

Toward a Unified Treatment for Emotional Disorders

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Over 40 years of development of cognitive behavioral approaches to treating anxiety and related emotional disorders have left us with highly efficacious treatments that are increasingly widely accepted. Nevertheless, these manualized protocols have become numerous and somewhat complex, restricting effective training and dissemination. Deepening understanding of the nature of emotional disorders reveals that commonalities in etiology and latent structure among these disorders supercedes differences. This suggests the possibility of distilling a set of psychological procedures that would comprise a unified intervention for emotional disorders. Based on theory and data emerging from the fields of learning, emotional development and regulation, and cognitive science, we identify three fundamental therapeutic components relevant to the treatment of emotional disorders generally. These three components include (a) altering antecedent cognitive reappraisals; (b) preventing emotional avoidance; and (c) facilitating action tendencies not associated with the emotion that is dysregulated. This treatment takes place in the context of provoking emotional expression (emotional exposure) through situational, internal, and somatic (interoceptive cues), as well as through standard mood-induction exercises, and differs from patient to patient only in the situational cues and exercises utilized. Theory and rationale supporting this new approach are described along with some preliminary experience with the protocol. This unified treatment may represent a more efficient and possibly a more effective strategy in treating emotional disorders, pending further evaluation.

EDITOR'S NOTE: Dr. Barlow was invited to contribute an article about his research program on the occasion of his receiving an award for Outstanding Contribution by an Individual for Research Activities at the 2001 convention of the Association for Advancement of Behavior Therapy. The following article, by Barlow, Allen, and Choate, was received in response to this request. It underwent a modified editorial review process, similar to the one used for AABT presidential address articles. The Editor and Associate Editors congratulate Dr. Barlow on his richly deserved award.

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In the 1960s, cognitive behavioral approaches to treating emotional disorders such as anxiety and mood disorders began to emanate from basic psychological science; specifically, theories and data pertaining to learning, emotional development and regulation and, somewhat later, cognitive science. To justify a relatively radical new and unified psychological approach to treating emotional disorders, it is important to provide some background.

In the late 1950s and early 1960s, treatments began to deviate from a common general psychotherapeutic approach by directly targeting specific psychopathology, such as phobias. These treatments represented the beginnings of behavior therapy. This trend is probably best represented by Wolpe's systematic desensitization, as well as the development of situational exposure for phobic behavior (Agras, Leitenberg, & Barlow, 1968; Marks, 1971; Wolpe, 1958). Wolpe's development of systematic desensitization intrigued many, particularly those who foresaw the promise of translational research from basic behavioral science to the clinic. Furthermore, his descriptions of systematic desensitization, operationalized as they were, provided a large boost to attempts to demonstrate its efficacy empirically. This allowed investigators to begin the task of refocusing psychotherapy research from an emphasis largely on process to one on outcomes (Barlow & Hersen, 1984; Hersen & Barlow, 1976). Unfortunately, systematic desensitization had only limited efficacy in the clinic (in contrast to its success with college sophomores with snake fears) and then only for some types of specific phobia. Attempts to apply systematic desensitization to more complex clinical conditions such as agoraphobia were unsuccessful (Barlow, 1988, 1994).

In the mid-1960s, we began experimenting with some different therapeutic strategies in which we encouraged individuals with what we would now call panic disorder with agoraphobia (PDA) to expose themselves to real-life frightening situations (e.g., Agras et al., 1968). At the same time, Isaac Marks in London was experimenting with similar procedures (e.g., Marks, 1971). This approach was innovative at the time because conventional wisdom held that experiencing anything more than small doses of anxiety might result in some harm to the patient, an idea based on prevailing theories. Gradually, through the 1970s, programmatic research revealed that situational exposure did not require the various trappings that were then associated with it (e.g., relaxation, contingent social reinforcement) and that it could be implemented relatively rapidly.

By the 1980s, it was clear that we had an effective treatment for phobic behavior, but it was at best a blunt instrument. Best estimates of outcomes by the 1980s suggested that 60% to 75% of people receiving exposure-based treatments for PDA showed some clinical benefit. However, in a review of 24 studies, it was clear that as many as 35% of those entering treatment received little or no benefit (Jansson & Öst, 1982). Furthermore, of those receiving some benefit, only 10% to 20% could be said to approach normal functioning. The remainder continued to suffer from anxiety, panic, and residual avoidance.

During this period of time, several important developments occurred in the treatment of anxiety and related emotional disorders. First, investigators began to focus on mechanisms of action and theories of behavior change. In considering exposure procedures, it was clear that “exposure” was simply a dry theoretical description of a process, with no heuristic value. Various theories of fear reduction began to be investigated, including habituation, extinction, and more cognitively based theories such as changes in self-efficacy (Bandura, 1977), modifying cognitive schemas (Beck et al., 1985), and emotional processing accounts (Foa & Kozak, 1986; Lang, 1979; Rachman, 1980). Second, we learned a great deal about the nature of emotional disorders from ongoing studies of psychopathology. These theories of behavior change and increased knowledge of psychopathology led directly to new interventions. In addition to exposure-based procedures, cognitive therapy, first developed to treat depression, became a staple of treatments for anxiety disorders (Beck, 1972; Beck et al., 1985). In addition to situational exposure, we developed interoceptive exposure initially targeting PDA, recognizing that the context of anxiety and fear was internal as well as external (Barlow, 1988). Yet another important development was the beginnings of research on outcomes of individual cognitive behavioral therapy (CBT) protocols, targeting specific anxiety (or other) disorders (e.g., Barlow, Hayes, & Nelson, 1984). An important consequence was the growing realization that meaningful research on outcomes required the generation of detailed individual therapeutic manuals so that subsequent clinical research efforts could attempt to replicate the therapeutic procedures. As a result, psychological treatments were increasingly characterized by individual protocols that contained specific strategies such as cognitive restructuring, coping skills, and, where necessary, situational and interoceptive exposure procedures targeted to specific forms of psychopathology. These treatments were then tested empirically in a variety of formats, uses, and settings.

Current Status of Treatment

Early evidence on the efficacy of these CBT protocols led directly to large-scale clinical trials, often conducted across several sites in order to include a large *N* and control for allegiance effects. For example, one large clinical trial tested the effectiveness of CBT, medication, and their combination as treatments for panic disorder (Barlow, Gorman, Shear, & Woods, 2000). A second study tested the effectiveness of cognitive behavioral group therapy (CBGT) compared to medication and several placebo groups, including a psychological placebo, for social phobia (Heimberg et al., 1998; Liebowitz et al., 1999). A third study looked at the separate and combined effects of a psychological treatment, medication, and their combination for chronic major depressive disorder (MDD; Keller et al., 2000). In this large study, patients received either nefazodone, a CBT constructed specifically for chronically depressed patients, or their combination. In yet another large, recently completed multi-

center trial, the effects of clomipramine, intensive behavior therapy consisting of exposure and response prevention, and a combination were compared for the treatment of obsessive-compulsive disorder (OCD; Kozak, Liebowitz, & Foa, 2000).

It is not our intention within the scope of this review to provide detailed outcomes from each individual protocol for specific disorders. Nevertheless, some general conclusions flow from the reports mentioned above and other studies, at least for adults (Barlow, 2001; Nathan & Gorman, 2002). Approximately 50% to 80% of patients undergoing treatment for one or more of the emotional disorders achieve "responder" status, with the definition of "responder" necessarily differing somewhat from study to study. In most of these cases, the individual has made a clinically significant improvement, although they may not be "cured" (symptom-free). These outcomes are typically better than a credible alternative psychological treatment or "placebo" in every anxiety disorder, with information on this issue less certain for the mood disorders. Thus, these results indicate that "common factors" of positive expectancies, remoralization, and a strong therapeutic alliance, while contributory to outcomes, are substantially enhanced by the addition of psychological treatments, at least for the anxiety disorders (Barlow, 2001; Nathan & Gorman, 2002). Finally, it seems clear from the multisite studies mentioned above that psychological and pharmacological treatments achieve approximately equal efficacy immediately after treatment is concluded (except possibly for OCD, where the psychological treatment seems more efficacious), but that psychological treatments are more enduring after treatment is discontinued. The evidence also suggests that, in those disorders where the question has been evaluated, simultaneously combining drug and psychological treatments does not confer a substantial advantage, with the possible exception of MDD. Sequential combinations of treatments, on the other hand, are more promising (Barlow, 2002; Nathan & Gorman, 2002).

Nevertheless, a number of significant limitations to current treatments exist. Obviously, there are still a considerable number of patients who do not respond well to this type of remedy, and the reasons for their lack of response are not yet known. Thus, although treatment is effective for many people, there is plenty of room for improvement. Another problem that has become apparent with manualized treatments is that there are simply too many of them. Clinicians must use separate handbooks, workbooks, and protocols for each disorder. Not only can this be quite costly, but it can take a significant amount of training to become adequately familiar with each of the distinct protocols. Finally, because the protocols are somewhat complex, dissemination of treatment to providers becomes an obstacle (Barlow, Levitt, & Bufka, 1999). For example, in the area of depression, a recent NIMH task force specified as a priority for treatment development the need for more "user-friendly" protocols (Hollon et al., 2002). Unless these treatments become more "user-friendly" as recommended, it is unlikely that most nonresearch

clinicians will have a sufficient understanding of, or access to, these empirically supported techniques for the emotional disorders.

The Nature of Emotional Disorders and “Negative Affect Syndrome”

One argument for a unified treatment approach to emotional disorders is the facilitation of dissemination and training focused on a single set of therapeutic principles rather than diverse protocols. A second, more fundamental argument concerns emerging research and theory based on conceptions of the major emotional disorders that emphasize their commonalities rather than their differences. These arguments point to major developments in the areas of phenomenology and nosology, with a particular focus on comorbidity, all suggesting considerable overlap among disorders. Additionally, the observed effects of current psychological treatments on comorbid conditions and the nonspecificity of treatment response support this overlap. Equally important is emerging research on the latent structure of dimensional features of emotional disorders. Finally, a body of evidence supports commonalities in the etiology of emotional disorders, which has been summarized recently in the form of a new model referred to as “triple vulnerabilities” (Barlow, 1991, 2000, 2002). Each of these will be briefly reviewed in turn. For purposes here, the focus will be on anxiety and unipolar mood disorders (MDD and dysthymia). However, we envision that the principles elucidated may be applicable more broadly to psychopathology in which negative affect plays a functional role, including bipolar, somatoform, dissociative, and anger-related disorders, as well as eating disorders.

Overlap Among Disorders

Currently the evidence strongly suggests considerable overlap among the various anxiety and mood disorders. At the diagnostic level this is most evident in the high rates of current and lifetime comorbidity (e.g., Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Kessler et al., 1996, 1998). We have collected data on the percentages of additional diagnoses in patients who have been diagnosed with a principal anxiety or mood disorder (Brown et al., 2001). These data were derived from a large sample: 1,127 patients who were carefully diagnosed with the Anxiety Disorders Interview Schedule for *DSM-IV* — Lifetime Version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994). As noted in that article, these summaries are most likely conservative due to limits in generalizability such as the nature of inclusion-exclusion criteria used. Overall, results indicate that 55% of patients with a principal anxiety or mood disorder had at least one additional anxiety or depressive disorder at the time of assessment, and this rate increases to 76% when additional diagnoses occurring at any time during the patient’s life, including currently (i.e., lifetime diagnoses), are considered. To take one example, of patients diagnosed with PDA, 60% out of 324 patients were determined to meet criteria

for an additional anxiety or mood disorder, breaking down to 47% with an additional anxiety disorder and 33% with an additional mood disorder. When lifetime diagnoses are considered, the percentages rise to 77% experiencing any anxiety or mood disorder, breaking down to 56% for any anxiety disorder and 60% for any mood disorder. The principal diagnostic categories of post-traumatic stress disorder (PTSD), MDD, dysthymia, and generalized anxiety disorder (GAD) were associated with the highest comorbidity rates. For specific patterns of comorbidity associated with each diagnoses, see Brown et al. (2001).

Further evidence of the conservative nature of this estimate is present in findings on the comorbidity of GAD and mood disorders. This is due to hierarchical exclusions remaining in the *DSM-IV*. For instance, when adhering strictly to *DSM-IV* diagnostic rules, the comorbidity of dysthymia and GAD was 5%. However, when the hierarchical rule that GAD should not be assigned when occurring exclusively during a course of a mood disorder was suspended, the comorbidity estimate increases to 90%. These data also ignore the presence of subthreshold symptoms that did not meet diagnostic thresholds for one disorder or another.

There are several possible explanations for these high rates of comorbidity that we have reviewed extensively elsewhere (Brown & Barlow, 2002). Among these are trivial problems with overlapping definitional criteria; artifactual reasons, such as differential base rates of occurrence in our setting; and the possibility that disorders are sequentially related and that the features of one disorder act as risk factors for another disorder. For example, depression seems to follow PDA, and panic disorder seems to follow PTSD. But another more intriguing explanation is that this pattern of comorbidity argues for the existence of what has been called a "general neurotic syndrome" (Andrews, 1990, 1996; Tyrer, 1989). Under this conceptualization, heterogeneity in the expression of emotional disorder symptoms (e.g., individual differences in the prominence of social anxiety, panic attacks, anhedonia, etc.) is regarded as trivial variation in the manifestation of a broader syndrome. This, in turn, is consistent with models we have developed that anxiety and mood disorders emerge from shared psychosocial and biological/genetic diatheses. If this is the case, then a unified treatment protocol cutting across current diagnostic categories to address core features of the emotional disorders could be a more parsimonious, and, perhaps, powerful option.

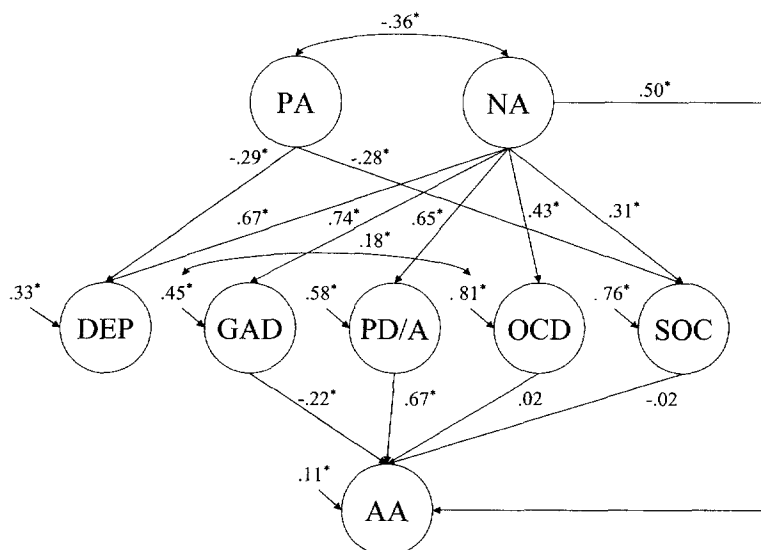
The Effects of Psychological Treatments on Comorbid Disorders, and Nonspecificity of Treatment Response

Other findings supporting our contention of a "general neurotic syndrome" or "negative affect syndrome" (NAS), as we prefer to call it, include the observation that psychological treatments for a given anxiety disorder produce significant improvement in additional comorbid anxiety or mood disorders that are not specifically addressed in treatment (Borkovec, Abel, & Newman, 1995; Brown, Antony, & Barlow, 1995). For example, we examined the course of additional diagnoses in a sample of 126 patients who were being treated

for PDA at our center. At pretreatment, 26% had an additional diagnosis of GAD, but the rate of comorbid GAD declined significantly at posttreatment to 9%, and remained at this level at a 2-year follow-up. Whether this represents the generalization of elements of treatment to independent facets of both disorders, or a way of effectively addressing "core" features of emotional disorders, is not significant to our purpose here. In both cases, the efficiency of a unified treatment protocol is suggested. The fact that a wide range of emotional disorders (e.g., MDD, dysthymia, OCD, and PDA) respond approximately equivalently to antidepressant medications has also been interpreted as indicating a shared pathophysiology among these symptoms (e.g., Hudson & Pope, 1990). Also, Tyrer et al. (1988) treated 210 outpatients with GAD, PDA, or dysthymia with either drug, placebo, CBT, or a self-help program. Although some differences were noted at posttreatment as a function of treatment condition (e.g., some drugs were less effective than other drugs and psychological treatments), no diagnostic group differences were observed. This suggested to Tyrer et al. (1988) that the differential diagnosis of anxiety and mood disorders does not provide a sound basis for treatment prescription. It should be noted, of course, that these data are only suggestive, because the current generation of more powerful psychological treatments was not utilized. But it does appear to offer support for the above conceptions.

Latent Structure of the Emotional Disorders

There is wide agreement that *DSM-IV* represents the zenith of a "splitting approach" to nosology, with the obtained advantage of high rates of diagnostic reliability. But, there is growing suspicion that this achievement has come at the expense of diagnostic validity, and that the current system, as suggested above, may be erroneously distinguishing categories that are minor variations of broader underlying syndromes. This would not imply a return to a non-empirical system of classification based on theories of etiology. Rather, this thinking points to a quantitative approach using structural equation modeling to examine the full range of anxiety and mood disorders without the constraints of artificial categories, given their strong relationship and potential overlap (Brown, Chorpita, & Barlow, 1998; Chorpita, Albano, & Barlow, 1998; Clark & Watson, 1991; Watson, Clark, & Harkness, 1994). We have been studying this question for the last 10 years (e.g., Brown et al., 1998; Zinbarg & Barlow, 1996) and have confirmed, with some modifications, the tripartite model of emotional disorders first proposed by Clark and Watson (1991). Some of our findings are presented in Figure 1. One of the intriguing and important findings from this line of research is that mood disorders show greater overlap with certain anxiety disorders such as GAD than do other anxiety disorders, supporting and reinforcing the commonalities of depression and anxiety at a phenomenological level (Brown et al., 1998; Clark, Steer, & Beck, 1994; Mineka, Watson, & Clark, 1998). The findings from Brown et al. (1998) using a sample of 350 patients with *DSM-IV* anxiety and mood disorders confirmed a hierarchical structure. In this structure, negative affect and



PA, positive affect; NA, negative affect; DEP, mood disorders; GAD, generalized anxiety disorder; PD/A, panic disorder with/without agoraphobia; OCD, obsessive-compulsive disorder; SOC, social phobia; AA, automatic arousal. $*p < .01$.

FIG. 1. Structural model of interrelationships of *DSM-IV* disorder constructs and negative affect, positive affect, and automatic arousal. From "Structural Relationships Among Dimensions of the *DSM-IV* Anxiety and Mood Disorders and Dimensions of Negative Affect, Positive Affect, and Autonomic Arousal," by T. A. Brown, B. F. Chorpita, & D. H. Barlow, 1998, *Journal of Abnormal Psychology*, 107, pp. 179–192. Copyright 1998 by the American Psychological Association. Reprinted with permission.

positive affect emerged as higher-order factors to the *DSM-IV* disorder factors, with significant paths from negative affect to each of the five *DSM-IV* factors, and significant paths from positive affect to the mood disorders and social phobia factor only. In this model, automatic arousal, which we consider to represent the phenomenon of panic, emerges as a lower-order factor with significant paths from PDA and GAD (where the relationship was negative). These findings indicate that the "key features" of the *DSM* anxiety and mood disorders cannot be collapsed indiscriminately into an NAS. But it seems safe to conclude that what is common outweighs what is not (*DSM-IV* factors). Our view, then, is that *DSM-IV* emotional disorder categories do not qualify in any sense as real entities (Kendell, 1975) but do seem to be useful concepts or constructs that emerge as "blips" on a general background of NAS. It also appears increasingly likely for a variety of reasons, including the findings from genetics (e.g., Kendler, 1996; Kendler, Heath, Martin, & Eaves, 1987), that *DSM-V* will turn to a more dimensional description of these phenomena that would reinforce conceptions of common underlying components (Kupfer, First, & Regier, 2002).

Etiology

Elsewhere, we have elaborated on an interacting set of vulnerabilities or diatheses relevant to the development of anxiety, anxiety disorders, and related emotional disorders. This “triple vulnerabilities” theory encompasses a generalized biological vulnerability, a generalized psychological vulnerability, and a specific psychological vulnerability emerging from early learning (Barlow, 2000, 2002). A generalized biological vulnerability involves nonspecific genetic contributions to the development of anxiety and negative affect. Much of the research on this generalized biological vulnerability has focused on temperaments labeled “anxiety,” “neuroticism,” “negative affect,” or “behavioral inhibition.” Although the relationships among these closely related traits and temperaments have yet to be fully worked out, it is likely that each partially represents a common theme associated with a biological vulnerability to develop emotional disorders generally (Barlow, 2000, 2002). Additionally, early life experiences under certain conditions contribute to a generalized psychological vulnerability or diathesis to experience anxiety and related negative affective states (Chorpita & Barlow, 1998). It is this set of experiences that produces a sense of uncontrollability that seems to be at the core of negative affect and derivative states of anxiety and depression. If these two vulnerabilities happen to line up, and are potentiated by the influence of life stress, the likely result are the clinical syndromes of GAD and/or depressive disorders as outlined in Figure 2. Notice that false alarms (panic attacks) may occur as a function of stressful life events, facilitated by high levels of baseline anxiety, and emerging as a function of these synergistic generalized vulnerabilities. But these false alarms are not in themselves necessarily implicated in a clinical disorder. For that to occur, an additional layer of a more specific psychological vulnerability must be considered. In this conception, certain learning experiences seem to focus anxiety on specific life circumstances; that is,

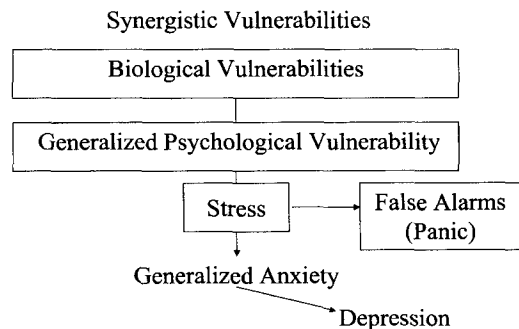


FIG. 2. Diathesis-stress model of the development of generalized anxiety and depression. From “Unraveling the Mysteries of Anxiety and its Disorders From the Perspective of Emotion Theory,” by D. H. Barlow, 2000, *American Psychologist*, 55, pp. 1247–1263. Copyright 2000 by the American Psychological Association. Reprinted with permission.

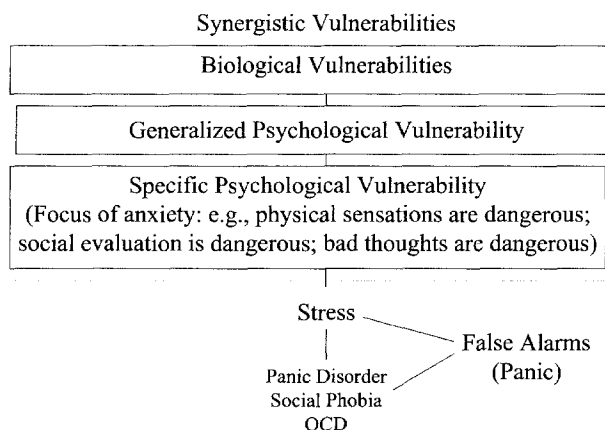


FIG. 3. Triple vulnerabilities model in the development of certain anxiety disorders. From "Unraveling the Mysteries of Anxiety and its Disorders From the Perspective of Emotion Theory," by D. H. Barlow, 2000, *American Psychologist*, 55, pp. 1247–1263. Copyright 2000 by the American Psychological Association. Reprinted with permission.

these circumstances or events become imbued with a heightened sense of threat or danger. For example, specific early learning experiences seem to determine whether individuals may view somatic sensations, intrusive thoughts, or social evaluation as specifically dangerous (Barlow, 2002; Bouton, Mineka, & Barlow, 2001). It is this specific psychological vulnerability that, when coordinated with the generalized biological and psychological vulnerabilities mentioned above, seems to contribute to the development of discrete anxiety disorders such as social phobia, OCD, panic disorder, and specific phobias, as represented in Figure 3. Evidence for this model has been reviewed in detail elsewhere (Barlow, 2000, 2002; Bouton et al., 2001; Chorpita & Barlow, 1998). While future research will determine the validity of this model, it is consistent with the emerging phenomenological evidence reviewed above on the overriding importance of common factors in the genesis and presentation of emotional disorders.

Implications for Treatment

Growing evidence on the unifying principles of emotional disorders (Barlow, 1991, 2002) with a focus on common underlying mechanisms suggests the possibility of distilling a set of psychological procedures that would comprise a unified intervention for emotional disorders. In 1988 one of us proposed, following the emotion theorists (e.g., Izard, 1971), that a coherent and consistent therapeutic approach to emotional disorders would ultimately be based on emotion theory and evolving knowledge of the modification of emotional states. Following Lang (1968), Rachman (1981), and Wilson (1982), whose

TABLE 1
COMPONENTS OF ANY AFFECTIVE THERAPY

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- A. Essential targets for change
 - 1. Action tendencies
 - 2. A sense of uncontrollability-unpredictability
 - 3. Self-focused attention
 - B. Helpful but not essential targets for change
 - 1. "Hot" apprehensive cognitions
 - 2. Hypervalent cognitive schemata and attention narrowing
 - 3. Coping skills and social support
 - 4. Elevated physiological responding and altered neurobiological functions
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Note. From *Anxiety and Its Disorders: The Nature and Treatment of Anxiety and Panic*, by D. H. Barlow, 1988, New York: The Guilford Press. Copyright 1988 by The Guilford Press. Reprinted by permission.

early speculations were influential, components of any affective therapy were outlined (see Table 1). Based on theoretical and empirical work at that time, and a lineage of knowledge dating back to Darwin (1872), there seems no quicker or more powerful way to change emotional expression than to modify action tendencies associated with a specific emotion. Other essential targets for change included increasing a fundamental sense of controllability and predictability over events in one's environment, and decreasing the powerful avoidant strategy of focusing attention on non-task-related consequences of excessive emotional activity (neurotic self-preoccupation). Other targets for change that were speculated as being insufficient but perhaps helpful in as much as they facilitated change in the essential targets included focusing directly on emotional cognitions, coping skills, social support networks, and heightened arousal. Evidence for these assertions was reviewed in Barlow (1988).

Development of these ideas was, for the most part, put aside during the 1990s as we concentrated on *DSM-IV*, large clinical trials, and other tasks, but these ideas were not entirely ignored by others. For example, Marsha Linehan brought the concept of modifying action tendencies (opposite action tendencies) to good use in her development of dialectical behavior therapy (DBT; Linehan, 1993). For the past 18 months, we have turned our attention once again to these fundamental themes, taking advantage of substantial progress in a number of related areas over the past decade. This progress has occurred in the context of developments in modern learning theory and cognitive neuroscience, as well as our greatly increased knowledge of naturally occurring processes in the regulation of emotional expression. We will briefly touch on developments in each of these areas.

Modern Learning Theory and Cognitive Neuroscience

In an article exploring theoretical conceptualizations of the etiology of anxiety disorders, Bouton et al. (2001) suggest that early panic attacks result

in conditioned associations between the attack and a variety of interoceptive and exteroceptive cues. When some of these cues are elicited in a nonpanic situation, a constellation of behavioral and physiological responses arise, which we collectively call "anxiety." As such, anxiety is a state of relatively low-level arousal preparing us for possible future danger. Panic, on the other hand, is associated with a surge in autonomic arousal enabling immediate action (fight-flight). These mechanisms are not necessarily mutually exclusive. In fact, modern learning theory suggests that anxiety may initiate panic because anxiety, too, can become a conditioned stimulus. It seems that small physiological, behavioral, and emotional changes can become associated with an extreme fear reaction (such as panic) with or without conscious knowledge of the cues. These conditioned events can begin to influence behavior at a subconscious level, such that strengthening of the association between physical and emotional cues and panic begins to occur (LeDoux, 1996). Brain imaging techniques have offered support for this interpretation, even indicating differences in the neurobiological bases of conscious and unconscious conditioning processes (Öhman, 1999). Future work in understanding the actual functional brain changes in emotional disorders continues, and researchers are now using imaging strategies to examine changes in brain function following cognitive-behavioral treatments for anxiety (e.g., Furmark et al., 2002). Thus, the future investigation of emotional disorders and their treatment will involve a comprehensive study of the psychological, emotional, and neurobiological correlates of emotion-based conditioning procedures. Work in this area has already influenced our knowledge of emotional cues from a variety of external and internal contexts that must be considered in treatment to maximize effects.

Emotion Regulation

A particularly important concept for understanding emotional disorders is that of emotion regulation (Brenner & Salovey, 1997; Mayer & Salovey, 1997). By this, we are referring to the strategies individuals use to influence the occurrence, experience, intensity, and expression of a wide range of emotions (Frijda, 1986; Masters, 1991; Richards & Gross, 2000). Emotion regulation and dysregulation seem to play an important role in emotional disorders (and other psychopathology), and levels of positive and negative emotions as well as their functional relationships often differ depending on the particular disorder (Gross & Levenson, 1997; Rottenberg & Gross, 2003; Thayer, 2000). Emotional disorders seem characterized to some degree by attempts to control both positive and negative emotions in a variety of contexts as outlined below. Individuals concerned about the expression and experience of their feelings may attempt to suppress, hide, or ignore them, with unintended consequences (Gross & Levenson, 1997; Pennebaker, 1997). This is because excessive attempts to control emotional experience lead to an increase in the very feelings the individuals are attempting to regulate, as demonstrated by attempts to control emotions after initial panic attacks (Craske,

Miller, Rotunda, & Barlow, 1990). Furthermore, the degree to which one attempts to control emotions is somewhat related to the degree of intensity to which an individual experiences negative emotions, which can quickly become overwhelming (Lynch, Robins, Morse, & Krause, 2001). These overpowering experiences often lead to attempts at thought suppression as a convenient and accessible way to reduce emotional responsiveness. It is this pattern that may erupt in a vicious cycle of increased physiological and emotional arousal, leading to more unsuccessful attempts at suppression, which in turn contributes to growing psychological distress. Thus, it is clear that future treatments for emotional disorders must focus on this issue and develop treatments specifically targeting emotion dysregulation.

A Unified Treatment

Based on theory and practice described above, over the past year we have distilled three fundamental therapeutic components that currently comprise our unified treatment approach to emotional disorders. Following a standard psychoeducational phase common to all psychotherapeutic approaches, these three components include (a) altering antecedent cognitive reappraisals — an intensive emotion-regulation procedure that directly facilitates the next two steps in treatment; (b) preventing emotional avoidance — a broad-based effort that goes well beyond traditional attempts to prevent behavioral avoidance in phobic disorders by targeting cognitive, behavioral, and somatic experiential avoidance; and (c) facilitating action tendencies not associated with the emotion that is disordered. This treatment takes place in the context of provoking emotional expression (emotion exposure) through situational, internal, and somatic (interoceptive) cues, as well as through standard mood-induction exercises, and differs from patient to patient only in the situational cues and exercises utilized. Notice also that “exposure” is not conceptualized as a mechanism of action. Rather, successfully provoking emotions is considered a setting condition in order to implement the essential treatment components. Of course, we recognize that the “mere-exposure” paradigm (Zajonc, 2001) has some emotion-regulating properties itself in terms of producing positive affect, even if the exposure is subliminal, most likely due to classical conditioning. These mechanisms may apply to emotion exposure as well.

Antecedent Cognitive Reappraisal

Since the 1970s, and under the enormous influence of cognitive therapy as innovated by Aaron T. Beck (Beck, 1972; Beck, Rush, Shaw, & Emery, 1979), clinicians have focused on the appraisals and judgments that individuals with emotional disorders make regarding external events, as well as their own efficacy in coping with these events. This approach was first developed in the context of depression. In this context Beck outlined the well-known “cognitive triad” in which individuals maintained negative beliefs about their own self, the world, and the future. Subsequently, this notion was extended to

anxiety disorders (Beck et al., 1985). It wasn't long before the importance of appraisals focused on internal events, such as physical sensations and emotions, began to be recognized. In panic disorder, this was first suggested by Goldstein and Chambless (1978), who outlined a "fear of fear" model of panic disorder. In this model, conscious negative appraisals of somatic and affective manifestations of the experience of "fear" as dangerous became an important focal point. Subsequently, the concept of interoceptive conditioning originally developed by Razran (1961) was applied to anxiety disorders to describe a relatively less conscious process by which internal cues, often generated by emotional activation, could trigger anxiety and panic, with the implication that direct exposure to emotion-associated internal somatic cues would be an important part of treatment (Barlow, 1988). The notion of appraising internal cognitive and emotional states in a negative way was extended to other anxiety disorders such as OCD (Steketee, 1993) and GAD (Craske, Rapee, Jackel, & Barlow, 1989; Wells et al., 1995).

Cognitive therapy focuses on evaluating the rationality of these negative appraisals and substituting more realistic evidence-based appraisals in their place. On one level, this may seem very much as an attempt to eliminate or suppress negative thoughts and immediately replace them with more adaptive or realistic appraisals, and it has been used in this way, as lucidly noted by Hayes, Strosahl, and Wilson (1999). But, in fact, this process can be conceptualized from an emotion-regulation perspective as altering antecedent reappraisals of threat and negativity. Support for this subtle but crucial reconceptualization emerges from the emotion-regulation literature, where evidence clearly exists that reappraisal of both internal and external threat and danger *before the fact* (that is, before heightened levels of negative emotion are provoked) has a salutary effect on the later expression of negative emotion, as noted above (Gross, 1998; Richards & Gross, 2000; Thayer, 2000). The importance of antecedent reappraisal versus more reactive strategies was first outlined in 1991 by Masters (1991). In a program of research, Gross (1998) has found that antecedent cognitive reappraisal does, in fact, reduce subjective experience of negative emotion.

For individuals with emotional disorders, we first demonstrated this phenomenon in 1989 (Sanderson, Rapee, & Barlow, 1989) in the context of manipulating antecedent appraisals of control over a threatening situation. In this experiment, patients with PDA were told that they would be able to control — in a CO₂ inhalation paradigm — the flow of CO₂ by turning a dial when a light was illuminated. For half of the patients the light was never illuminated, leading them to believe they had no control over the sensation. For the other half, the light did come on, indicating that the dial was operative and leading them to believe that they had some control over the situation. In fact, the dial had no bearing on CO₂ flow and thus provided only an illusion of control (perceived control). Nor did patients ever really attempt to use the dial.

Nevertheless, patients in the perceived control condition reported significantly fewer panic attacks and less emotion in general than those in the no-

control group, despite receiving the same amount of CO₂. More recently, Telch and colleagues have demonstrated this principle. For example, patients with specific phobias who were instructed to focus on their identified core threats and who received brief guidance on threat reappraisal evaluation did significantly better than those simply receiving exposure exercises without reappraisal (Kamphuis & Telch, 2000; Sloan & Telch, 2002). In our hands, we have adapted cognitive interventions to focus on two fundamental antecedent misappraisals: first, the probability of a negative event happening (probability overestimation), and second, the consequences of that negative event if it did happen (catastrophizing; Barlow & Craske, 2000; Craske, Barlow, & O'Leary, 1992). We have now extended these concepts to the full range of emotional disorders.

Now Hariri and colleagues (Hariri, Bookheimer, & Mazziotta, 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003), building on work by Damasio and colleagues (Bechara, Damasio, Damasio, & Lee, 1999; Damasio, 1994), have begun to elucidate the neural circuits underlying this process using functional magnetic resonance imaging (fMRI). They demonstrated that activation of the right prefrontal and anterior cingulate cortices during conscious evaluation and appraisal of emotional stimuli modulates and regulates bilateral amygdala responding. These findings also suggest that implementing antecedent cognitive reappraisal strategies is an important step in altering emotional responding.

Emotional Avoidance

There is also evidence that many emotional disorders are related to attempts to down-regulate (avoid) excessive unexpected emotional experiences. Examples of this process in the context of depression, anger, and excitement (mania), as well as fear, are provided in Barlow (2002). Preliminary data also exist on the prevalence of the occurrence of unexpected and sometimes distressing emotions in the nonclinical population (Craske, Brown, Meadows, & Barlow, 1995). Specifically, over 300 undergraduates were surveyed with a questionnaire to determine the prevalence of both cued and uncued panic attacks, anger outbursts, episodes of sadness, and surges of excitement, as well as the degree of worry or distress over the recurrence of each type of emotional experience. While fully 32% of the sample reported no uncued emotional experiences, the fact that 68% of this sample reported experiencing at least one uncued emotion in the previous 3-month interval was surprising. It is also noteworthy that while 10% of the population experienced uncued panic, which is consistent with other surveys, as many as 34% of the sample reported uncued depressive episodes, and many found these episodes very distressing. The fact that distress was associated with a substantial proportion of these unexpected episodes implies attempts to regulate these emotional experiences through suppression (although this was not specifically tested) with resulting consequences.

These results, of course, are consistent with observations of emotional

avoidance in other disorders. For example, Roemer, Litz, Orsillo, and Wagner (2001) reported that veterans with PTSD were more likely to report intentionally withholding their emotions, both positive and negative, than were veterans without PTSD. In a related study of rape-related PTSD, Orsillo, Roemer, and Litz (2001) found that women with PTSD described their sexual assault experiences with fewer fear words than did women without PTSD, although women with PTSD displayed higher levels of arousal. Evidence on the deleterious use of avoidant techniques extends to calming procedures so much a part of our own earlier protocols for treating anxiety and panic (Barlow & Cerny, 1988). Specifically, when calming techniques such as relaxation and breathing control are conceptualized to the patient as a *specific strategy for reducing negative emotions and distress*, in which the focus is to "cope" with the emotions and distress (rather than as a noncontingent calming exercise), the results seem counterproductive. For example, Schmidt et al. (2000) concluded that breathing retraining did not add any clear benefits to a treatment package consisting of education, cognitive restructuring, and exposure-based techniques for patients with panic disorder. In fact, a trend in the data indicated that patients who received breathing retraining showed lower end-state functioning on both self-report and clinician-rated measures. Similar results have been obtained from our prior work evaluating distraction strategies (Craske, Street, & Barlow, 1989; Craske, Street, Jayaraman, & Barlow, 1991; Kamphuis & Telch, 2000). And, of course, the deleterious effects of down-regulating (avoiding) emotion through reliance on talismen or safety signals have been conclusively demonstrated (Salkovskis, Clark, Hackman, Wells, & Gelder, 1999; Sloan & Telch, 2002; Wells et al., 1995). Finally, laboratory studies are directly demonstrating the effects of avoiding or suppressing emotion. For example, Feldner, Zvolensky, Eifert, and Spira (2003) divided non-clinical subjects into high or low emotional avoiders and subjected them to 4 breaths of 20% CO₂-enriched air. Half in each group were instructed to inhibit negative emotional reactions, the other half to simply observe their emotional response. High emotional avoiders reported greater distress and anxiety, whether suppressing or not, compared to low avoiders. In our laboratory, Levitt, Brown, Orsillo, and Barlow (in press) divided 60 patients with PDA into three groups, each of whom listened to a 10-minute audiotape describing one of two emotion-regulation strategies (acceptance or suppression) or a neutral narrative. Patients then underwent a 15-minute 5.5% CO₂ challenge. Following this challenge, they were asked to participate in a second challenge. The acceptance group was significantly less anxious and less avoidant than either the suppression or control groups in terms of subjective anxiety during the CO₂ challenge and willingness to participate in a second challenge.

Now we have also developed some direct evidence of the maladaptive use of emotion-regulation strategies in patients with a wide range of emotional disorders (Campbell-Sills, Barlow, Brown, & Hofmann, 2003). In this study, 60 patients who met diagnostic criteria for an anxiety or mood disorder and 30 individuals with no history of emotional disorders experienced an induction

of a negative emotion by watching an emotional film. In Study 1, the spontaneous emotion appraisals and emotional-regulation strategies were observed in both the clinical sample and the control sample. The patients in the clinical sample reported significantly different emotional appraisals and emotional-regulation strategies than nonclinical participants. Consistent with data reported above, clinical participants reported greater anxiety focused on the occurrence of emotions, as well as less emotional clarity. They also endorsed more reliance on maladaptive emotion-regulation strategies (e.g., suppression, cognitive rehearsal). The patients also rated their resulting emotions as less acceptable and engaged in more emotional suppression. Higher levels of suppression were associated, in turn, with elevated heart rate during the emotion induction, as well as inhibited recovery from subjective distress, skin conductance, and finger temperature changes after the induction.

In the second study, patients were instructed to engage in either emotion suppression activities during the emotional induction exercise, or emotion acceptance activities. Suppression participants failed to recover from subjective distress after the induction, and they manifested a different heart rate pattern than acceptance participants. Specifically, when patients were instructed to suppress their emotions, once again heart rate actually increased from anticipation to termination of the film, while heart rate in the acceptance group decreased during this period. Thus, patients with emotional disorders endorsed more negative emotion appraisals and utilized counterproductive emotion-regulation strategies compared to individuals without disorders.

Modifying Emotional Action Tendencies

As Izard pointed out in 1971, theories and evidence from emotion theory indicate that "the most efficient and generalized principles and techniques for emotion control [are] focused on the neuromuscular component of emotion . . . striate muscle action can initiate, amplify, attenuate, or inhibit an emotion" (p. 415). In other words, "the individual learns to act his way into a new way of feeling" (p. 410). As I suggested in 1988, it is possible that the crucial function of exposure in the treatment of phobic disorders is to prevent the action tendencies associated with fear and anxiety and facilitate different action tendencies (Barlow, 1988). For example, Fridlund, Hatfield, Cottam, and Fowler (1986) determined that physiological elevation during anxiety did not represent generalized arousal, but rather specific action tendencies associated with anxiety. I also speculated (Barlow, 1988) that it is possible that attention to action tendencies forms an important part of the treatment of other emotional disorders. For example, Beck, Rush, Shaw, and Emery (1979) and others (e.g., Lewinsohn & Lee, 1981) spent a considerable amount of time countering the tendencies of their depressed patients to behave in a "passive, retarded, and apathetic manner" (p. 312). More recently, behavioral activation has become a central and defining principle in some new treatments for depression that show considerable promise (Jacobson, Martell, & Dimidjian, 2001).

In fact, these strategies have a long history. Laughter, humor, and associated facial expression induced during successful paradoxical intention techniques (Frankl, 1960), a technique successfully used to counteract fear and anxiety in previous decades (e.g., Ascher, 1980), may be effective not because of the induction of cognitive changes, as was often assumed, but rather because of the prevention of behavioral responses, including facial expressions, and the substitution of action tendencies associated with alternative emotions. As noted above, Linehan (1993) has adapted this strategy creatively and with good effect to patients with borderline personality disorder (which, with its core feature of the avoidance and intolerability of negative affect, may be fundamentally a severe emotional disorder). Also Hayes, in his creative conceptualization of acceptance and commitment therapy (ACT; Hayes et al., 1999), has underscored the importance of encouraging action as an alternative coping strategy, with the purpose of instituting a sense of control as opposed to focusing on decreasing unwanted internal events.

Applications to Emotional Disorders

As reviewed above, it is our contention that these three basic therapeutic principles can be applied, with relatively minor modifications, to each of the emotional disorders. Antecedent cognitive reappraisal in each of the emotional disorders is easily categorized into overestimating the probability of a negative event happening (probability overestimation) and exaggerating the consequences of that negative effect if it did happen (catastrophizing). These concepts are relatively well-known by cognitive behavioral therapists and need no further elaboration as applied to individual disorders. Tables 2 and 3 provide some examples of both the general implementation of strategies for preventing emotional avoidance and modifying action tendencies across emotional disorders, as well as some specific adaptations of these strategies that seem useful when dealing with individual emotional disorders.

We have reviewed evidence above on the variety of cognitive and behavioral avoidance strategies—including cognitive rituals, distraction, emotion suppression, etc., as noted in Table 2—that cut across emotional disorders. Providing examples across some specific emotional disorders, it is clear that the lives of individuals with PDA revolve around avoiding intense emotions, particularly fear, and associated somatic sensations. This also extends to contexts or situations that produce strong emotional and somatic responding. We have referred elsewhere to this pattern of avoidance as “interoceptive avoidance,” and have developed a questionnaire to assess the extent of its presence (Brown, White, Forsyth, & Barlow, 2004; Rapee, Craske, & Barlow, 1994/1995). Cognitive and behavioral rituals have long been recognized as avoidant strategies in OCD. More recently, we have recognized similar features associated with GAD in the form of “worry behaviors,” which are fundamentally checking rituals (Craske et al., 1992), as well as the behavioral pattern of “perfectionism,” particularly negative/maladaptive perfectionism. This behavioral

TABLE 2
PREVENTING AVOIDANCE

General	Specific		
Safety Signals	Water bottles	PDA	Avoidance of: somatic sensations— “emotional” experiences
	Cell phones	Social phobia	Avoidance of: performance—interactions
	Medications	Specific phobia	Avoidance of: object-situation
Cognitive rituals		OCD	Behavioral rituals
Distraction		GAD	Perfectionism, worry-worry behaviors
Rationalization		PTSD	Avoidance of: strong affect—trauma cues
Emotion suppression		MDD	Withdrawal
Worry-rumination-rehearsal			

Note. PDA = panic disorder with agoraphobia; OCD = obsessive-compulsive disorder; GAD = generalized anxiety disorder; PTSD = posttraumatic stress disorder; MDD = major depressive disorder.

pattern seems to reflect a tendency to impose control over perceived uncontrollable daily events in one's life, thereby reducing distress and negative affect (Frost, Heimberg, Holt, Mattia, & Neubauer, 1993; Scott & Cervone, 2002). Similarly, for MDD, behavioral tendencies toward withdrawal seem clearly associated with avoiding interactions and contexts that may provoke negative affect.

In modifying the behavioral action tendencies driven by fundamental emotions, the first step is to provoke the emotions in so far as possible, usually through emotion-inducing exposure-based procedures (see Table 3). Adopting strategies that encourage experience of the emotion without engaging in the associated action tendencies (accepting the emotion) is a very basic strategy

TABLE 3
MODIFYING ACTION TENDENCIES

General	Specific
Emotion exposure	GAD: emotional arousal
Emotion recognition	MDD: behavioral activation, increased coping behavior
Facilitate emotion experiencing and acceptance	PDA: approach behavior, activation of fear-related somatic sensations

Note. GAD = generalized anxiety disorder; MDD = major depressive disorder; PDA = panic disorder with agoraphobia.

in this regard. When applied to specific disorders, emotional and behavioral activation, especially in situational context, becomes a particularly powerful tool. Modifying action tendencies often, but not always, coincides with and complements techniques to prevent avoidance of emotional expression. In fact, preventing avoidance in some emotional contexts, such as anxiety and fear, is one way to modify action tendencies. Thus, for most phobic situations it is clear that encouraging approach behavior in place of avoidance is an important step. But this strategy of modifying action tendencies also might involve provoking a different emotion, or facial expression, with its associated action tendencies in the phobic context (e.g., laughter or humor as in paradoxical intention). In GAD, implementing this strategy might involve actively prescribing "nonperfect" behavior at home or in the workplace. Passivity and detachment may be more appropriate actions in the context of anger, or more positive, active coping skills to counter withdrawal in depression. It is also clear that individuals may initiate different action tendencies (e.g., approach) during emotion induction while simultaneously engaging in substantial avoidance behavior (e.g., distraction). Thus, attention to both strategies is essential.

At present we are in the beginning process of evaluating the efficacy and feasibility of this protocol. Thus far, we have collected data from several groups of 6 to 8 patients with heterogeneous emotional disorders, including such disparate principal diagnoses as PTSD, MDD, GAD, and a variety of phobic disorders. While final results await more systematic data collection and experimentation, patients in these groups are doing as well as or a bit better than patients in more homogeneous groups. More importantly, patients express their understanding of the fundamental similarities in their experiences, despite different diagnoses and somewhat different presenting symptoms. Confirming this impression through systematic and rigorous experimentation is the next step, as well as further elucidating mechanisms of therapeutic action.

In conclusion, it is of interest to note that the first author had the good fortune to be "present at the creation" of behavior therapy (at least as a student), at a time when the concept of "neuroses" was in ascendance and long-term depth psychotherapy was the "unified" approach to these problems. Now the junior authors are beginning their careers with concepts of "negative affect syndrome" and a proposed unified psychological approach. While ironic on the face of it, the fundamental differences in these two seemingly similar conceptions represent, in a very real way, the revolution of the last 40 years. For now we have a broader and deeper understanding of the psychopathology of emotional disorders that is based on the slow but inexorable process of science. And now we have successful and effective ways — empirically derived, but also firmly grounded in theory — to address the suffering occasioned by negative affect in its many manifestations. The ability to submit the many and varied hypotheses generated over the years to the scientific method, as well as the fact that those generating the hypotheses were often proven wrong along

the way, resulting in mid-course corrections, has brought us to where we are today. Most of us going down this road will be wrong again in the future and it is possible that many of the ideas presented in this article will not be sustained. But we will all be better off finding that out, and in a fundamental sense, this is the fulfilled promise of CBT that differentiates us from our forebears.

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