nega-tive mood state. Thus, Bower's model can explain why individuals who hold dysfunctional beliefs or a negative attributional style might be unable to report these cognition unless they are experiencing a negative mood state.

The associative links of Bower's (1981) model might be the result of a mood-state-dependent effect, a mood-state-congruent effect, or both (Blaney, 1986). In a mood-state-dependent effect, the individual learned the pathogenic cognitions when in a negative mood state, so the reinstate-ment of a negative mood activates the cognition. In a mood-state-congruent effect, the cognitions are activated by the negative mood state because of the congruence between the negative valence of the mood and the negative valence of the cognition.

Although we are proposing the subjects' ability to report underlying dysfunctional beliefs depends on mood-state effects, Riskind (1989) has offered a somewhat different viewpoint. He proposed that what appear to be effects of mood induction are really cognitive priming effects, and he suggested that mood induction procedures really serve as cognitive priming procedures. This idea is quite consistent with the observation that the Velten (1968) mood induction, used in our empirical studies and those of many others, actually involves a cognitive priming procedure: Subjects are asked to repeat and concentrate carefully on a list of self-statements. Riskind's model would appear to produce the same results as the model we propose, but of course the proposed mechanisms producing the results differ. Empirical work to examine the precise mechanisms would be of considerable interest. One way of doing this might be to examine effects of mood inductions varying in "cognitive content" on reporting of dysfunc-tional beliefs.

According to the etiological theories of depression, only certain indi-viduals, those with certain learning histories, hold the predisposing depres-sogenic cognitions. A nonvulnerable individual, one who does not hold the dysfunctional belief or attributional style, is not expected to report the dysfunctional cognition when in a negative mood state. For those nonvulner-able individuals, the dysfunctional beliefs were never learned and, therefore, are not present in the associative network.

The model presented here is not original. In fact, it is simply a re-statement of the cognitive theories of depression. The theories were pro-posed as diathesis-stress models: That is, underlying cognitions were not postulated to be problematic unless activated by some stress. The cognitive theories generally describe activation as resulting from life event stresses. In the model proposed here, we suggest that a negative mood state might serve this activating role. However, even the activating role of negative mood state has been offered previously by others, including Ingram (1984), Riskind and Rholes (1984), Segal 1988), Segal and Shaw (1986), and Teasdale (1988). What may be new here is the idea that unless the underlying beliefs have been activated, they are not observable, measurable, or report-able even though they are present in a "latent" or unconscious form.

## Accounting for Evidence Against the Cognitive Theories

The mood-state hypothesis accounts for the three types of evidence against the etiological theories described above. First, according to the mood-state hypothesis, underlying beliefs "remit" as depressive symptoms remit because, as patients' mood states improve over the course of their illness, their underlying beliefs are deactivated and become inaccessible and unavailable. We propose that the dysfunctional attitudes and negative attributional style remain, but in a latent, inactive form. Second, according to the mood-state hypothesis, recovered depressives do not report elevated scores on measures of underlying beliefs because they are not experiencing a negative mood state; therefore, the underlying cognitions are deactivated and inaccessible (but still present). Finally, underlying vulnerabilities should not predict to subsequent depression unless the vulnerabilities are assessed during negative mood states. Thus, dysfunctional thinking of asymptomatic patients who are in a good mood should not predict to subsequent depres-sion.

The mood-state hypothesis can also explain why a few studies have reported positive results that are consistent with the theories, in contrast with the many studies with negative results reviewed above. For example, Eaves and Rush (1984), unlike all other investigators, reported that recov-ered depressed patients (as compared to normals) exhibited a negative attributional style. The mood-state hypothesis would explain the Eaves and Rush finding by pointing out that their remitted patients were still some-what depressed, and more depressed than remitted patients in other studies, an observation also made by others (Hollon et al., 1986; Silverman et al., 1984b). Because the remitted patients were somewhat depressed, they had the ability to access and report dysfunctional attributions that were inaccessible to remitted depressives in others studies.

Unlike the two studies cited earlier (Lewinsohn et al., 1985; Hammen, Marks, deMayo, & Mayol, 1985), two prospective longitudinal studies (Hammen, Ellicott, Gitlin, & Jamison, 1989; Hammen, Marks, Mayol, & deMayo, 1985b) found evidence that dysfunctional beliefs measured in asymptomatic individuals *are* related to subsequent depression. In both studies, Hammen and colleagues found that individuals with specific cognitive vulnerabilities (dependent vs. autonomous) reported subsequent depressive symptoms that were related to specific (interpersonal vs. achievement) life events.

Hammen et al. (1985b) classified subjects as dependent or autonomous by asking subjects to recall past episodes of negative mood. Subjects who recalled failure events were classed as autonomous, and those who recalled rejections or losses were classed as dependent. This task did not require the subjects to endorse current dysfunctional beliefs, but rather to remember incidents from their past that were associated with a particular mood state. We speculate that this task allowed subjects to accurately report latent dysfunctional beliefs because of its focus on negative mood. Because negative mood serves as an explicit recall cue, subjects were able to retrieve the information needed to assess their underlying vulnerabilities. Of course, this hypothesis is speculative; however, an empirical study to test this hypothesis would be of considerable interest.

In a later study, Hammen et al. (1989) studied chronic depressives, individuals who are likely to be in a negative mood state during any assessment. As a result of their negative mood, these subjects were likely to have access to their dysfunctional beliefs. Thus, we argue that the mood-state hypothesis accounts for many findings that appear to contradict the cognitive theories of depression, and it may also explain the positive results obtained in a few studies that support the theory.

## EVIDENCE SUPPORTING THE MOOD-STATE HYPOTHESIS

Three studies provide direct evidence supporting the mood-state hypothesis (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Teas-dale & Dent, 1987).

Miranda & Persons (1988) asked 43 nondepressed women to rate their mood and complete a measure of dysfunctional attitudes (the Dysfunctional Attitude Scale or DAS; Weissman, 1979; Weissman & Beck, 1978) before and after either a positive or negative mood induction. A variation of Velten's (1968) procedure was used. As predicted, the mood induction produced reliable changes in mood and in dysfunctional atti-tudes.5

The study also tested the hypothesis that individuals with a past his-tory of depression would report more dysfunctional attitudes than those without a history of depression, but that high scores on dysfunctional beliefs in vulnerable individuals would only appear for subjects who were in a negative mood state. This prediction was confirmed. Miranda, Persons, and Byers (1990) reported results of two experiments. In the first, depressed psychiatric patients (both male and female) showed changes in dysfunctional attitudes as a function of spontaneous di-urnal fluctuations in mood. This result extended the previous demonstra-tion of the mood-state-dependent effect to men and to clinically depressed subjects, and showed that the effect occurred when mood fluctuations were spontaneous rather than induced.

In the second experiment, nondepressed individuals who had a history of a previous depressive episode showed elevated scores on a measure of dysfunctional attitudes if they were in a negative mood state at the time they were assessed. Nonvulnerable individuals did not show elevated dysfunctional attitudes, even when they were in a negative mood state. This finding is depicted in Fig. 1.

Teasdale and Dent (1987) also assessed cognitive functioning in never depressed and previously depressed individuals (women) following a negative mood induction. They used a measure developed by Kuiper and his colleagues to assess depressive schema and they used sad music to induce a negative mood. Following the mood induction, subjects read negative and positive trait adjectives and indicated which described themselves; then a surprise free-recall task was given. Although previously depressed and never depressed subjects did not show the predicted differences in number of negative adjectives rated as applicable to the self (perhaps due to a floor effect; few negative adjectives were rated as applicable to the self by sub-jects in either group), the groups did show the predicted difference in recall of negative adjectives previously rated as applicable to the self. Twenty-eight percent of previously depressed subjects recalled one or more nega-tive self-rated adjectives, whereas only 5% of the never depressed group did.

Thus, three studies provide direct evidence supporting the mood-state hypothesis. Although these results are encouraging, they have (at least) three important limitations. First, sample sizes are small. Second, these findings do not provide direct evidence that the dysfunctional cognitions are stable traits. The data are correlational and do not contradict the view that the dysfunctional cognition are simply concomitants or consequences of depression for vulnerable individuals. We simply argue that these data support the mood-state hypothesis, which provides an alternative explana-tion for data widely viewed as contradicting the cognitive theories.

Third, subjects vulnerable to depression were obtained by selecting individuals who reported a previous depressive episode. However, evidence that these individuals are cognitively different from controls does not pro-vide compelling evidence for the causal hypothesis outlined in the cognitive theories, because this evidence is also consistent with the "scar" hypothesis that is, the hypothesis that the dysfunctional attitudes are the result, not the cause, of depression. To demonstrate that the dysfunctional atti-tudes and negative attributions are stable traits and play an active, causal role, repeated assessments of subjects who later become depressed are required.

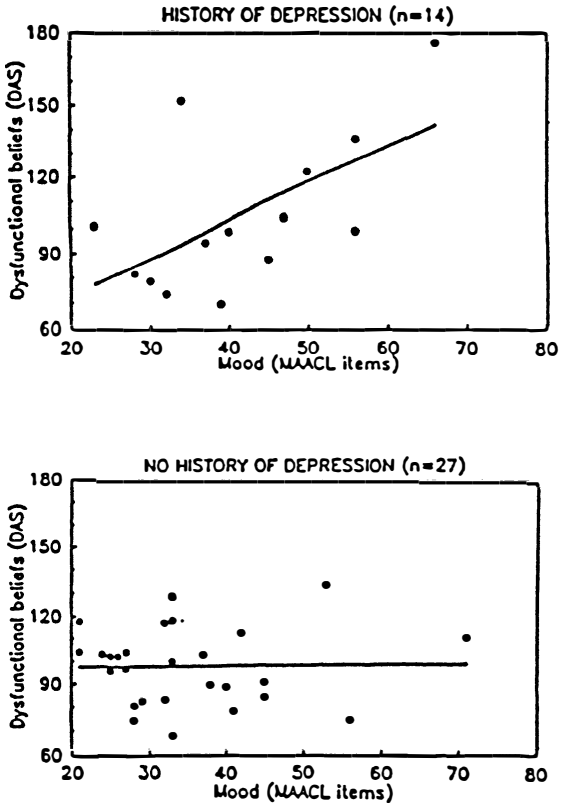


**Fig. 1.** Scatter plots and estimated regression lines for the relation of dysfunctional beliefs (measured on the Dysfunctional Attitude Scale; DAS) to current mood state (measured with items on the Multiple Affect Adjective Check List; MAACL) in subjects who reported a history of depression and those who had never been depressed.

Thus, although the three studies described here provide evidence sup-porting the mood-state hypothesis, they have important limitations and much more evidence is needed. However, if the mood-state hypothesis is correct, it has important implications for theory and research in several areas.

## IMPLICATIONS OF THE MOOD-STATE HYPOTHESIS

The mood-state hypothesis has implications for the cognitive theories of depression that have been the focus of our discussion. In addition, we believe it has implications for other theories of depression, including biological theories, for studies of psychotherapy process, for studies of other psychopathologies, for epidemiological studies of depression, for attempts to prevent and treat depression, and for theories of cognition.

### *Implications for Cognitive Theories of Depression*

Many investigations have interpreted evidence showing that underly-ing dysfunctional beliefs and attributions fluctuate with clinical status as disconfirming the etiological portions of the cognitive theories of depres-sion. However, the mood-state hypothesis accounts for this evidence in a way that is consistent with the theories. Therefore, the widespread senti-ment to view the underlying beliefs and attributions as concomitants of depression, rather than stable vulnerability factors, may be premature.

The mood-state hypothesis dictates changes in the design of studies testing the cognitive theories of depression. The mood-state hypothesis indicates that any attempt to assess cognitive vulnerability factors in non-depressed individuals must involve some procedure to activate the vulnerability. We recommend adding an activation procedure immediately before assessing dysfunctional attitudes, schema, or negative attributions in nondepressed experimental subjects, whether subjects are college students, randomly selected individuals from the community, previously depressed individuals, or individuals deemed vulnerable to depression by virtue of early, current, or upcoming life experiences. An activation procedure is nec-essary in cross-sectional studies ( e.g., comparisons of never depressed and

previously depressed individuals) and in longitudinalstudies (e.g., prospec-tive studies of nonpatient samples or repeated assessment of depressed individuals, including posttreatment assessment of cognitions in an attempt to predict relapse). Unless an activation procedure is included in these types of studies, the failure to demonstrate the presence of the underlying beliefs or attributions may be due to the investigator's failure to activate a cognitive vulnerability that is present but latent and therefore not re-ported.

A variety of procedures might be used to activate the cognitive underpinnings of depression. We have used the Velten (1968) mood induction procedure; other investigators have used musical mood inductions, scenes from films imagined stressful life events, or hypnosis. Subjects might also be assessed at times of naturally occurring stress likely to activate their vulnerability.

Matching of the subject's cognitive vulnerability and the activation procedure is likely to be important. Thus, for example, Beck's theory (and other theories) propose that two types of vulnerability underlie clinical de-pression: some individuals (dependent types) feel worthless unless they are loved and become depressed when they experience an interpersonal loss or rejection. Others (autonomous or achievement types) feel worthless unless they are independent and successful and become depressed when they experience a failure event. Thus, an induction procedure in which subjects imagine an interpersonal rejection is likely to prime the cognitive under-pinnings of the dependent individual but not the autonomous one; analogously, imagining a failure event is likely to prime the cognitive vulnerability of the autonomous individual but not the dependent one.

### *Implications for Other Theories of Depression*

The central point of this article, namely, that accurate assessment of underlying cognitive structures requires a procedure designed to activate those structures, has implications for other theories of depression as well. Psychodynamic theories allocate an etiological role to stable underlying personality structures that is quite similar to that described by the cognitive theories (Arieti & Bemporad, 1978; Blatt, 1974; Chadoff, 1970; Hirschfeld, Klerman, Chodoff, Korchin, & Barrett, 1976; Millon & Kotik, 1985). The mood-state hypothesis suggests that accurate assessment of these cognitive structures is likely to require an activation procedure as well.

Investigators studying biological underpinnings of depression (Depue & Monroe, 1983) have suggested that the failure to find biological markers for depression may be due to problems similar to those encountered by cognitive investigators searching for cognitive markers. They have suggested that some sort of activation or priming procedure may be needed before assessing the hypothesized underlying vulnerability.

Some investigators have already begun to do this. For example, before assessing thyroid stimulating hormone (TSH), researchers administer a dose of thyrotropin-releasing hormone (TRH), a hormone found in the hy-pothalamus that causes secretion of TSH from the pituitary, which then acts on the thyroid to stimulate release of thyroid hormone (Rosse, Owen, & Horihisa, 1987). Investigators evaluate TSH only after administering a dose of TRH, to ensure that the system has been "activated." We speculate that joint activation of both the biological and psychological systems might yield a more accurate assessment of both the underlying biological and psy-chological process. Both systems could be stimulated at once by giving a negative mood induction and administering TRH, for example, before assessing dysfunctional attitudes and TSH.

### *Implications for Studies of the Psychotherapy Process*

Attempts to show that different therapy specific modes of action different have met with dissapointing and nearly uniform failure. For example, recent studies of cognitive therapy have found that comparable changes in dysfunctional thinking occur during treatment of depression regardless of the intervention (Rehm, Kaslow, & Rabin, 1987; Rush, Beck, Kovacs, Weissenburger, & Hollon, 1982; Simons et al., 1984; Zeiss, Lewinsohn, & Munoz, 1979). This finding contradicts the proposed mechanism of action of cognitive therapy, which predicts that only patients treated with cognitive therapy will experience changes in under-lying cognition. The mood-state hypothesis may account for this negative result.

The mood-state hypothesis proposes that apparent cognitive changes in patients who were not treated with cognitive therapy are a result of improved mood state. Thus, patients treated with antidepressant medication appear to show changes in dysfunctional thinking during treatment. However, the mood-state hypothesis proposes that these patients still retain their dysfunctional attitudes; these attitudes are simply not reported because patients are in a positive mood state at the time their attitudes re assessed. The dysfunctional attitudes are present, but they are latent, m-active, and unavailable for report.

To test this hypothesis, patients treated with antidepressant medica-tion and cognitive therapy can be given a negative mood induction before they report their dysfunctional attitudes. We predict that this pro dure would show that patients treated with cognitive therapy have expenenced a true remission of dysfunctional attitudes but that patients treated with antidepressant medication have not. We propose that addition of a negative mood induction to the post-treatment assessment of patients receiving different treatments will allow investigators to observe effects resulting from differences between treatments. The mood induction ( or other activation procedure) "turns on the light" that allows the investigator to observe real differences between groups that are otherwise hidden from view.

### *Implications for Other Psychopathologies*

It seems unlikely to us that the mood-state hypothesis is specific to depression and depressed mood. Investigators studying anxiety have long been aware of the fact that anxiety-relevant cognition in phobics depends on the presence of fear-inducing stimuli (Lang, 1977). For example, a patient may tell a therapist that he is afraid to cross a bridge, but be unable to retrieve the thoughts that fuel his fears while sitting peacefully in the therapist's office. The patient has access to the fear cognitions ( e.g., "I will faint before I reach the other side," "I will drive over the edge and drown") only when he is frightened, and this fear occurs only when the patient is near the bridge, either in vivo or imagination. Cognitive models of anxiety (e.g., Foa & Kozak, 1986) already make use of Bower's associative network. model and emphasize the need to activate the fear before assessing and treating it. Investigators studying other phenomena, including relapse from alcohol and other addictions (Marlatt, 1985), multiple personality disorder (Bower, 1986), bulimia, sexual dysfunction, anger, impulsivity, and guilt, among others, have also found the mood-state hypothesis helpful.

### *Implications for Epidemiological Studies of Depression*

The mood-state hypothesis suggests that epidemiological studies of depression based on interviews with nondepressed subjects may underestimate lifetime prevalence of depression. The mood-state hypothesis suggests that nondepressed subjects fail to report previous episodes of depression because their positive mood prevents them from recalling those earlier epi-sodes. This hypothesis is supported by many studies showing that recall of negative memories is mood-state-dependent (e.g., Clark & Teasdale, 1982; reviews by Blaney, 1986; Bower, 1981, 1987).

We suggest that an interview probing for past depressive episodes is likely to elicit a more accurate report of depressive symptomatology if the interview is preceded by some type of induction procedure. For example, memory for past negative events could be primed by asking subjects to recall times in their past when they felt sad, lonely, or inadequate. The same argument applies to lifetime prevalence studies of other disorders, most obviously the anxiety disorders.

### *Implications for the Prevention of Depression*

The notion that latent cognitive structures may require activation before they are assessed addresses an important problem encountered by de-pression prevention researchers (Mufioz, Glish, Soo-Hoo, & Robertson, 1982). Investigators have had difficulty identifying persons vulnerable to future depressive episodes-targeting individuals that need a prevention intervention. The use of a mood-state or other type of induction procedure might be useful here. If a negative mood state is induced first, persons vulnerable to depression will be more likely to be able to report pathogenic cognitions or other vulnerability factors that may otherwise be hidden from view.

The activation idea may also be helpful to clinicians working to prevent depressive episodes. Cognitive and behavioral prevention interventions may be more useful if pathogenic cognitive structures are activated before the interventions are taught, for (at least) two reasons. First, if these structures are not activated, subjects may not be motivated to work to correct their thinking because they may not be aware of any distortions or any negative consequences ( e.g., depressed mood) of the distortions. Activation procedures may expose these distortions. Second, use of an activation strategy may enhance generalization of learning. Vulnerable individuals will need to utilize prevention interventions when they notice their mood is deteriorating, and they may be more likely to recall and use these strategies in this negative mood state if they have learned the interventions in a nega-tive mood state.

### *Implications for the Treatment of Depression*

The mood-state hypothesis implies that effective treatment of the un-derlying dysfunctional attitudes and attributional styles requires activation of these cognitive structures. This model is analogous to the cognitive model of anxiety proposed by Foa and Kozak (1986) that emphasizes the need to activate the cognitive fear structure before it can be effectively treated. Activation is necessary both to assess the nature of the problem and to produce some change. A detailed account of the implications of the mood-state hypothesis for cognitive therapy of depression is provided elsewhere (Persons & Miranda, 1991).

### *Implications for Theories of Cognition*

The mood-state hypothesis may also account for difficulties encountered in verifying Bower's associative network model of mood and memory. In his 1987 review, Bower acknowledges that difficulties have arisen in attempts to replicate the finding that recall of material learned in the laboratory following an induced mood depends on the

presence of that mood (see also the review by Blaney, 1986). Bernard Baars (personal communi-cation, June 1989) has made the intriguing suggestion that the finding that mood-state effects occurred only in subjects with past episodes of depression but not in never depressed individuals might account for the failures to replicate. That is, Bower's theory about the effects of induced negative mood on learning and recall might be true only for subjects who are vul-nerable to depression.

In sum, we propose that the mood-state hypothesis can account for evidence generally viewed as contradicting the etiological theories of depression. We propose that the hypothesis has important implications for future tests of those theories as well as for other theories of depression, including biological theories, for studies of the psychotherapy process, for studies of other types of psychopathology, for epidemiological studies of depression, for the prevention and treatment of depression, and for theories of cognition.

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