
Cognitive Therapy

A 30-Year Retrospective

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Several lines of investigation have evolved from the initial cognitive model of depression and other disorders. A large number of studies have tested the cognitive model using both clinical and laboratory-based strategies. In general, studies that most closely approximate the clinical conditions from which the theory was derived are supportive of the cognitive model of depression. Studies of anxiety and panic, although fewer, generally support the cognitive model of anxiety and panic. The application to the treatment of clinical problems has been promising and supports the concept of cognitive specificity. The cognitive therapy of depression has led to the utilization of specific cognitive strategies based on the specific conceptualizations of a given disorder to a wide variety of disorders. Study of abnormal reactions has also provided clues to the cognitive structure of normal reactions.

Fifteen years have elapsed since I called for the admission of cognitive therapy into the therapeutic arena (Beck, 1976, p. 337), and 30 years have gone by since I first formulated my cognitive model of depression based on research on dreams and other ideational material (Beck, 1961). I suggested in 1976 that in order to qualify as a system of psychotherapy a particular brand of psychotherapy had to provide (a) a comprehensive theory of psychopathology that articulates with the structure of psychotherapy, (b) a body of knowledge and empirical findings that support the theory, and (c) credible findings based on outcome and other studies to demonstrate its effectiveness.

What is the status of cognitive therapy today? A steady flow of studies largely support the cognitive model of depression (Ernst, 1985). This model has facilitated the development of strategies and techniques to provide a psychotherapeutic structure. Numerous outcome studies have supported the effectiveness of the therapy in the treatment of unipolar outpatient depression, anxiety disorders, and panic disorder. A meta-analysis of 27 studies (Dobson, 1989), for example, has demonstrated the efficacy of cognitive therapy in unipolar depression and its superiority to other treatments, including antidepressant drugs. More striking has been the success of cognitive therapy in maintaining gains and preventing relapse. Five published studies have indicated that cognitive therapy has a greater prophylactic effect than do antidepressant drugs (Hollon & Najavits, 1988). A more recent study,

the much-publicized National Institute of Mental Health collaborative study of the treatment of depression, has shown superiority of cognitive therapy in follow-up, in comparison with antidepressant drug and interpersonal therapy (Shea et al., 1990).

Even more impressive has been the application of cognitive therapy to panic disorder. On the basis of the cognitive model of panic (Beck, 1976, 1987a; D. M. Clark, 1986), practically complete reduction of panic attacks after 12–16 weeks of treatment has been reported (Sokol, Beck, & Clark, 1989; Sokol, Beck, Greenberg, Berchick, & Wright, 1989). Also impressive has been the successful application of cognitive therapy to generalized anxiety disorder (Butler, Fennell, Robson, & Gelder, 1991), eating disorders (Garner & Bemis, 1982), heroin addiction (Woody et al., 1984), and inpatient depression (Miller, Norman, & Keitner, 1989). Further clinical work suggests the utility of cognitive therapy in treating diverse disorders such as couples' problems (Beck, 1988) and schizophrenia (Perris, 1988). A striking feature of the diverse application has been the importance of cognitive specificity. Each disorder has its own specific cognitive conceptualization and relevant strategies that are embraced under the general principles of cognitive therapy (Beck, 1976; Beck & Freeman, 1990; Beck, Rush, Shaw, & Emery, 1979).

Because of the breadth of cognitive therapy and its therapeutic eclecticism, and the ability of cognitive theory to explain changes in psychopathology, the question has been raised as to whether cognitive therapy might be viewed as the product of the integration of the effective psychotherapies (Alford & Norcross, in press). The theoretical framework of cognitive therapy appears to articulate well with contemporary developments in cognitive psychology and social psychology (Hollon & Garber, 1990), as well as earlier concepts of developmental psychology (Beck, 1967). In fact, there appears to be a kind

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of convergent evolution of concepts from the cognitive model of psychopathology and those of cognitive psychology. Moreover, many of the recent studies of the cognitive model of depression and anxiety disorders have borrowed techniques from cognitive psychology (e.g., Mathews, 1990).

Initial Observations and Formulations

My original observations of depressed patients were based on their verbalizations and free associations while they were undergoing psychoanalytic treatment with me. At one point I observed to my surprise that my patients experienced specific types of thoughts of which they were only dimly aware and that they did not report during their free associations. In fact, unless they were directed to focus their attention on these thoughts, they were not likely to be very aware of them. Although these thoughts seemed to be on the periphery of the patients' stream of consciousness, they appeared to play an important role in the psychic life of these patients (for a fuller description of this "discovery" see Beck, 1976, pp. 29–35). These thoughts (cognitions) tended to arise quickly and automatically, as though by reflex; they were not subject to volition or conscious control and seemed perfectly plausible to the individual. They were frequently followed by an unpleasant affect (in the case of the depressed patients) that the patients were very much aware of, even though they were unaware of, or barely aware of, the preceding automatic thoughts.

When I directed the patients to focus their attention on these "automatic thoughts," they began to report a string of them, particularly in response to a cognitive probe, "What are you thinking right now?" Connecting these thoughts brought out certain negative themes such as deprivation, disease, or defeat. Grouped together they fell into the category of a negative view of the present, past, and future experiences. Later, in working with more severely depressed patients, I noted that these types of thoughts were no longer peripheral but occupied a dominant position in consciousness and were repetitive.

It seemed to me that I had tapped another level of consciousness in the recognition of automatic thoughts, perhaps analogous to the phenomenon described by Freud as "preconscious." This level of consciousness seemed to be relevant to what people say to *themselves* and was involved in the system of self-monitoring rather than what they might say in the conversational mode, their customary way of communicating with other people. Thus, the automatic thoughts were conceived of as being part of an internal communication system, as opposed to the interpersonal communication that was more involved in the discussion with other people (Beck, 1976, pp. 24–46). Hence, the patients were less likely to report these cognitions in free association but were readily taught to focus on them when the therapist (or the patients themselves), noting a change in affect, used the cognitive probe. Following my initial observations, I discovered that Ellis (1962) reported similar observations.

The negativity permeated the "internal communi-

cations" such as self-evaluation, attributions, expectancies, inferences and recall, and were manifested in low self-esteem, self-blame and self-criticism, negative predictions, negative interpretations of experiences, and unpleasant recollections. In ambiguous situations, the depressed patients were particularly prone to make a negative interpretation when a positive one would seem to be more appropriate; they would not only magnify their own unpleasant experiences but would either blot out or label as negative their experiences that other people would consider positive.

I also noted a variety of errors in the patients' depressive thinking, which I labeled selective abstraction, overgeneralization, dichotomous thinking, and exaggeration (of the negative aspects of their experiences). Furthermore, I noted that depressed patients tended to predict specific negative outcomes from specific tasks that they might undertake and expected long-range, bad outcomes to their life in general. A high degree of such negative expectations ("hopelessness") appeared to be predictive of suicide. These phenomenon appeared to be universal across all types or subtypes of depression: reactive (nonendogenous), endogenous, bipolar, or organic. They also appeared whenever depressive symptomatology was present—irrespective of whether the primary diagnosis was depression, schizophrenia, or some other disorder.

To account for the regularities in negative thinking in depression, I postulated the presence of a *negative cognitive shift*. This thesis stipulates that there is a change in the cognitive organization so that much positive information relevant to the individual is filtered out (cognitive blockade), whereas negative self-relevant information is readily admitted.

Meaning, Symbolism, and Schemas

I was struck by how ascertaining the idiosyncratic or special meanings people attached to events helped to explain what might otherwise have represented quite inexplicable affective and behavioral reactions. Highly personal meanings did not usually revolve around esoteric themes such as castration anxiety or psychosexual fixations, as might be suggested by classical psychoanalytic theory, but were related to vital social issues such as success or failure, acceptance or rejection, respect or disdain. Moreover, these meanings were accessible to introspection. At times I would pick out what seemed to be a common theme across diverse circumstances and then induce the patients to focus on their thoughts or images in these situations. The psychotherapy sessions consequently provided a rich source of data for theoretical constructions.

Many of the meanings were fairly elaborate and were packed into a rather discrete stimulus situation. A man, for example, always reacted with the thought "She does not respect me" when his wife did not respond to him. A wife not receiving a smile from her husband consistently interpreted this as "He doesn't care for me." For them, a discrete (although ambiguous) behavior had a fixed meaning. Of course, at times such meanings may be rel-

atively accurate. But symbols are different from other complex stimuli in that the evoked meanings are not only powerful but invariant.

The internal representation of the meanings evoked by the symbols constitute a network of beliefs, assumptions, formulas, and rules and are often connected to memories relevant to the development and formation of such beliefs (Beck, 1964). The relevant beliefs interact with the symbolic situation to produce the "automatic thoughts."

Because of the continuity of content of beliefs over each recurrence of the disorder, I proposed that they existed in an inactive or latent state prior to and between depressions. These beliefs were encased in schemas that had a variety of properties in addition to content: valence, permeability, density, flexibility. However, whatever factors initiated the occurrence or recurrence of the disorder activated these schemas.

The depressed patients' interpretations of their experiences seemed to be shaped by certain absolute beliefs such as "I am worthless," "I can't do anything right," and "I am unlovable." Any situation that was remotely relevant to self-worth, ability, or social desirability was interpreted in terms of the corresponding belief. I suggested that these beliefs were formed earlier in life (Beck, 1967) and became embedded in a structure (cognitive schema). Subsequently, when specific life experiences impinged on these beliefs (schemas), they might precipitate a depression. I proposed that beliefs were generally framed in a conditional form such as "If I don't succeed, I am helpless."

A later observation suggested that two "types" of individuals—sociotropic or autonomous—were prone to become depressed after the occurrence of an adverse experience congruent with their personality. (Of course, these types simply represented extreme forms on the dimensions of sociotropy and autonomy.) Patients who were heavily invested in autonomy (independent achievement, mobility, solitary pleasures) were prone to become depressed after an "autonomous stressor" such as failure, immobilization, or enforced conformity. Patients who deeply valued closeness, dependency, and sharing were hypersensitive to and prone to become depressed after "sociotropic traumas" such as social deprivation or rejection (Beck, 1983).

It eventually became apparent that the same types of beliefs predisposed individuals to develop anxiety disorders—the difference being that in anxiety disorders the congruent stressors were the *threat* of failure or abandonment rather than the actual occurrence of the event.

Extrapolations to the Normal From the Abnormal

I believe that we have learned and can still learn a great deal about normal functions from the study of psychopathology. For example, the systematic negative bias in depression and positive bias in mania (Beck, 1967) supports the presence of similar but more subtle biases in normal everyday reactions. Indeed, positive bias has been

long recognized and demonstrated more recently in "illusory glow" experiments (Alloy & Abramson, 1979). Not so obvious is the clarification of everyday worries about health, and so forth, initiated by studies of cognitive aspects of hypochondriasis (Salkovskis, 1989) and panic (Beck, 1976, 1987a). The overconcern about evaluation in the social phobias and of physical danger in the impersonal phobias (e.g., heights, crowded spaces, small animals) point to similar sources of anxiety in the psychology of everyday life.

The biases also suggest how the "cognitive shift" can influence the content at each stage of cognitive processing. Starting with preferential selection (abstraction) of data, through the evaluation, interpretation, and recall from short-term memory, the content of cognitive processing is determined by the activated schemas. Even retrieval from long-term memory is influenced by these schemas.

Continuity Hypothesis

Various psychopathological syndromes appear to represent exaggerated and persistent forms of normal emotional responses. Thus, there is a continuity between the content of "normal" responses and the excessive or inappropriate emotional experiences associated with psychopathology. In depression, the sense of defeat and the withdrawal of investment in people and customary goals becomes pervasive and unremitting and, consequently, sadness is pervasive and unremitting. In mania, the investment in expansion and goal-directed activity, and consequently, euphoria, is increased. Anxiety disorders are manifested by a generalized, intensified sense of vulnerability and a consequent motivation toward self-defense and escape.

The model of psychopathology proposes that the excessive dysfunctional behavior and distressing emotions or inappropriate affect found in various psychiatric disorders are exaggerations of normal adaptive processes (Beck, 1976).

Typology of Emotions

The study of the clinical data led to the formulation of a typology of "normal" emotions. I conceived of at least four basic emotions that were evoked by a specific cognitive profile or conceptualization. In short, the cognitive structuring of loss, gain, or threat led to a specific corresponding emotion. Sadness appeared to be invoked by the perception of loss, deprivation, or defeat. The response is withdrawal in the lost goals and emotional investment from the source of disappointment. In contrast, elation is produced by perception of a gain. Anxiety and anger, "negative" emotions, are both elicited by perceived threats, but the content of the focus differs. In anxiety, the focus is on the individuals' vulnerabilities, which they attempt to protect through avoidance, escape, or inhibition. (As a way of clarifying the confusing terminology, I have used the term *fear* to denote the cognitive, or intellectual, appraisal of a danger—for example, fear of falling—and the term *anxiety* to designate the emotional consequence of this appraisal; Beck & Emery, 1985.) In

contrast, angry individuals focus more on the offensive qualities of the threat than on their own vulnerability and seek to eliminate the threat through counterattack. Limited support for the cognitive configurations have been provided by Wickless and Kirsch (1988).

Evolutionary Origins of Cognitive Programs

Another set of speculations attempts to tie in the structural patterns (cognitive schemas) to ethology and evolutionary mechanisms. I proposed that the analog of cognitive structures relevant to depression, anxiety disorders, and the like did not originate *de novo* with *homo sapiens* but evolved through the millennia. Programs that could have had survival value in the wild that were not well adapted to the complexities of modern life could be involved in psychopathology (Beck & Freeman, 1990).

Although there are some risks in extrapolating from animal to human ethology, the similarities are so striking that writers have used animal observations as a way of clarifying human reactions (Darwin, 1872). I think that animal analogies provide a basis for clarifying many aspects of normal and abnormal human behavior (for anxiety and its disorders see Beck & Emery, 1985). More recently I have been impressed by the relevance of observations of primate behavior to depression in humans (Beck, 1987b).

Challenges to the Cognitive Model

The Question of Causality

One of the propositions most frequently attributed to the cognitive model of depression is "cognitions cause depression" (e.g., see Lewinsohn, Steinmetz, Larson, & Franklin, 1981). I have argued elsewhere that it seems far-fetched to assign a causal role to cognitions because the negative automatic thoughts constitute an *integral* part of depression, just like the motivational, affective, and behavioral symptoms. To conclude that cognitions cause depression is analogous to asserting that delusions cause schizophrenia (Beck et al., 1979).

First, consider the definition of the term *cognitions*. I have used this word at times as a more technical term for automatic thoughts. As such, cognitions or automatic thoughts, according to my observations, exist as a common denominator of all kinds of depression and in fact may be essential signs of depression. Confusion may arise, however, as a result of the primacy hypothesis, which states that when the depression is established the interpretations as manifested in automatic thoughts or cognitions shape the affective, behavioral, and motivational responses. Intervention at the cognitive level may reduce the other symptoms, whereas persistence or exacerbation of the cognitive processes may maintain or increase the other symptoms.

Cognition as a singular noun refers to various processes in cognitive or information processing: perception, interpretation, recall, and, as such, comprises a component of a circular model. Each of the psychological systems (cognition, affection, motivation) is interconnected

so that changes in one system may produce changes in other systems. Thus, an individual made artificially sad or anxious (e.g., as a side effect of a drug) may then "read" the sadness or anxiety as indicative of loss or danger. The motivational system relevant to relapse into passivity or flight may be activated.

A second source of confusion, related to the first, is that depression is commonly viewed as a mood state, pure and simple (Beck, 1971). This concept has perhaps been abetted by the subsuming of depression under the rubric of affective or mood disorders in the various diagnostic and statistical manuals of the American Psychiatric Association. Consequently, negative cognition has been treated as something apart from depression—as an epiphenomenon, cause, or consequence (Lewinsohn et al., 1981). A related source of confusion has arisen from experimental studies of mood induction. As Riskind (1983) has pointed out, these manipulations can as well be described as "cognitive priming" as mood induction.

A third source of confusion has been my postulation of the role of cognitive schemas in depression and anxiety. In this instance, the schemas (according to theory) become activated. The highly charged negative schemas preempt the more adaptive schemas and thus constitute the negative cognitive shift. Other writers may have assumed that I regarded the schematic change as the "cause" of depression. However, I have considered the activation of the schemas to be a mechanism by which the depression develops not as the cause. The cause may be in any combination of biological, genetic, stress, or personality factors, which also may be offset by any combination of such factors (Beck, 1967).

Having said this, I acknowledge that my theory does include the notion that *in some cases* the congruence of personality and stressor, in the presence of other possibly unidentifiable factors, may play a causal role ("reactive depression").

A more complete elaboration of the role of cognition must address questions such as (a) What factors produce a shift in the information processing to the negative and what factors maintain the shift? We know, for example, that certain drugs (e.g., antihypertension drugs) can produce such a shift. (b) How do stress factors interacting with personality lead to such a shift? (c) What is the role of protective factors (e.g., social support, insight, coping mechanisms, etc.) in preventing such a shift? (d) Because antidepressant drugs and cognitive therapy produce the same end result (e.g., cognitive change as well as change in biological factors), do they operate through similar or different brain mechanisms? (e) Because follow-up studies consistently indicate greater stability of results and fewer relapses with cognitive therapy than with antidepressant drugs, is this an indication of its impact on additional brain mechanisms or on a more durable impact on the same brain mechanisms?

Role of Interpersonal Factors

The cognitive model has been criticized for ignoring interpersonal factors in the genesis of depression (Coyne &

Gotlib, 1983). In fact, I have argued elsewhere that in most cases (except possibly for continuous cycling bipolar cases) depression does not occur in a vacuum. Perhaps the most frequent environmental stressors have to do with relations with other people. The role of the interaction of the cognitions of one person with those of another has been described at some length in a recent volume (Beck, 1988). In essence, a dyadic interaction, as in a married couple, may lead to pathological outcome when the individuals consistently misunderstand each other's behavior and misread each other's motives and act on this misconstruction. Thus, an autonomous wife may interpret her dependent husband's behavior as "He wants to control me." (He has a fear of abandonment and wants to get constant reassurance.) She interprets this as "He wants to control me." She withdraws angrily, which he interprets as "She doesn't really care about me." He demands more reassurance, resulting in further distancing himself, and he slips into a depression: "Since she doesn't love me, I'm unlovable."

Obviously, the depressed individual's psychological systems continue to interact with those of other people even after depression has occurred. A depressed wife, for example, may interpret her husband's frustration at not being able to help her as a sign of rejection (husband's cognitions: "I can't do anything to help her"; wife's cognitions: "He has given up on me because he doesn't care"). The wife reacts with further withdrawal, which triggers further withdrawal of support by the husband (Beck, 1988). The fact that cognitive therapy can help to reverse depression indicates that interpersonal factors have an impact on depression.

Depressive Realism

A number of articles (e.g., Alloy & Abramson, 1979) have suggested that the problem in depression is that the patients see events too realistically (for a critique of this research, see Ackerman & DeRubeis, in press). The clinical material, however, seems to suggest the following: First, when negative events occur that are complex or abstract, the patient attaches a broad global self-evaluative meaning or explanation. Second, the patient does not think of alternative explanations. Often in treatment therapists find that the patient, when prodded for a more logical explanation, is able to drop his or her negative interpretation. Third, the negative bias is most likely to be manifested when the data (a) are not immediately present in the here and now, (b) are not concrete, (c) are relevant to self-evaluation, and (d) are ambiguous (Riskind, 1983).

Thus, clinically, the patient is more likely to produce exaggerated negative inferences when integrating past events or projecting into the future, when making attributions for which there are no clear-cut criterion on which to base judgments, or when making vague (but crucial) inferences about his or her character.

At this stage of knowledge, it seems that the greatest explanatory power is provided by a model that stipulates that (a) the nondepressed cognitive organization has a

positive bias, (b) as it shifts towards depression, the positive cognitive bias is neutralized, (c) as depression develops, a negative bias occurs, (d) in bipolar cases there is a pronounced swing into an exaggerated positive bias as the manic phase develops.

Empirical Studies of Depression

Considerable research designed to test various hypotheses generated by the cognitive model of depression has been conducted. In a review of 180 articles incorporating 220 studies of this model, Ernst (1985) reported that 91% supported and 9% did not support the model. He divided his survey into three parts: cognitive triad (150 supportive, 14 nonsupportive), schemas (31 supportive, 6 nonsupportive), and cognitive processing (19 supportive, 0 nonsupportive). In general, he found that the more the studies approximated the clinical observations, the more likely they were to confirm the derived hypotheses. For example, studies of dysphoric student subjects were less likely to be supportive than studies of clinically depressed patients. A more recent critical analysis (Haaga, Dyck, & Ernst, in press) pinpointed a number of methodological deficiencies in many of these studies.

Negativity Hypothesis

Of all the hypotheses, pervasiveness of negative thinking in all forms of depression, symptomatic or syndromatic, has been the most uniformly supported (Haaga et al., 1990). In early studies (Beck, 1967), dream themes, early memories, measures of self-concept, and responses to projective tests showed a heavy degree of the idiosyncratic content typical of depressives when compared with nondepressed psychiatric patients. Specific questionnaires designed to test components of the cognitive triad (e.g., Beck, Brown, Steer, Eidelson, & Riskind, 1987; Beckham, Leber, Watkins, Boyer, & Cook, 1986; Crandall & Chambless, 1986) have been well documented.

Eaves (1982), for example, showed that the Automatic Thought Questionnaire (Hollon & Kendall, 1980) correctly separated 97% of depressive from normal subjects and did not misidentify any of the normal subjects as depressed.

The *universality* of the cognitive phenomena has been found across all types and subtypes of depression, unipolar and bipolar, reactive and endogenous (see, e.g., Hollon, Kendall, & Lumry, 1986).

The *content-specificity* hypothesis proposes that each disorder has a specific, exclusive cognitive profile. Because most of the clinical overlap occurs between depressive and anxiety disorders, the bulk of the research has been directed toward contrasting the specific cognitive content of depression (loss, defeat, deprivation) with anxiety (danger, threat). The Cognition Checklist, for example, differentiated depressed and anxious patients on the basis of their reciprocal scores on the subscales. Depressive patients scored higher on Loss-Defeat subscales, whereas anxious patients scored higher on the Danger subscale (Beck et al., 1987). Furthermore, compared with anxiety patients, depressed patients assigned high probabilities of

a negative outcome of their specific problems and a low probability of a positive outcome (Beck, Riskind, Brown, & Sherrod, 1986).

The specificity hypothesis was further supported by Greenberg and Beck (1989), who found that on self-endorsement and recall, depressives tended to endorse depressive-content (loss, etc.) cognitions, whereas anxiety patients endorsed anxiety-content (danger, etc.) cognitions.

Finally, a factor analysis of all of the cognitive scales designed to measure specifically the cognitive content of depression or anxiety produced the appropriate loadings on the depression and anxiety factors (D. A. Clark, Beck, & Brown, 1989).

Cognitive Primacy

I proposed that in depression the negative processing of information leads to the other symptoms (Beck, 1964). Although it is difficult to establish primacy of any single phenomenon, the tests of this hypothesis have been diverse and generally supportive. One approach indicated that changes in cognition preceded changes in affect (Rush, Weissenburger, & Eaves, 1986). Another line of inquiry showed that manipulation directed at increasing negative thought content in depressed patients increased self-report and corrugator electromyograph indices of depression. Conversely, Teasdale and Fennell (1982) demonstrated that active negative thought content reduction led to the greatest reduction in negative affect in depressed patients.

Finally, Beck, Kovacs, and Weissman (1975) attempted to address this issue through focusing on a specific hypothesis, namely, that hopelessness is the crucial cognitive ingredient of suicidal intent. Clinical studies indicated that hopelessness was the variable linking depression to suicidal wishes. Two prospective studies showed that patients with elevated scores on the Beck Hopelessness Scale were more likely to commit suicide over a five-year follow-up than were patients with lower scores (inpatients: Beck, Steer, Kovacs, & Garrison, 1985; outpatients: Beck, Brown, Berchick, Stewart, & Steer, 1990).

Cognitive Processing

Studies for the most part have supported the observation that in linking the cognitive chain—perception, recall, interpretation—a biased processing of negative material among depressives becomes obvious. It should be pointed out that this cognitive processing is no more conscious than the functioning of the internal organs, but its products may be conscious (Beck, 1987b).

Perception. A number of studies (Dunbar & Lishman, 1984; Powell & Hemsley, 1984) have indicated a lower recognition threshold for briefly exposed negative verbal or pictorial stimuli in depressed patients than in nondepressed control subjects. A more “physiological” study showed more efficient processing of negative verbal stimuli in depressed than in nondepressed patients using the P300 waves as a marker (Blackburn, Roxborough, Muir, Glabus, & Blackwood, 1990). Gilson (1983) found

that although normal subjects were more likely to perceive positive scenes, depressed subjects perceived negative scenes more frequently in a binocular rivalry experiment.

Recall. Negative bias in recall of negative adjectives in depression has been reported by Bradley and Mathews (1988). Furthermore, depressed patients are more likely to underestimate recall of positive relative to negative feedback (DeMonbreun & Craighead, 1977; Gotlib, 1981).

Long-term memory. D.M. Clark and Teasdale (1982) retrieved more negative memories at a time of day when patients were more depressed than at a time of day when they were less depressed.

Negative inferences. A number of studies administered scenarios to patients with multiple choices for conclusions or outcomes. The studies consistently showed a bias in favor of a negative personal meaning among depressed patients (e.g., Krantz & Hammen, 1979).

Congruence Between Personality and Stressors

After my own clinical observations that patients who placed great stock in closeness, intimacy, and dependency and had relevant beliefs (e.g., “If I am not loved I can never be happy”) were hypersensitive to any event that appeared to represent withdrawal of affection or support, I proposed that congruence between external events and specific personality types might produce depression. At the same time, Shaw (personal communication, 1980) suggested that the more autonomous patients he treated were hypersensitive to perceived failure.

In order to test this notion, my group developed a scale (the Sociotropy–Autonomy Scale) designed to locate patients on belief dimensions of autonomy and sociotropy. The “pure” groups selected for high scores on one dimension and low on the other would, for experimental purposes, be designated as sociotropic and autonomous. A number of studies then sought to show relations between the “personality type” and the corresponding stressor. A number of somewhat problematic retrospective studies of depressed patients provide mixed support for this hypothesis. In one study, however, Hammen and her group (Hammen, Ellicott, Gitlin, & Jamison, 1989) reported a congruence of life events and type of personality. A later, more refined study (Hammen, Ellicott, & Gitlin, 1989) showed that this relationship held only for patients scoring high on the autonomy scale. In contrast, Segal, Shaw, and Vella (1989) found congruence of life events only among sociotropic patients who relapsed.

Prospective studies of normal individuals exposed to naturalistic stressors provided useful information for understanding the stress–diathesis relationship. Stiles’s (1990) study of depressive symptom formation in Norwegian Army recruits separated from their families and assigned to training in northern Norway indicated that those individuals who developed symptoms of depression scored higher on the Dysfunctional Attitude Scale (Weissman & Beck, 1978) at time of induction than those who did not. In future studies, cognitive vulnerability may best be studied during asymptomatic periods, using

priming techniques such as those described by Miranda and Persons (1988).

Conclusion

To return to the question I posed in 1976, Can a fledgling psychotherapy challenge the giants in the field—psychoanalysis and behavior therapy?, it seems that the work of the past three decades supports the cognitive model of depression and, to an increasing degree, that of panic disorders, generalized anxiety disorder, and other disorders. Work has been done to address the concerns of Coyne and Gotlib (1983) and other critics, but more remains to be done to shore up the cognitive model.

Clinical studies indicate the utility of cognitive therapy in a wide variety of disorders, particularly depression and the anxiety disorders. Further systematic studies of the efficacy of cognitive therapy in the treatment of a broad range of psychopathology remain to be executed. The preparation and publication of treatment manuals incorporating specific cognitive conceptualizations and congruent strategies for diverse conditions such as delusional and impulsive disorders have already laid the groundwork for these studies. At this point in time, cognitive therapy is no longer fledgling and has demonstrated its capacity to fly under its own power. How far it will fly remains to be seen.

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