# The Current State of Cognitive Therapy

## A 40-Year Retrospective

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The basic framework of the cognitive theory of psychopathology and cognitive therapy of specific psychiatric disorders was developed more than 40 years ago. Since that time, there has been continuing progress in the development of cognitive theory and therapy and in the empirical testing of both. A substantial body of research supports the cognitive model of depression and, to a somewhat lesser extent, the various anxiety disorders. Cognitive therapy (CT), often labeled as the generic term *cognitive behavior therapy*, has been shown to be effective in reducing symptoms and relapse rates, with or without medication, in a wide variety of psychiatric disorders. Suggestions for future research and applications are presented.

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Recent evaluations of the cognitive approach have characterized it as "the fastest growing and most heavily researched orientation on the contemporary scene."[1(p369)] Approximately 40 years after the initial publication in *Archives of General Psychiatry* of the formulation of the role of cognition in depression[2] and in therapy,[3] it is appropriate to review the evolution of the cognitive models of psychopathology and CT from their earliest description to their current status.

From the beginning, the formulation of a coherent conceptual framework preceded the development of therapeutic strategies. Although my original focus was on depression, I considered that this approach could also clarify other disorders.[3] My research strategy thus involved several stages: to try to identify the idiosyncratic cognitive elements derived from clinical data in various disorders, to develop and test measures to systematize these clinical observations, and to formulate guidelines for therapy. My guidelines for developing and evaluating a novel system of psychopathology and psychotherapy consisted of the following plan: (1) to construct a comprehensive theory of psychopathology that articulated well with the psychotherapeutic approach; (2) to investigate empirical support for the theory; and (3) to conduct empirical studies that tested the efficacy of the therapy.[4] This theoretical and therapeutic set of principles was systematically applied to a sequence of disorders starting with depression,[5,6] suicide,[7] then anxiety disorders and phobias,[8] panic disorder,[9] followed by personality disorders,[10] and substance abuse.[11] I also made brief forays into the areas of interpersonal problems[12] and anger, hostility, and violence.[13] More recently, this approach was used to clarify the psychological structure and CT of schizophrenia.[14,15]

## EVOLUTION OF THE COGNITIVE MODEL

My formulation of the theoretical structure of CT was influenced in part by the cognitive revolution in psychology in the 1950s and 1960s, especially the writings of George Kelly, PhD,[16] and Albert Ellis, PhD.[17] The original cognitive approach to psychopathology was based on an information-processing model. Simply stated, the cognitive model of psychopathology stipulates that the processing of external events or internal stimuli is biased and therefore systematically distorts the individual's construction of his or her experiences, leading to a variety of cognitive

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errors (eg, overgeneralization, selective abstraction, and personalization).[3] Underlying these distorted interpretations are dysfunctional beliefs incorporated into relatively enduring cognitive structures or schemas. When these schemas are activated by external events, drugs, or endocrine factors, they tend to bias the information processing and produce the typical cognitive content of a specific disorder.

The cognitive model was initially constructed to explain the psychological processes in depression. On the basis of systematic clinical observations, I proposed that the symptoms of depression could be explained in cognitive terms as follows: the biased interpretations of events are attributed to the activation of negative representations of the self, the personal world, and the future (the negative cognitive triad). A variety of dysfunctional beliefs that make individuals prone to depression make them vulnerable to specific life events that impinge on this vulnerability. For example, depression-prone individuals have beliefs such as "If I can't succeed at what's important, I'm a failure." The individual's perception of a major failure or a series of failures can activate the negative cognitive triad: the negative representations of the self, personal world, and future.[5] Underlying the cognitive triad is a cluster of dysfunctional beliefs and assumptions.[18]

## TESTS OF THE THEORY OF DEPRESSION

A considerable body of research testing the cognitive model of depression has accumulated since its initial formulation in 1967.[5] A 1985 review of 180 articles, which incorporated 220 studies of this model,[19] reported that 91% of the research studies supported the model, whereas 9% were nonsupportive or contradictory. The survey addressed 3 components of the cognitive theory: the negative cognitive triad in depression (150 supportive and 14 nonsupportive), negatively biased cognitive processing of stimuli (19 supportive and 0 nonsupportive), and identifiable dysfunctional beliefs (31 supportive and 6 nonsupportive). Negatively biased interpretations have been found universally in all forms and subtypes of depression: unipolar and bipolar, reactive and endogenous.[20] A critical evaluation of these studies[21] cited methodological problems in several of them. A subsequent analysis of several hundred later studies showed that more methodologically adequate studies yielded findings similar to those in the original study by Ernst (Donald Ernst, PhD, unpublished data, 1985).

A crucial hypothesis of the cognitive model has been the notion that certain beliefs constitute a vulnerability to depression (stress diathesis model). A refined model of depression proposed that the predisposing beliefs could be differentiated according to whether the patient's personality was primarily autonomous or sociotropic. Although the findings were mixed, the predominant evidence indicated that autonomous individuals were more likely to become depressed following an autonomous event (eg, a failure) than following a sociotropic event (eg, loss of a relationship), and the reverse was true of sociotropic individuals.[22] At least 1 prospective study[23] supported the stress diathesis hypothesis.

## COGNITIVE SPECIFICITY

The cognitive specificity hypothesis[4,24] proposes a distinct cognitive profile for each psychiatric disorder. Much research has been devoted to differentiating the cognitive themes in depression (loss and self-devaluation) and anxiety (threat and vulnerability). When relatively pure samples of depression and anxiety have been studied, a clear differentiation has been obtained. Depressed patients, for example, scored higher than anxious patients on the loss-defeat subscales of the Cognitive Checklist for Depression and Anxiety, whereas the reverse was true for anxious patients.[25] Moreover, depressed patients consistently received higher scores on the Hopelessness Scale than anxious patients in a broad range of studies[21,26]; compared with anxious patients, they attached a much higher probability of a negative outcome and lower probability of a positive outcome to their specific problems (A.T.B., Amy Wenzel, PhD, Marjan Ghahramanlou-Holloway, PhD, John H. Riskind, PhD, Gary Brown, PhD, and Robert A. Steer, EdD, unpublished data, 2005).

Specific cognitive profiles have been demonstrated in a wide variety of disorders, including anorexia nervosa,[27] obsessive-compulsive disorder,[28,29] panic disorder,[30,31] and body dysmorphic disorder.[32] The cognitive specificity hypothesis was validated in a study that differentiated 4 disorders on the basis of their content-specific selective attention effect: depression, anxiety, bulimia, and type A personality.[33] Moreover, each of the personality disorders has been differentiated on the basis of its distinctive set of dysfunctional core beliefs.[34,35]

## STUDIES OF SUICIDAL BEHAVIOR

Although not a standard psychiatric disorder in itself, suicidal behaviors are related to the psychiatric disorders. My group has studied suicidal behavior continuously since 1969, starting initially with a new classification of suicidal behavior: suicide ideation, attempted suicide, and completed suicide,[36] with qualifying variables of suicidal intent and degree of lethality of attempt. Assessments of intent for each group, as well as lethality for attempters, were found to be reliable[7] and had construct validity.[37,38] Analyses of prediction of ultimate suicide among suicidal ideators demonstrated the key psychological variables that independently predicted ultimate suicide: hopelessness[39] suicidal intent, especially at the worst point in the patient's history,[40] and an index of wish to die vs wish to live.[41] Key predictors of ultimate suicide among suicide attempters included regret due to the failure to succeed[42] and accuracy of expectation of the degree of lethality for the unsuccessful attempt.[43] In addition, we found that if the patients were accurate in their expectations regarding the degree of lethality of their index (unsuccessful) attempt, they were more likely to kill themselves at a later date than if they were inaccurate. A short-term 10-session CT intervention and prevention trial reduced the rate of reattempt by approximately 50% during an 18-month observation period.[44] It is now feasible to select the highest-risk suicidal patients among ideators and attempters and to provide adequate monitoring and interventions to reduce the likelihood of further suicide attempts.

## TESTS OF THEORIES
## OF ANXIETY DISORDERS

The basic cognitive model of the anxiety disorders stipulates that danger-oriented beliefs (embedded in cognitive schemas) predispose individuals to narrow their attention to threat, engage in dysfunctional "safety behaviors," and make catastrophic interpretations of ambiguous stimuli.[8] The danger-oriented bias is present in every phase of information processing (perception, interpretation, and recall)[45] and is present across all anxiety disorders (generalized anxiety disorder, posttraumatic stress disorder [PTSD], social anxiety, social phobia, and simple phobias). We have found a memory bias toward threat stimuli in generalized anxiety disorder,[25] and others found similar memory biases in panic disorder and PTSD.[46] Selective attention to threat words and pictures[47] and to masked stimuli[48] confirms that the biases occur automatically and are not necessarily under conscious control.

The cognitive model applied to panic disorder proposes that panic-prone individuals have exaggerated beliefs regarding the significance of ambiguous subjective experiences, which predispose them to make catastrophic interpretations of these sensations and to fixate their attention on their presumed pathological meaning.[30] Research has confirmed the presence of an array of dysfunctional beliefs regarding the presumed danger of these sensations,[49] the triggering of fearful cognitions during a panic attack,[8,31] and the total preoccupation with the symptoms and their catastrophic meaning to the exclusion of accessing corrective information (Amy Wenzel, PhD, Ian R. Sharp, PhD, Leslie Sokol, PhD, and A.T.B., unpublished data, 2005). The absorption with frightening visual imagery (eg, having a heart attack, dying, losing control, or going crazy) further consolidates the panic experience and blocks realistic appraisal of the event.[50]

The cognitive model applied to social anxiety and social phobia[8,51] proposes that patients focus their attention on the image they believe that others have of them and their performance and on their subjective sensations in socially evaluative situations. As a result, they become oblivious to objective social cues. Research has confirmed that these patients negatively distort their actual evaluation by others[52] and that their recollection of social experiences is also negatively biased.[53] Furthermore, the patients' anxiety increases and their objective performance decreases when they perceive negative, as opposed to neutral, images during exposure to socially threatening situations.

The cognitive model applied to PTSD by Clark and Ehlers[54] consists of 2 components: first, negative appraisal of the event itself and exaggerated negative appraisals of the symptoms produced by the traumatic event, and second, inadequate integration of the traumatic experience into the patient's autobiographical memory. The negative appraisal leads to beliefs such as "Nowhere is safe," "The next disaster will strike soon," "I'm dead inside," or "I'm going mad." In essence, the traumatic experience threatens patients' view of themselves and their personal world.

## DEVELOPMENT OF CT

The development of therapeutic packages was initially drawn from my own experience in conducting psychoanalytic therapy. The therapeutic strategies and concepts of CT included exploring the meaning (albeit conscious rather than unconscious) of the individuals' experiences, identifying consistent themes across their verbal reports and behavior, and connecting present and past experiences (especially with the personality disorders). The concepts of transference and early childhood experiences turned out to be crucial in understanding the personality disorders.

The focus on present problems as opposed to uncovering hidden traumas from the past and on analyzing accessible (rather than unconscious) psychological experiences differentiated the cognitive from the psychoanalytic approach. My introduction of the cognitive model in place of the psychoanalytic motivational model provided an explanation of dreams as reflections of cognitions rather than wish fulfillment.[55] The behavior therapy movement also made a solid impact on the structure of CT: greater activity by the therapist, operationalizing the specific procedures, setting goals for each session as well as for the long term, assigning homework, and especially measuring mediational variables and outcomes. Cognitive theory with its focus on intrapsychic processes rather than overt behavior was more akin to psychoanalytic theory, but the therapeutic procedures were more like behavior therapy.

The terms CT and *cognitive behavior therapy* (CBT) are frequently used as synonyms to describe CT based on the cognitive model. However, the term CBT is also used to designate a package of techniques in which a CT module is used in combination with a set of behavioral modules. In addition, CBT has been used as an umbrella term to include both standard CT and the atheoretical combination of cognitive and behavioral strategies. Because the literature reviews generally combine studies labeled CBT and CT under the CBT label, I will present the findings of these reviews and, where possible, summarize the more obvious CT studies.

## META-ANALYSES OF CT/CBT STUDIES

Butler et al[56] reviewed the meta-analyses of treatment outcomes of CBT/CT for a wide range of psychiatric disorders. A search of the literature from 1967 to 2003 ascertained a total of 15 methodologically rigorous meta-analyses that covered 9138 subjects and 332 studies. The review focused on effect sizes that contrasted outcomes of CBT with the outcomes for control groups for each disorder, providing an overview of the efficacy of CBT/CT. Large effect sizes (grand mean=0.90) were found for unipolar depression, generalized anxiety disorder, panic disorder with or without agoraphobia, social phobia, and childhood depressive and anxiety disorders. Moderate effect sizes (grand mean=0.62) were found for CBT of marital distress, anger, childhood somatic disorders, and chronic pain. Relatively small effect sizes (grand mean=0.32) were found for sexual offenders. Large uncontrolled effect sizes were also found for adjunctive CBT for schizophrenia from pretreatment to posttreatment (grand mean=1.23) and for bu-

limia nervosa (mean = 1.27). Among the limitations of the meta-analytic approach are the assumptions of uniformity across the studies in the samples, in the content of therapy, and in therapists.

Several challenges have been made to the CT and CBT studies[57,58]: overstating the findings, the allegiance effect, and the presumed lack of bona fide control. The bona fide control issue was addressed by the comparison of CBT with the gold standard for treatment of depression, specifically antidepressant medication (ADM). Seventeen studies[59] found that CT had a minimal superiority to ADM (effect size = 0.38). A more recent study by DeRubeis et al[60] found that CT and ADM had equivalent efficacy but that CT performed better in terms of relapse.[61] Severely depressed patients fared as well with CT as with ADM in an analysis of 4 studies.[62] In addition, CT was found to be effective in the treatment of atypical depression; CT performed as well as phenelzine sulfate, and both were superior to placebo.[63] For a comprehensive analysis of research on process, moderator, mediator, alliance, and other variables, as well as dismantling research studies, see Lambert.[64]

## PREVENTION OF RELAPSE

Eight studies[59] reported that the relapse rate 1 year following the end of treatment was 29.5% for CT/CBT vs 60% for ADM. Other studies have found that CT added to ADM after partial response to the ADM had a lower relapse rate at 6 weeks (29%) than ADM alone (47%).[65] A similar prevention of relapse and recurrence by CT was found in the highest-risk patients with recurrent major depression.[66] A recent study by Hollon et al[61] found a significant difference in relapse rate for responders who entered the continuation phase of CT (31%) vs the placebo withdrawal group (76%). The ADM continuation group had a relapse rate of 47%. Furthermore, 6-year follow-up[67] of short-term CBT after successful ADM showed a significant reduction in the recurrence of depression.

## CT OF ANXIETY DISORDERS

Both CT and CBT are effective in reducing anxiety symptoms for each of the *DSM-IV* anxiety disorders.[68] Although CT and CBT protocols for anxiety may emphasize specific cognitive and behavioral techniques to different degrees, their structure and procedures are similar, and both approaches to treatment are associated with substantial reductions in psychiatric symptoms, avoidance of feared stimuli, and dysfunctional cognitions.[69]

Meta-analyses indicate that CT/CBT protocols are more effective in reducing panic and anxiety symptoms than pharmacological treatments.[70] The efficacy of specific CT for panic disorder has been supported by several studies.[71-74] Meta-analyses of CT and CBT for social phobia have demonstrated the efficacy of these approaches.[75,76] D. M. Clark et al[77] found that a "pure" CT approach was more effective than fluoxetine. Standard CT for generalized anxiety disorder (ie, based on Beck and Emery[8]) has been shown to have a clear advantage to behavior therapy at follow-up.[78,79] In addition, CT was more effective than psychodynamic psychotherapy at posttreatment.[80] Of special note, patients who received CT for vari-

ous anxiety disorders who were followed up for 4 years showed significantly lower levels of symptom severity and negative affect than the randomized treatment control groups (behavior therapy or psychodynamic therapy).[81]

Theory-driven CT for PTSD was shown to be efficacious in several studies.[82,83] An effectiveness study compared patients who developed PTSD following a car bomb that exploded in the center of Omagh, Northern Ireland, in August 1998.[84] Significant improvement in PTSD was observed in the pretreatment-to-posttreatment effect sizes.

## OTHER APPLICATIONS

It appears that CT/CBT offers a promising addition to the standard treatment of severe mental disorders. Besides the interesting results of CT/CBT as an adjunctive treatment for schizophrenia, a promising lead to the prevention of schizophrenia was reported in a randomized controlled study of CT with very high risk groups.[85] The application of CT as an adjunctive treatment in the prevention of relapse in bipolar disorder as well as its cost-effectiveness has also been reported.[86,87] Some other empirically supported applications of CT/CBT include anorexia nervosa,[88] body dysmorphic disorder,[32] pathological hoarding,[89] pathological gambling,[90] Gulf War syndrome,[91] PTSD in abused children,[92] obsessive-compulsive disorder in children,[93] and seasonal affective disorder.[94]

Randomized controlled trials have provided strong empirical support for the efficacy of cognitive interventions, often as an adjunct to therapy as usual, in the treatment of a broad range of medical conditions including heart disease,[95] hypertension,[96] cancer,[97,98] headaches,[99,100] chronic pain,[101] chronic low back pain,[102] chronic fatigue syndrome,[103] rheumatoid arthritis,[104] premenstrual syndrome,[105] and irritable bowel syndrome.[106,107] Additional outcome studies have documented the beneficial role of CT in reducing depression and improving overall quality of life in patients diagnosed as having various medical disorders.[108]

## APPLICATION OF CT/CBT
## TO REAL-LIFE CIRCUMSTANCES

Only a few effectiveness studies of CT/CBT have been conducted. Persons et al[109] reviewed outcomes for a series of unselected patients treated at a clinic and found improvements comparable to those reported in randomized controlled trials. Stirman et al[110] similarly found that clinical characteristics of participants in randomized controlled trials matched those of patients presenting for treatment in clinical settings. Morrison et al[85] demonstrated improvements in the symptoms of schizophrenia with adjunctive CT with pharmacotherapy in a National Health Service clinic.

A comprehensive evaluation analyzed the utility and cost-effectiveness of CBT in community settings.[111-113] Evidence-based care consisting of either CBT or ADM resulted in a significantly greater improvement in depression for minority and nonminority patients than for those receiving usual care. Improvement in employment, however, was found only in the nonminority group. A 5-year follow-up of patients who received evidence-based CT or ADM revealed a significant reduction in the percent-

age of participants with probable depressive disorder, as compared with usual care.[113]

Several studies have demonstrated interesting neuropsychological correlates of the dysfunctional thinking and beliefs in depression[114,115] as well as the neurophysiological changes associated with CT for depression.[116] Of empirical interest is the severely dysfunctional negative beliefs related to severe loss of extracellular serotonin; studies of this nature can greatly expand our understanding of the mind-brain relationship.[115]

Ways of decreasing the cost of therapy are being explored. Wright et al,[117] for example, found that computer-assisted CT significantly reduced therapy time and was as effective as standard CT for depression.

## FUTURE DIRECTIONS

Several official reports in the United Kingdom and the United States have recommended the use of CT/CBT for a variety of the common psychiatric disorders. In the guidelines of the National Institute for Clinical Excellence (NICE) of the British Health Service, CBT has been recommended as a treatment of choice for mild depression, as an option for moderate depression, and in combination with ADM for severe depression.[118] The NICE guidelines also recommend CBT for generalized anxiety disorder as a first option because of its long-term effectiveness,[119] panic disorder,[119] PTSD,[120] and schizophrenia.[121] Dissemination of these therapies in clinical practice has been a problem because of the relative scarcity of therapists trained in these modalities. At the present time, training in the evidence-based therapies has been mandated by the Accreditation Council for Graduate Medical Education in psychiatric training programs in the United States.[122,123] However, many of the training programs lack trained cognitive therapists to provide supervision to the residents. Clinical psychology internships, in contrast, are not required to provide practical training in evidence-based therapies, and only a minority do so. To "export" the large body of research findings into real-world settings, it would be necessary to provide an extensive infrastructure for the delivery of evidence-based therapies as well as for training professionals in these therapies.

When the pool of professionals trained in evidence-based therapies reaches an adequate level, it may be feasible to consider an algorithm that takes into account the patient's needs and the necessary structures for meeting those needs in the most cost-effective way. One possible plan would be to institute a triage system encompassing hierarchical levels of specialization. The milder, less resistant problems (eg, uncomplicated depression, anxiety, or panic attacks) could be assigned to master's-level health care professionals or nurse practitioners for either psychotherapy or ADM, as in the Quality Improvement Program described by Wells et al.[124] The next level of severity (more complex Axis I disorders with or without comorbid Axis II) would receive ADM or psychotherapy alone or preferably the combination from highly experienced professionals. The most challenging disorders, including severe depression, bipolar disorder, schizophrenia, and borderline personality disorder would be treated by highly skilled specialists in those specific dis-orders. To meet these needs will, of course, require a significant expansion of training programs in the evidence-based therapies as well as appropriate modifications for various demographic groups. An integrated plan for the dissemination of the evidence-based therapies has been proposed[125] and would enhance their acceptability to both health care professionals and the consumers of mental health services. These suggestions may seem visionary at this time given the economic restraints and limitations in training, but the enormous strides made in developing and refining both pharmacotherapy and psychotherapy during the past 4 decades provide hope that similar progress can be made in optimizing the delivery of these treatments in a cost-effective way.

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