Reading #12

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A Cognitive–Expectancy Theory of Therapy for Helplessness and Depression

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INTRODUCTION

Treatment research is typically atheoretical and pragmatic. To the practicing clinician, it is often more important to know whether something works than to know how it works. Drawing on Bandura's (1977a) distinction between mechanism and manipulation, interest in the *processes* that underlie change often develops in the aftermath of the serendipitous discovery of those *procedures* that produce change. Witness the order of development of Freud's dynamic theory, which followed the observed efficacy of Breuer's "talking cure," or the articulation of amine theory in depression (cf. Schildkraut, 1965), which followed the accidental discovery of the antidepressant medications.

Basic research, on the other hand, has typically focused on those theoretical mechanisms involved in the etiology of a disorder—an emphasis on process rather than procedure and on induction rather than remediation. Helplessness theory (Seligman, 1975) and the reformulated helplessness theory (Abramson, Seligman, & Teasdale, 1978; Abramson, Garber, & Seligman, Chapter 1 of this volume) are essentially process models of etiology and, as such, can provide guides to the development of process models of

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remediation. Although they ought not be contradicted by the results of outcome research, the theories themselves do not speak directly to the processes or the procedures involved in therapy.

In this chapter, we present a cognitive–expectancy theory of the process of change in clinical depression. We argue that a strategy that focuses on identifying and explicitly testing those negative expectations typically held by depressed individuals provides the most powerful, most expedient mode of clinical intervention.

In a recent article, Akiskal and McKinney (1975) suggested that there exists a biochemical "final common pathway" in depression. Any of several processes (e.g., environmental, cognitive, behavioral, biological) were seen as sufficient to initiate a causal chain of events leading to a common biological process. This biological process produces the full syndrome of clinical depression which, once triggered, takes on a life of its own.

We are postulating a cognitive "final common pathway," one in which negative expectations for the near and distant future play the central role. Although any of several different processes may serve to produce negative expectations—such as perceptions of lack of control, negative views of the self, deficits in obtained reinforcements, or disturbances in neurotransmitter balances in the limbic system—those negative expectations become the mediators of the full syndrome of depression. Although any of several different sets of etiological factors are seen as *sufficient* to produce negative expectations, those negative expectations are seen as *necessary* antecedents of the full depressive syndrome.

Helplessness (Seligman, 1975) and reformulated helplessness (Abramson et al., 1978, and Chapter 1 of this volume), as essentially etiological models, may detail the ways in which some people become depressed. In our development of a cognitive-expectancy theory of change, we focus on an exposition of a *process* model of change, one which is consistent with, although neither directly deducible from nor ultimately dependent on, the validity of either the original or the reformulated helplessness models. This cognitive–expectancy model is also compatible with Beck's larger cognitive model (Beck, 1967, 1976). It represents not so much a departure from as an elaboration on Beck's theory by specifying what we believe to be the most efficient sequences of procedures for effecting rapid and long-lasting change in depression. In fact, our formulation has been guided by our experience with Beck's cognitive therapy (Beck, 1964, 1967, 1970). We have sought to explain the processes of change manipulated by those procedures that comprise the cognitive–behavioral interventions.

The theoretical significance of the model lies in its attempt to specify the nature and sequence of those processes involved in the reduction of syndrome depression. Processes central to the remediation of a disorder need not be isomorphic with those processes central to its etiology. Many causal events either alter the nature of the organism in some permanent way or follow a definite temporal sequence (Hollon & Beck, 1979). We will argue that both types of processes may occur in depression and that the most effective interventions are those that teach compensatory cognitive–behavioral strategies. These strategies focus on expectations (an end process in the reformulated helplessness theory) rather than on attributions, even though the latter may have indeed preceded the expectational aberrations in the initial causal chain leading to the episode of depression.

The practical significance of the cognitive–expectancy theory lies in its potential to guide the application of existing therapeutic procedures (e.g., see Beck, Rush, Shaw, & Emery, 1979), and to encourage the development of new, sharply focused intervention techniques. In this chapter, we first present the cognitive-expectancy theory and examine its relationship to existing theories of etiology. We then evaluate the adequacy of the model vis-a-vis existing empirical outcome data. Finally, we conclude with a discussion of potential procedural innovations and practical implications that follow from such a model.

A COGNITIVE-EXPECTANCY THEORY

Cognitions and cognitive processes play a central role in the various cognitively oriented etiological theories of depression (cf. Beck, 1963; Seligman, 1975). Cognitive approaches to the treatment of depression (cf. Beck, 1964, 1967, 1970; Seligman, Note 1) have in common an emphasis on changing the depressed individual's beliefs and ways of processing information and suggest a variety of techniques for altering depressive symptomatology. Although these approaches agree on the general processes involved, they differ somewhat in terms of the specific focus and procedures utilized. The major issue for cognitive–behavioral interventions concerns specifying what procedures targeted at what processes provide the maximum change with respect to the magnitude, stability, and generality of change in depressive symptoms.

An overview of a cognitive–expectancy model is presented in Figure 7.1. The basic model, adapted from Hollon and Kendall (1979), presents a functional analysis of the interrelationships between antecedent stimulus events (Sd's), organismic variables including cognitions (Cog 1) and affective and autonomic reactions (Aff/Auto 1), voluntary motoric behaviors (R's), and consequent environmental events (S*'s). As in all cognitive theories, cognitions are seen as: *(a)* mediating behavioral responses to events; and *(b)* directly determining the nature and intensity of affective reactions (Mahoney,

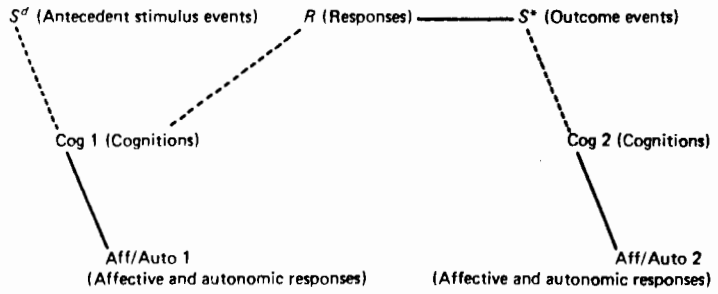

Figure 7.1. This is a graphic example of cognitive–behavioral functional analysis.

1977). Both classical and operant learning paradigms can be incorporated within this S-O-R framework, with the vertical dimension representing the classical paradigm and the horizontal dimension representing the operant paradigm. In both instances cognitions are viewed as playing a central mediational role (see Bandura, 1977*b*; Bolles, 1972; Grings, 1973; or Mahoney, 1977, for more detailed critiques of peripheralistic, as opposed to centrally mediated, learning theories). We have added the additional component of cognitive evaluations (Cog 2), with the attendant affective and autonomic components (Aff/Auto 2) following those responses (R's) that produce the consequent outcome events (S*'s).

Specific classes of cognitions are more likely to occur at different times. For example, in Figure 7.1, expectations will occur at Cog 1 and play a larger role in the maintenance and/or treatment of depression, whereas attributions of causality occur at Cog 2 and are more cental to the etiology of the disorder. Figure 7.2 presents a partitioning of what we regard as the major classes of cognitions central to depressive processes: perceptions, attributions, beliefs, values, and expectations. We assert that attributions of causality typically refer to events that have already occurred; expectations

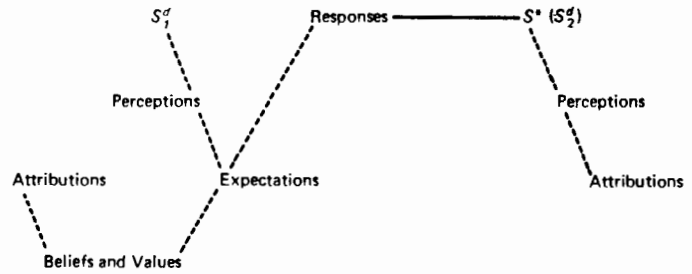

Figure 7.2. This diagram presents the organismic components of the S-O-R model.

refer to the subjective probabilities held regarding events in the near and distant future. Attributions may or may not contribute to the formulation of expectations, but it is the expectation which most directly produces the concurrent affect and subsequent behavior. As we shall describe in the sections to follow, expectations are seen as the *end point* of a cognitive causal chain. Although attributions may well play a major part in the initiation of an episode (and, perhaps, subsequent expectations), it is the expectation which is seen as most directly impacting key affective and behavioral processes.

A Taxonomy of Cognitive Processes

The distinction between expectations and attributions, based on each process's temporal relationship to external events, is crucial to our argument. It is this difference in temporality which, we argue, makes expectational processes the most *feasible* point of therapeutic intervention. In the sections to follow, we expand our discussion of the various classes of cognitions that comprise our taxonomy.

Perceptions

The first cognitive feature is the perception of the stimulus event (S^d). Perception includes identifying, discriminating, recognizing, and judging objects and events by means of sensory information. Perception, at some level, is, of course, essential for an event to have an impact on subsequent cognitions and behaviors. Furthermore, the recognition of contingencies between classes of events may be essential for learning. For example, people are not much affected by paired stimulation unless they recognize that the events are correlated (Brewer, 1974; Dawson & Furedy, 1976; Dulaney, 1968; Grings, 1973). When contingency awareness and conditioning are measured concurrently, predictive stimuli do not elicit anticipatory reactions until the point at which awareness is achieved. Alternatively, even in the absence of a specific objective event, the subjective perception of its occurrence may influence subsequent responses.

Many of the cognitive distortions identified by Beck (1963, 1967) as occurring in depressives are distortions in perception. *Arbitrary inference*, the process of drawing a conclusion that is contrary to the objective evidence in a situation, represents one such example. Other examples include *selective abstraction*, the drawing of an inference based on only part of the information available in a situation; *magnification*, the tendency to exaggerate the importance of an event; *minimization*, the tendency to underestimate the importance of positive events; and *all-or-none* thinking, the tendency to perceive situations in absolute terms.

Attributions

The second cognitive component involves the attributions made for events. Attributions are simply the causes one assigns to an event or, less important for our purposes, those characteristics ascribed to an individual or object (Heider, 1958; Jones & Davis, 1965; Kelly, 1967). Attributions are by no means necessary or automatic processes in the perception of events. An individual may or may not make an attribution in a given situation (Wortman & Dintzer, 1978). When individuals do make attributions, however, the attribution made can differentially influence subsequent cognitions, affects, and behaviors.

Recently, there has been increasing interest in the role of attributional processes in the etiology of depression (Abramson et al., 1978; Klein, Fencil-Morse, & Seligman, 1976; Kuiper, 1978; Metalsky & Abramson, in press; Rizley, 1978; Seligman, Abramson, Semmel, & von Baeyer, 1979). In general, these investigations find that depressives are more likely to make internal attributions for negative events (i.e., failure) and external attributions for positive events (i.e., success) than are nondepressives.

Beliefs

The third component of the model involves the beliefs held by the individual. Fishbein and Ajzen (1972) define beliefs as the subjective probability that an object has a particular characteristic.[1] Beliefs do not exist in isolation but are often associated with other beliefs in an organized system (Kihlstrom & Nasby, in press). The centrality of a belief, that is, its role in the individual's belief system, can be defined in terms of its degree of connectedness with other beliefs (Rokeach, 1968). For example, beliefs about one's own characteristics and capcities, the *self-concept,* may play a particularly central role in the individual's belief system.

Values

The fourth major component, values, represent important life goals or standards of behavior (Oskamp, 1977). Values are evaluative and represent relatively arbitrary preference hierarchies, either chosen or acquired, by an individual.

Expectations

The fifth and, for our purposes, major component of this model is the expectation. With the growing emphasis on cognitive views of behavior, the concept of expectancy is assuming an increasingly prominent place in con-

[1]Of course, attributions and expectations can be defined as specific types of beliefs. Attributions can be defined as the subjective probability that an event had a specific cause. Expectations can be defined as the subjective probability that an event will occur.

temporary psychological throught (Bandura, 1977*a;* Bolles, 1972; Heneman & Schwab, 1972; Irwin, 1971). An expectancy is a propositional statement about the anticipated nature of events. It is a person's subjective estimate that a given event is likely to occur.

Bandura (1977*a)* suggested that there are two important types of expectancies: *(a)* an outcome expectancy, which is a person's estimate that a given behavior will lead to certain outcomes; and *(b)* an efficacy expectancy, which is the conviction that one can successfully execute the behavior required to produce the outcome. These two types of expectancies correspond to the distinction between universal and personal helplessness suggested by Abramson et al. (1978). In addition to expectancies about controllability—responses lead to outcomes—one can also have expectancies about predictability—stimulus events (CS) signal the occurrence of other stimulus events (UCS). The notion of controllability typically has implications for motivation and behavior, whereas predictability has implications for affect.

Cognitive representations of future outcomes generate current motivators of behavior. The expectation that responding in a certain way will result in anticipated benefits or avert future difficulties may help produce a particular behavior (Bolles, 1972). The expectation of reinforcement is seen as motivating the associated behavior. Thus, behavior is largely controlled by voluntary, decisional processes that are influenced by response–outcome (R-S*) expectancies, whereas affect tends to be more automatic and autonomic and may be more influenced by event–outcome (Sd-S*) expectancies.

The Acquisition of Cognitions

According to Bandura's (1977a) social learning analysis, expectations develop from four major sources of information: performance accomplishments, vicarious experiences, verbal persuasion, and physiological states. Cognitive events are induced and altered most readily by experiences of mastery arising from successful performances. The strength of one's self-efficacy expectancy determines the amount of effort and persistance in the behavior. A meaningful expectancy analysis, therefore, requires a detailed assessment of the magnitude, generality, and strength of the expectations, measured with the same precision used to assess changes in behavior.

Both beliefs and values are generally considered to be learned. A variety of factors can operate in the acquisition process. Direct personal experiences, parental influence, peer and reference group norms, and mass media influences may all play a role. There is some evidence that attributional styles that are stable over time vary across individuals (Seligman et al., 1979), although it is not clear how these tendencies are acquired. The processes behind individual differences in cognitive styles are particularly

poorly understood. For example, although Beck's cognitive model clearly describes distinct depressive styles in perception, no clear statement regarding the acquisition of these styles has been proposed.

It is likely that the different classes of cognitions influence one another reciprocally over time. The literature on perceptual defnese (Brown, 1961) suggests that values or hedonic relevance factors influence the nature of the perceptual process. Beliefs appear to be particularly stable over time, and it is likely that preexisting beliefs play a major role in both perceptual and attributional processes.

The discussion thus far has described the architecture of a cognitive system for depression. The various components of cognition are hypothetical constructs: an attribution represents the way things were; a belief represents the way things are; and an expectancy represents the way things will be. These processes are basically automatic habits of thought of which the individual is not always totally aware at the time, although if asked he or she may be able to produce them. None of these cognitions is necessarily a veridical reflection of reality, but instead represent the individual's perception of reality. Moreover, the certainty with which the individual maintains these beliefs determines the magnitude, stability, and strength of his or her subsequent responses, both behavioral and affective.

Thus, the current framework is a cognitively mediated model rather than a simple S-R model and is consistent with Bandura's (1977a) self-efficacy theory, Beck's (1967) cognitive schema, Bower's (1978) information processing theory, Ellis' (1962) A-B-C model, and Seligman's learned helplessness model (1975). The direction of causality among the various components is interactional rather than unidirectional, with each component having an effect on the others. Of course, any specific cognition can be influenced only by antecedent cognitions, and any given cognition can only influence subsequent cognitions. A careful recognition of the temporal dimension in interactive processes can help clarify the nature of the causal process.

In any given situation, depending on their particular cognitive structures, individuals may differ in the way they extract and combine information from the environment. Beck (1967) referred to this cognitive structure as a schema. A schema can be defined as a "complex pattern, inferred as having been imprinted in the organism's structure by experiences that combine with the properties of the presented stimulus object or of the presented idea to determine how the object or idea is to be perceived and conceptualized English & English, 1958, p. 258]." It is the mode by which the environment is screened, coded, evaluated, and organized into relevant psychological facets. Moreover, even in the absence of immediate environmental stimulation, schemata can channel thought processes. The schemata pattern the stream of associations and ruminations as well as the cognitive responses to external stimuli.

Among the various components of the cognitive schemata, beliefs tend to be the most stable over time. Beliefs play significant roles in the way information is processed. How we organize information tends to influence how we organize subsequent input. There is a kind of self-fulfilling prophesy or confirmation bias (Snyder, Tanke, & Berscheid, 1977) that affects the process of assimilation and accommodation of new information. The focus of attention, attributions, and memory for events are influenced by existing belief systems. In turn, attributions then serve to confirm or disconfirm the original beliefs. Similarly, expectancies about the future are influenced by both present beliefs and explanations of the past. It is the expectancies, however, which, mediated by motivation, most directly influence behavior and affect.

The schematic diagrams presented in Figures 7.1 and 7.2. represent the typical sequence of events, cognitions, and responses in the development of depression. When an event is perceived to occur, an attribution that both influences, and is influenced by, the existing belief system may be made. This leads to the formulation of an expectancy about future action and events that then determines the response (both motoric and affective). Following the response, a new event (outcome or S*) may occur, which again may be followed by an attribution, and so on. Thus, although some attributions precede expectations, any given attribution for an event follows that event, whereas expectations about that event preceded the event and, hence, the attribution.

Expectancies are not necessarily tied to attributions for previous events and may be influenced instead by a variety of other factors that intervene between an original attribution and a later expectancy. For example, new experiences or new information about the past event or future consequences can alter the expectancy that logically might have followed from the original attribution. This hypothesized time lapse between attributions at Cog 1 and expectancies at Cog 2 has important immplications for subsequent responses.

The end result of the sequence of cognitions are behavioral, emotional physiological, as well as additional cognitive, responses. With respect to depression, specifically, these include such symptoms as dysphoric mood, passivity, low self-esteem, pessimism, and guilt. These various symptoms may result from the various cognitive components. For example, self-esteem is hypothesized to depend on the attributions made for events, with internal attributions for negative events leading to low self-esteem (Abramson et al., 1978). It is the expectancy of not obtaining desirable outcomes, however, that produces such symptoms as behavioral passivity, lack of motivation, and pessimism. The magnitude and certainty of these negative expectations about near and distant events produce the dysphoric mood that defines the syndrome. It is hypothesized here that negative expectations about the future produce much of the depressive symptomatology, and therefore, the

cognitive-expectancy model of intervention suggests that the major focus of the process and procedure of therapy should be on expectations.

A COGNITIVE–EXPECTANCY THEORY OF INTERVENTION

The cognitive–expectancy theory is not intended to be a unique theoretical formulation of the etiology of depression. With regard to theory, the model overlaps with the general cognitive models of Bandura, Bower, and Ellis, and with the more specific models of depression by Beck and Seligman. Rather, the present model is an attempt to generate a theory of therapy for depression, one which is compatible with existing theories of etiology but which incorporates existing evidence regarding the efficacy of various intervention procedures into a coherent model of change. It provides a component analysis of cognitive and behavioral change processes in order to determine the most efficient and powerful procedures for producing change.

Because the cognitive–expectancy model is interactive, altering any one of the components of cognition should, in turn, alter the other components. The goal of any intervention is, of course, the eventual amelioration of undesirable symptomatology (i.e., dysphoria, lack of energy, anhedonia, hopelessness). The potency of intervening at various points in the model should be evaluated in terms of the magnitude, stability, and generality of change of these various symptoms. According to the S-O-R model, simply changing the environment (S_d-S^*) or the behaviors (R) is not a sufficiently powerful intervention, particularly with respect to stability over time, or generality across situations, of the symptoms.[2] This has been a consistent criticism of strict behavior modification programs based on peripheral S-R learning models (cf. Bandura, 1977*b*; Beck, 1970; Hollon & Kendall, 1979; Mahoney, 1977). Although these approaches may alter behavior in the laboratory, where the reinforcement contingencies are explicit and powerful, once the individual is away from the laboratory and the external reinforcers, the behavioral improvements disappear rapidly. As a result of these failures, clinicians and researchers alike have begun to examine the role of cognitions in behavior change. Cognitive theorists have stopped asking whether they should intervene at the level of cognition or behavior and have begun to ask which cognitive processes are most important. Should interventions be made at the level of perception, attribution, belief, value, or expectation?

[2]The term "simply" here refers to the restriction of intervention technique, not the ease with which those techniques can be accomplished. Important external reinforcers are frequently beyond the therapist's direct control, particularly when clients are adults seen in a noninstitutional setting—the modal depressed client.

We will argue that the most efficient point of intervention is that which stands at the end, rather than at the beginning, of the presumed causal chain. Specifically, those interventions that most directly impact on expectations, rather than perceptions, attributions, beliefs, or values, ought to produce the most rapid and the powerful change.

Onset of an Episode

Let us provide an example of this model in action. As shown in Figure 7.3, an individual competes for a job with several others (S_d), engages in various activities (e.g. submitting a resume, going to an interview (R's), and does not receive the position (S^*). The individual can then make any of several attributions for not getting the job. According to the reformulated helplessness model, if the individual makes a global, stable, internal *attribution* (e.g., "I am incompetent"), he or she is most likely to become depressed over the event. So far, we have described the *onset* of an episode of depression and have done so largely by recourse to an attributional mechanism. Also note that one component of the attribution, specifically, *stability*, involves a temporal component. It is likely that the characteristic (incompetence) seen as causal to the disappointing outcome will remain constant over time, remaining a factor in future situations. When an individual *thinks about* a stable characteristic interfering with obtaining desired outcomes in the *future*, that individual is generating an *expectation* about the future. An individual may well make an attribution to some stable characteristic without considering the implications of that stability. For example,

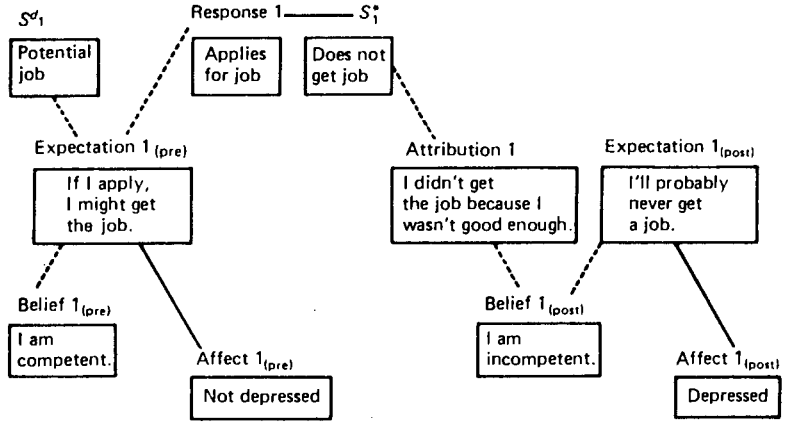

Figure 7.3. This diagram traces the onset of an episode of depression in terms of the model.

an individual can make an attribution to stable factors without necessarily generating an expectation (e.g., "I didn't get the job because I am incompetent"), but as soon as the individual considers the implications of that attribution (e.g., "Because I am incompetent, I will never get another job"), he or she is, by definition, generating an expectation. We will argue later that one essential difference between sadness (or normal grief) and depression (or melancholia) is the generation of negative expectations. The individual who does not get a job is sad, the individual who believes he or she will never get a job is depressed.

We have now generated an episode of depression in accordance with the reformulated helplessness model. The individual has experienced an undesirable outcome (it as easily could have been the loss of an important relationship) and has attributed that outcome to an internal, stable, global cause—his or her own incompetence. The individual's belief prior to this sequence, that he or she was generally competent, has been altered such that he or she now holds a belief in personal incompetence. The generality of the attributed causal factor leads to the expectation of nonsuccess across disparate situations, and the stability factor of the attribution makes it likely that the individual will generate expectations of subsequent nonsuccess in future endeavors. Given this constellation, the affect is one of profound dysphoria.

Maintenance of the Episode

We now move into the maintenance phase of the episode. As depicted in Figure 7.4, the already depressed individual enters the next situation convinced of his or her own incompetence. Upon encountering the stimulus of a second potential job opening, the belief in personal incompetence is likely to lead to the formulation of a negative expectation, namely that, even if he or she tries, he or she still will not get the job. Note that we are not dealing directly with an attribution for a past event at this point, we are dealing with an expectation generated from a belief generated by an earlier attribution. The relevant expectation concerns the *events to come*, and the relevant attribution concerns the *events that have occurred*. The relevant belief has served as the unit of cognition that has stayed stable over time from the first to the second situation; after being altered in the first situation from one of competence to one of incompetence, it has bridged the temporal gap between the prior attribution and the current expectation. Note that the prior attribution, in a sense, has led to the current expectation. Hence, prior attributions at times may cause subsequent expectations.

The individual, now expecting not to succeed even if he or she engages in the appropriate behaviors, does not apply for the job. The individual's

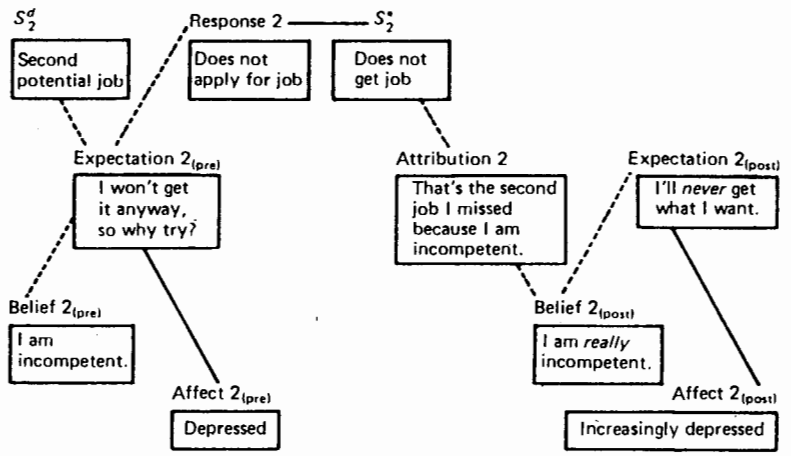

Figure 7.4. This diagram illustrates the next phase of the model—maintenance of an episode of depression.

nonactivity guarantees nonsuccess; one does not get jobs when one does not apply for them. Note that the individual's negative expectation has had two consequences: *(a)* he or she has not acted in a potentially adaptive fashion; and *(b)* he or she has experienced an increase in dysphoria even prior to the time for action and prior to the time when the actual event (obtaining or not obtaining the job) could possibly have occurred. These two consequences represent, respectively, the behavioral and affective consequences of negative expectations. The individual is passive, and, prior to the actual point of nongratification, the individual grieves for the loss. This capacity of humans to anticipate loss or disappointment and to respond affectively in advance of that event represents a major distinction between cognitive (or cognitive–behavioral) and strictly behavioral theories of depression.

Note also that we have not postulated any deficit or distortion in the individual's sytem of values. The individual need not have any desire to fail or desire to suffer; he or she need have no retroflected anger against the self to cathart nor any guilt to expiate. It is only necessary that the individual *expect* that he or she cannot do something that he or she values greatly. As hypothesized in the reformulated helplessness model, it may even be the case that the greater the value (importance) of the goal, the more severe the dysphoria over its perceived nonobtainability. This emphasis on disruptions in the expectational side of the expectancy–value theory of motivation (Heckhausen, 1967; Lewin, 1935) rather than on distortions in values or

desires represents a major distinction between cognitive (or cognitive–behavioral) and traditional dynamic theories of depression.[3]

There is a third, essentially cognitive, consequence resulting from this expectancy-mediated passivity; that is, the depressed individual, by not acting, confirms his or her own negative expectation. He or she does not get the desired job. The attribution, if one is made, is similar to that made for the precipitating event—specifically, "Again, I have failed to obtain a job *because* I am incompetent." The effect is to provide yet another piece of evidence to support the belief that one is incompetent. The individual does not have to *want* to believe in his or her own lack of ability; he or she may even *wish* that it were not so. But it would be a very dysfunctional organism, indeed, which could not perceive what seem to be the facts in the situations—two job possibilities and two instances of failure. The problem, of course, lies with the errors in thinking based on the inaccurate attributions, although from the individual's point of view the problem lies in a deficit in his or ability to get a job. The individual has engaged in an instance of *self-fulfilling prophecy;* he or she has acted in accordance with an inaccurate belief (by not trying for the job) in such a way as to generate evidence that seems to confirm that initial belief. The individual has not acted irrationally; rather, the individual has acted rationally given an inaccurate premise. We shall return to this point later.

Reversal of the Episode

At this point, the individual is depressed; he or she is without a job and is convinced of his or her own incompetence. This is the point in the sequence of events at which the individual is most likely to consult a professional therapist. What is the referral question? Typically, the individual approaches the therapist for help in reducing the symptoms of depression: the negative affect, sense of personal helplessness and/or hopelessness, behavioral lethargy, motivational difficulties, and vegetative symptoms that comprise the syndrome of depression (Beck, 1967). The individual typically leaves

[3]There is, of course, a wide range of dynamic theories of depression, several moving beyond Freud's classic retroflected anger model (Freud, 1917). Recent revisions by Jacobson (1971) and Arieti and Bemporad (1978), for example, rely heavily on ego mechanisms, which are, in part, related to expectational processes. Bibring's (1953) formulation of depression, although still retaining a place for unconscious, repressed motivational aberrations, emphasized the role of the conscious perception of hopelessness in the etiology of depression and served as an important point of departure for subsequent, nondynamic cognitive formulations. Even though we see little justification for retaining a role for unconscious motivational processes, we are struck by the increasing tendency of revisionist dynamic theorists to incorporate expectational mechanisms in their theories.

unchallenged the belief in his or her own inability; the therapist is asked to help the client feel better despite the perceived deficits or to provide skills to overcome those deficits rather than to explore the validity of this perception.

Figure 7.5 presents a schematic diagram that depicts the optimal point of intervention from the perspective of a cognitive-expectancy theory. Paralleling the maintenance phase of the episode in Figure 7.4, the client enters the situation convinced of his or her own incompetence. Presented with a third possible job opportunity, the client generates the following expectation: "I won't get the job anyway, so why try?". At this point, the therapist directly intervenes, remarking, "How can you be sure? Although you don't know for sure that you won't get the job, you do know that if you don't try, you can't get the job." The emphasis is on altering the patient's expectation, or at least on questioning the certainty with which the client holds that expectation. Assuming, as the reformulated helplessness model does, that the initial attribution to incompetence was incorrect, the client should have some greater than zero probability of success, *if the necessary behaviors are emitted.* Once the behaviors are emitted and once some one or more successful outcomes are experienced, the client begins to be confronted with

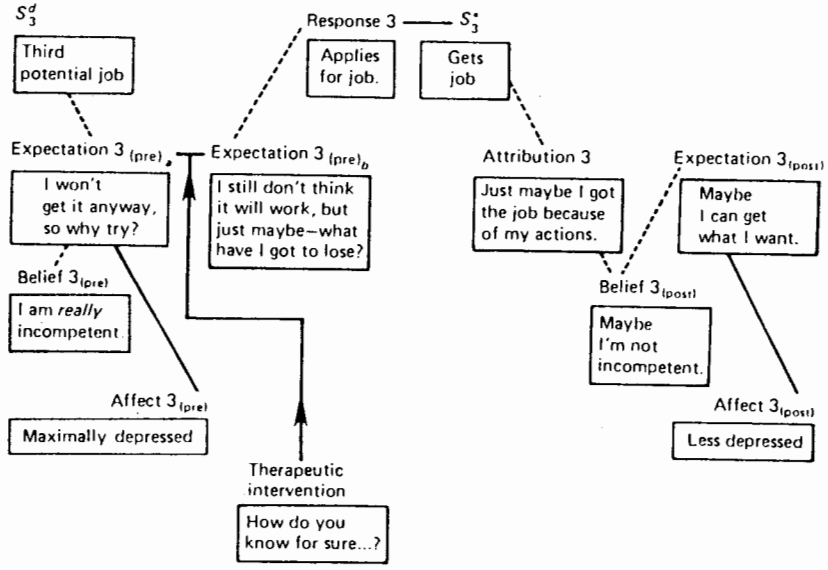

Figure 7.5. This graphic representation shows a reversal of an episode of depression at the optimal point of intervention.

evidence that is inconsistent with his or her belief in personal incompetence, the belief held when entering the situation. To the extent that those beliefs can be questioned and to the extent that subsequent expectations can be altered (e.g., "Maybe it's not totally hopeless"), the patient experiences a lessening of depression.[4]

Alternative Points of Intervention

Why not begin by attempting to alter the patient's initial attributions, if, according to Abramson et al. (1978), attributions are assumed to be central to the onset of the episode and to the subsequent alteration of self-concept and generation of negative expectations. We are not arguing that this should not be attempted; rather, we suggest that it is less likely to be effective. Discussions of previous events and the attributions made in their wake are necessarily post hoc; the therapist was not present to observe the sequence of events. Our experience has been that clients are typically skeptical of explanations that are more benign than those they have generated for an event. Although they would like to believe the therapist's rationale, most people share the common-sense notion that reexplanations of past events are, no matter how persuasive, essentially unconfirmable. Efforts directed at changing attributions essentially are focused on the past; by their very nature, attributions cannot be tested in any rigorous, prospective fashion *unless one specifically formulates expectations about events to come.* Just as for the scientist, for whom theory change proceeds most powerfully when hypotheses are confirmed or disconfirmed by actual observations, so too are individuals' beliefs most amenable to change on the basis of prospective disconfirmation rather than on the basis of simple reexplanation.

Bandura (1977a) has presented data that suggest that the use of enactive procedures provides the most powerful means of producing cognitive change. We would concur and point out that an initial emphasis on testing expectations forces the client and therapist to utilize enactive procedures in the course of therapy. The reexplanation of causality, the heart of reattribution

[4]We would not want to imply that all negative perceptions or beliefs are inaccurate. Undesirable events do occur, and unpleasant expectations may, at times, be borne out. Reality-based depressions probably do occur, although it is important to distinguish conceptually between probable and necessary precipitants of depression. Probable precipitants are events and/or situations that most individuals would become depressed in response to if they occurred. These types of events clearly exist. Necessary precipitants are those events that guarantee that a depression must follow if those events occur. It is not clear that any such conditions exist. Probable precipitants may well interact with cognitive and/or biologic factors to produce the resultant variability in susceptibility observed.

techniques (Beck et al., 1979), does not. Therein lies the central difference between an expectational and an attributional focus of intervention.

Evidence for the Theory

What evidence exists that speaks to this formulation? Actually, we are aware of little in the way of existing data that relates directly to our formulation. Bandura's work (Bandura, 1977a; Bandura, Adams, & Beyer, 1977) may be indirectly related. Focusing on change in approach behavior for snake phobics, Bandura and his colleagues compared modeling plus enactive contact, modeling alone, and a nonspecific control and found that the modeling plus enactive contact most powerfully altered both expectations and subsequent behavior. Furthermore, within each modality, changes in expectations proved to be a more powerful predictor of subsequent behavior than even previous behavior. This finding, although not relevant to the expectational versus attributional distinction, can be interpreted as supportive of the notion that an enactive exposure which tests expectations is a more powerful change technique than is the simple acquisition of information alone.

Is there any evidence that addresses the expectational versus attributional focus? Nisbett, Borgida, Crandall, and Reed (1976) have reported on attempts to alleviate mild depressions in ambulatory college students by means of reattribution therapy. The authors provided mildly depressed college students with normative information regarding weekly mood swings, namely, that some variability was normal and not an indication of psychopathology. The attributional manipulation had no observable effect on mood levels.

Dweck (1975) minimized the effects of failure in school children by manipulating the attributions made for those failures. However, in this instance, the intervention was at the same time as the occurrence of one precipitating event (a failure experience). We would not be at all surprised if an intervention that prevents an individual from making the initial misattribution served to *prevent* the onset of an episode of depression. Indeed, the potential capacity of the reformulated helplessness model to serve as a guide to the development of powerful *preventative* interventions may prove to be the greatest contribution made by the model. But, as we have described, those procedures that produce reversals in a phenomenon need not be isomorphic with those factors that produced it. In fact, a cognitive-expectancy model of intervention would argue that expectancy change procedures are preferred only when attempting to reverse dysphoria in an individual who is already depressed (the typical clinical situation), whereas an attributional focus would be preferred when attempting to forestall the onset of a depression. If our analysis of the causal chain of events is correct, then misattributions

should play a central causal role in the etiology of negative expectations, and attribution therapy should play a central role in prevention. Alternatively, negative expectations play a major role in the maintenance of the syndrome (including the maintenance of negative affect), and expectational interventions should play a central role in the reversal of the phenomenon.

Klein and Seligman (1976) provided an additional set of observations that speak to the issue. Undergraduate college students were exposed to either solvable or unsolvable cognitive problems. Those subjects exposed to unsolvable problems were then given either solvable problems, a form of enactive exposure therapy, or no problems. Moreover, groups of mildly depressed college students also were given either the "therapy" treatment or not. Subsequent performance on a novel escape–avoidance task indicated that both groups given the "success" therapy, whether initially depressed or initially nondepressed but made "helpless" by exposure to the insolvable problems, showed no indication of performance deficits on the subsequent task. In this case, we may assume that the experience of success changed subjects' expectations about the ability of their responses to produce outcomes, and therefore averting the performance deficits typically associated with depression and helplessness.

Do clinically depressed individuals evidence patterns of change similar to those of analogue populations? Again, we are unaware of any direct evidence that speaks to this issue. Several studies are somewhat relevant, however. Loeb, Beck, and Diggory (1971) found that the experience of success on laboratory tasks increased optimism and enhanced performance among depressed outpatients. Such findings, of course, speak only to the notion that the actual disconfirmation of negative expectations via enactive exposure to success can enhance subsequent expectations and behaviors. It does not contrast expectational versus attributional modes of intervention.

Taylor and Marshall (1977) have provided such data. In the conext of a controlled clinical trial involving a quasi-analogue population (community volunteers for what was promoted as a therapy study), Taylor and Marshall contrasted a strictly cognitive, a strictly behavioral, and a combined cognitive–behavioral intervention. A fourth cell provided a waiting list control. Treatment was conducted in a six weekly, individual therapy sessions. Although the cognitive therapy did not consist solely of reattributional procedures, it relied heavily on such procedures. Change depended largely on the therapists' capacity to *persuade* the clients that they were not weak, incompetent, unlovable, etc. The procedures closely approximated those that comprise the heart of rational–emotive psychotherapy (Ellis, 1962; Ellis & Grieger, 1977; Ellis & Harper, 1975). The strictly behavioral approach relied heavily on the use of activity scheduling to ensure that clients experienced both pleasurable and mastery (success-producing) experiences. As such, the therapy relied heavily on enactive procedures without paying explicit attention to cognitive processes, either expectational or attributional. The combined cognitive–behavioral cell involved the use of both cognitive- and behavior-change procedures.

The results indicated that although both the strictly cognitive and the strictly behavioral cells outperformed the waiting list control, both single-focus modalities were outperformed by the combination of cognitive and enactive procedures. Shaw (1977) similarly found a combined cognitive–enactive approach superior to either a strictly enactive or a nonspecific modality, although both of these latter modalities proved superior to a waiting list control.

In recent reviews, Hollon (in press) and Hollon and Beck (1979) observed that in each separate study in which the combination of cognitive and enactive procedures was tested against alternative procedures, those cognitive–behavioral procedures proved superior to those alternative interventions. This finding was observed in the comparisons between cognitive–behavioral versus strictly cognitive procedures (Taylor & Marshall, 1977), versus strictly behavioral procedures (Shaw, 1977; Taylor & Marshall, 1977), versus systematic relaxation behavior therapy (McLean & Hakstian, 1979), versus nonspecific and/or dynamic interventions (Fuchs & Rehm, 1977; McLean & Hakstian, 1979; Shaw, 1977; Shipley & Fazio, 1973, studies I and II), and pharmacotherapy (McLean & Hakstian, 1979; Rush, Beck, Kovacs, & Hollon, 1977). These last two studies are particularly noteworthy, for traditional or dynamic psychotherapies have consistently fared poorly when compared to drugs (Covi, Lipman, Derogatis, Smith, & Pattison, 1974; Friedman, 1975; Klerman, DiMascio, Weissman, Prusoff, & Paykel, 1974). Although it is clear that pharmacotherapies (particularly the tricyclics, but also the monoamine oxidase inhibitors and, perhaps, lithium) are the current standard of efficacy in the treatment of clinical depression (Hollon & Beck, 1978) and are clearly more effective than traditional approaches, the superiority of cognitive–behavioral interventions to pharmacotherapy in the only two available completed trials is striking indeed. Two final studies deserve mention at this point. Hollon, Bedrosian, and Beck (Note 2) found that the addition of pharmacotherapy to cognitive–behavior therapy did not produce any greater change than did the cognitivie–behavior therapy alone, although both replicated the superior rates of change shown by the cognitive–behavioral intervention in Rush et al.'s 1977 comparison with drugs alone. Finally, Zeiss, Lewinsohn, and Munoz (1979) found no difference between strictly cognitive, strictly behavioral, and interpersonal approaches in treating outpatient community volunteers, essentially replicating Shaw's (1977) and Taylor and Marshall's (1977) findings of comparability between any of several *single* modality interventions.

All of these data are admittedly only indirectly relevant to our major thesis. They are, however, consistent; in controlled therapy outcome studies, combined cognitive–enactive procedures outperform strictly cognitive, strictly enactive, traditional dynamic, and even pharmacologic interventions. Available basic research findings, although again indirect, are also consistent with our formulation. Although strong data based on prospective tests of the hypothesis are clearly needed, the available data appear supportive of the cognitive–expectancy formulation.

ATTRIBUTIONAL VERSUS EXPECTATIONAL APPROACHES

Abramson et al. (1978) and Seligman (Note 1) have proposed a system of intervention based on the reformulated helplessness model. This system consists of four major components; *(a)* environmental enrichment; *(b)* personal control training; *(c)* resignation training; and *(d)* attributional retaining. We would argue that these procedures, when active, operate largely through expectancy–disconfirmation procedures.

Environmental enrichment, which Seligman defines in expectational terms as changing the estimated probability of the relevant event's occurrence, need not depend on cognitive processes. When it does, however, it relies largely on altering the individual's expectations of gratification or security in the near and distant future.

Personal control training, defined by Seligman as changing the individual's expectation from uncontrollability to controllability, may require skills training when deficits exist but, more typically, requires the remediation of the *perception* of deficit. As with the bulk of the helplessness remediation procedures, (e.g., Klein & Seligman, 1976), the reversal of the inaccurate *expectation* of noncontrol is seen as a key process in the treatment of depression.

Resignation training, defined as making highly preferred outcomes less preferred (or highly aversive outcomes less noxious), is seen by Seligman as resulting largely from challenging beliefs held about the *implications* of those events [e.g., "If I fail in anything, it means I'm unsuccessful" (Beck, 1976; Ellis, 1962]. Implications are by definition conditional expectations about the future. We would argue that prospective tests of these expectations may provide the most compelling means of disconfirming these beliefs.

Finally, attributional retraining refers to changing attributions for failure from internal, stable, global to external, unstable, specific, and changing attributions for success from external, unstable, specific to internal, stable, global. Although it is not *necessary* to focus on events in a prospective fashion (e.g., one could, for example, discuss the reasons for losing one's *last* job), we would argue that it is more powerful to do so (e.g., focusing on efforts to test the client's expectation that he or she could not get, or could not hold, the *next* job). In a sense, the cognitive–expectancy theory argues for testing, often via enactive procedures, expectations about the stability and globality components of the reformulated helplessness model's attributional style.

We would concur that attributional processes are central to the onset of the disorder but would argue that expectations play an equally large role in the maintenance of the disorder and that the explicit disconfirmation of those expectations provides the most efficient means of reversing all the various dysfunctional cognitive processes. Efforts at therapy require shifting one's frame of reference from processes of etiology to processes of intervention.

THEORIES OF ETIOLOGY AND THEORIES OF INTERVENTION

We have, in our formulation, operated on the assumption that the reformulated helplessness model adequately describes the etiology of depression. The evidence for this model has been presented elsewhere (Abramson et al., 1978, and Chapter 1 of this volume). It is important to note that the validity of the cognitive–expectancy model does not depend on the validity of reformulated helplessness or its applicability to any given type of depression (i.e., helplessness depressions). Just as the efficacy or lack of efficacy of a pharmacological intervention does not prove that a biological process did or did not produce a depression, neither does the success or lack of success of a cognitive–behavioral intervention prove or disprove a psychological formulation of etiology. However, treatment efficacy does speak directly to the validity of the model of intervention on which it is based. Treatments that have little, if any, direct impact on those factors that caused a disorder may well prove effective. Theories of intervention must be flexible enough to encompass effective interventions.

Psychoactive drugs appear to be active interventions in about 70% of all unselected primary depressives; cognitive–behavioral interventions in at least that many or more (Hollon, in press). Are we to assign a unique, unimodal process of etiology to account for those same disorders that appear to be responsive to multiple interventions? Perhaps, but it may be preferable for any model of intervention to be able to account for the apparent multiplicity of points of intervention in depression. What has yet to be determined is exactly what types of depression exist and which types of interventions are most likely to be effective with which types of the disorder. Our suspicion is that any of several points of intervention will prove possible for the majority

of individuals and that the most effective type of intervention may not necessarily be that which is most similar to those processes that initiated that particular episode. Various pharmacologic approaches may well create an impact on exactly those cognitive processes discussed in the preceding section. The various cognitive–behavioral approaches may well influence exactly those neural processes central to the biologic models.

We suggest that a cognitive–expectancy model represents a "final common pathway" for interventions in depression, one that may operate *in relative independence of the specific nature of the etiological factors involved*. We prefer intervention procedures that focus on using enactive procedures to disconfirm negative expectations because these procedures represent the most effective means of nonpharmacological intervention, not because they are tied to any specific model of etiology.

Elsewhere (Hollon, in press; Hollon & Beck, 1979) we have argued for the necessity of discriminating between different parameters of change. For example, the *magnitude* of the reduction of symptoms during an episode may be more differentially affected than the *stability* of those changes over time. Clearly, pharmacologic interventions, once discontinued, provide little protection against subsequent relapse or reoccurrence of the disorder. Similarly, such relapses or reoccurrences are more likely to be forestalled by cognitive–behavioral interventions for *some types of etiological processes than for others*. Helplessness-based depressions, should they exist, are most likely to be forestalled by cognitive learning-based interventions that alter information processing styles. Genetically based biolgic depressions are less likely to be prevented. Either type, or other types, of depression may well prove amenable to any of the several types of interventions with respect to the magnitude and rapidity of the reversal of symptomatology.

SUMMARY

We have described a cognitive–expectancy theory of therapy for depression. This theory of change holds that those procedures (typically some combination of enactive and symbolic techniques) that most directly and explicitly disconfirm negative expectations most rapidly and effectively reverse the negative affect, cognitive distortions, and behavioral passivity central to depression. The optimal model for the therapeutic relationship is one of collaborative empiricism (Hollon & Beck, 1979), in which client and therapist work together to test the client's beliefs.

The cognitive–expectancy theory of change is seen as compatible with, although not dependent on, reformulated helplessness; if that model's attributional account of the etiology of depression is correct, the procedures generated by a cognitive–expectancy theory of change should provide the optimal description of effective intervention. Specific emphasis on changing attributions is seen as being inefficient, because it requires altering beliefs about events that have already occurred in a post hoc fashion. Specific emphasis on testing those expectations that were, perhaps, generated by dysfunctional attributional styles permits the use of prospective, enactive hypothesis testing and serves as the most efficient means of altering existing belief systems or attributional styles. We believe that we have not presented a theory of change that breaks with the reformulated helplessness model. We have instead formulated a model of treatment that describes the most efficient procedures of intervention, if that reformulated helplessness model proves to be correct.

REFERENCE NOTES

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