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ENDURING EFFECTS FOR COGNITIVE BEHAVIOR THERAPY IN THE TREATMENT OF DEPRESSION AND ANXIETY

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■ **Abstract** Recent studies suggest that cognitive and behavioral interventions have enduring effects that reduce risk for subsequent symptom return following treatment termination. These enduring effects have been most clearly demonstrated with respect to depression and the anxiety disorders. It remains unclear whether these effects are a consequence of the amelioration of the causal processes that generate risk or the introduction of compensatory strategies that offset them and whether these effects reflect the mobilization of cognitive or other mechanisms. No such enduring effects have been observed for the psychoactive medications, which appear to be largely palliative in nature. Other psychosocial interventions remain largely untested, although claims that they produce lasting change have long been made. Whether such enduring effects extend to other disorders remains to be seen, but the capacity to reduce risk following treatment termination is one of the major benefits provided by the cognitive and behavioral interventions with respect to the treatment of depression and the anxiety disorders.

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ENDURING EFFECTS FOR COGNITIVE BEHAVIOR THERAPY IN THE TREATMENT OF DEPRESSION AND ANXIETY

Psychosocial interventions have long been touted as providing enduring change. Not only are they said to reduce existing distress or improve functioning, they are believed to do so in a manner that produces lasting change over time. The question is whether this is true: Do psychosocial interventions truly produce enduring effects, and, if so, how do they compare with other major interventions like psychoactive medications?

Our primary focus is on enduring effects: Do the benefits of treatment last over time, beyond the termination of the intervention? We also address the nature of the underlying mechanisms. Enduring effects could be a consequence of the resolution of causal processes that contribute to risk or they could reflect the acquisition of compensatory factors that offset the pernicious effects of those causal processes (Barber & DeRubeis 1989). However, in most instances, we will be lucky if we can establish whether intervention effects endure, much less how those enduring effects are achieved.

Enduring effects can be of at least two kinds. *Treatment* effects reduce existing problems that would not have gone away on their own; they can be said to be enduring to the extent that the problem does not come back. In such instances, all that is required to document that a treatment has an enduring effect is that the changes produced be stable over time. If problems do return, then a treatment still can be said to have an enduring effect if problems return at a lesser rate or intensity than would have been the case if the treatment had not been provided. *Preventive* effects reduce risk for future problems. Such effects may not be immediately apparent in terms of the outcomes of interest; rather, their beneficial effects may become apparent only over time. Nonetheless, to have an enduring effect, an intervention must set in motion causal processes that interrupt the sequence of events leading to the onset or return of the disorder. That means that even delayed effects must reflect ongoing causal processes that may be subject to detection.

If psychosocial interventions do have enduring effects, how would they be detected? For an effect to be said to endure, its benefits must extend beyond the end of the period of intervention. When enduring effects are absolute (when symptoms do not return or expected onsets do not occur), then no comparison is required other than the lack of change from the end of treatment over time. When enduring effects are probabilistic (when symptoms do return to some extent or onsets do occur for some), then gains must be maintained or deterioration forestalled relative to some

type of comparison condition. With respect to treatment, evidence for enduring effects is most compelling when subsequent symptom return is reduced relative to some other equally efficacious intervention and the relative value of those effects is best established by comparison to the most efficacious continuing interventions.

Enduring effects may also affect the rate at which symptoms appear; a treatment can be said to have an enduring effect if it slows the progression of a disorder even if it does not prevent its ultimate onset or return. For this reason, it may be important to chart the rate at which symptoms are manifest over time. The situation is further complicated when symptom onset or return leads patients to seek additional treatment, since efficacious subsequent treatment can reduce the very symptoms that led patients to seek this additional treatment. Cross-sectional assessments at fixed time points that do not take into account differential rates of onset and return or intercurrent treatment are less than wholly adequate, and most investigators now prefer to use some type of survival analysis (Greenhouse et al. 1989, Willett & Singer 1993) or model individual trajectories over time in a manner that allows for the consideration of multiple causal influences (Gibbons et al. 1993, Willett et al. 1991).

Efficacious treatments can fall into at least three categories, depending on their mechanisms of action and the nature of the underlying disorder. Some may be purely *palliative*; that is, they suppress the expression of symptoms so long as they are applied but do nothing to address the processes that drive the underlying disorder. Other interventions may be *curative* in the sense that they eliminate or reverse the underlying process that would otherwise lead to the continuation of the disorder. Still other interventions can be said to be *prophylactic*. These interventions eliminate or offset processes that contribute to risk for future onsets. Both curative and prophylactic interventions can be said to have enduring effects, with the former keeping symptoms from coming back (relapse) and the latter preventing wholly new onsets (recurrence).

Existing interventions can be classified with respect to this typology. As effective as medications are for many psychiatric disorders, there is no evidence that they are anything more than palliative; that is, they suppress symptoms so long as they are taken, but often do little to alter the course of the underlying disorder or to reduce subsequent risk once their use is discontinued. The cognitive and behavioral interventions, on the other hand, actually may be curative and possibly even prophylactic; that is, there is evidence that they produce lasting change or even reduce future risk. To the extent that this is true, it means that patients need not stay in treatment forever or that disorders can actually be prevented and possibly never even expressed (Hollon et al. 1992b). Dynamic-interpersonal and humanistic-experiential interventions typically have not been tested in a way that would allow for the detection of enduring effects.

In this article, we highlight the evidence for the statements above. The bulk of the evidence to date comes from the literature on depression and the anxiety disorders and involves the cognitive and behavior therapies (collectively referred to throughout the review as CBT). Typically, this takes the form of comparisons

between patients treated to remission with CBT versus medication, then followed over time following treatment termination. The review is illustrative rather than exhaustive, and not all relevant studies are described, although we have made a special effort to discuss studies that could have shown enduring effects for other kinds of treatments or that failed to show enduring effects for CBT.

TREATMENT AND PREVENTION OF DEPRESSION

Depression is one of the most prevalent of the psychiatric disorders and a leading cause of disability worldwide. Although most patients remit from any given episode, symptoms often come back at some later time, and depression is now considered to be a largely recurrent disorder (when it is not chronic). Several different types of interventions have been shown to be efficacious in its treatment (see Hollon et al. 2002 for a review). Antidepressant medication (ADM) has been shown to be superior to placebo controls in literally thousands of trials and tends to suppress symptoms for as long as it is continued or maintained, but there is no evidence that it does anything to reduce underlying risk once its use is terminated. Both interpersonal psychotherapy (IPT) and CBT appear to be about as efficacious as ADM with respect to the reduction of acute distress; moreover, IPT appears to have a greater breadth of effect with respect to enhancing the quality of relationships, whereas CBT appears to be more enduring than ADM. More purely behavioral interventions have not been tested often, but have performed well in recent trials. Family-focused therapy shows promise in preventing relapse or recurrence in bipolar disorder, but has been little tested in unipolar depression. More traditional dynamic psychotherapy has fared poorly in direct comparisons with other interventions (although questions can be raised about the adequacy with which it has been implemented), and humanistic-experiential therapies have not been often tested.

Cognitive Therapy and the Prevention of Relapse

Among the various efficacious interventions, cognitive therapy (CT) has produced the most consistent evidence of enduring effects. In this variant of CBT, patients are trained to collect information in a systematic fashion to offset the influence of maladaptive information-processing strategies and to conduct behavioral experiments to test the accuracy of their negative beliefs (Beck et al. 1979). Patients also are encouraged to examine the accuracy of their beliefs using a series of logical tools (cognitive restructuring), and a premium is placed on teaching the patient to do the therapy for him- or herself, in recognition of the chronic recurrent nature of depression. Preparation for termination is addressed from the beginning of therapy (serving to justify the extensive use of homework) and explicit practice is provided in relapse prevention throughout.

Patients treated to remission in CT appear to be about half as likely to relapse following treatment termination as are patients treated to remission with medications.

A recent trial conducted by our group is illustrative. In that study, patients with moderate to severe depression were found to be more likely to respond to 8 weeks of treatment with either CT or ADM than with pill-placebo (a demonstrable treatment effect); by 16 weeks, response rates to the two active interventions were virtually identical (just under 60%) (DeRubeis et al. 2005). At that point, patients who had responded to medications were randomly assigned to continuation ADM or withdrawn onto pill-placebo and followed over the ensuing year. Patients who responded to CT terminated treatment and were allowed no more than three booster sessions (not more than one per month) over that same interval. As shown on the left in Figure 1, patients withdrawn onto pill-placebo were considerably more likely to relapse over the ensuing 12-month interval than were patients continued on medication (adjusted relapse rates of 76.2% versus 47.2%, 23.8% versus 52.8% survival); patients with a history of prior exposure to CT (30.8% relapse or 69.2% survival) did as least as well as patients continued on ADM (Hollon et al. 2005). With a hazard ratio of 0.30, prior exposure to CT reduced risk for relapse by about 70% relative to medication withdrawal. By way of contrast, continuation medication

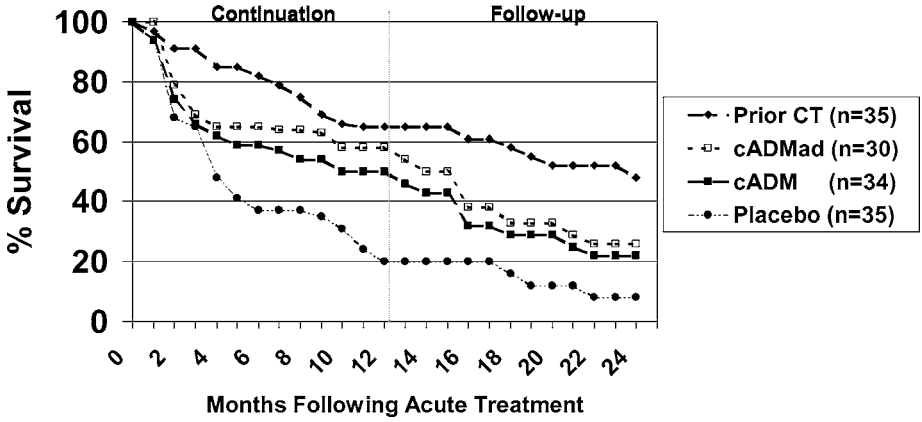

Figure 1 Cumulative proportion of depressed treatment responders who survived without relapse during continuation (first 12 months) and cumulative proportion of recovered patients who survived without recurrence during subsequent follow-up (months 13–24). Prior cognitive therapy allowed only three booster sessions following acute response (first 12 months) and no sessions following recovery (months 13–24); continuation antidepressant medication (cADM) patients continued on active medications following acute response (first 12 months), then withdrawn from all pills following recovery (months 13–24); cADMad patients represent that subset of cADM patients who adhered to prescribed medication during continuation phase (first 12 months); placebo patients withdrawn from active medications onto pill-placebo following acute response (first 12 months), then withdrawn from all pills following recovery (months 13–24). Adapted from Hollon et al. (2005), copyright 2005 by the American Medical Association. Reprinted by permission.

reduced risk by half relative to medication withdrawal (with a hazard ratio of 0.50). Even when nonadherence was taken into account (*dashed line* in Figure 1), prior CT did as well as continuation medication. This suggests that prior CT has an enduring effect that is at least as large in magnitude as keeping patients on medications, a purely palliative intervention that is the current standard of treatment for recurrent depression (Am. Psychiatric Assoc. 2000).

This is one of the most robust findings with respect to enduring effects in the literature. In several earlier trials, patients treated to remission in cognitive therapy were only about half as likely to relapse as were patients treated to remission with medications alone following treatment termination (Blackburn et al. 1986, Kovacs et al. 1981, Simons et al. 1986), and no more likely to relapse than were patients continued on medications (Evans et al. 1992). Only two studies have failed to find enduring effects for prior CT (Perlis et al. 2002, Shea et al. 1992); one involved the use of a medication (fluoxetine) with a particularly long half-life, and questions have been raised about the adequacy of the therapy provided in the other (Jacobson & Hollon 1996).

Although promising, these findings do not speak directly to the prevention of recurrence, defined as the onset of wholly new episodes (Frank et al. 1991). Depressed patients appear to be at elevated risk for symptom return (relapse) for the first six to nine months following initial response to medications when it is likely that the underlying episode has yet to run its course (Hollon et al. 1990). No good pharmacotherapist would withdraw patients from medications so soon after initial response, and it has become standard practice to continue patients on medications for at least six months after initial remission. Clearly, the early withdrawal practiced in the studies just described was done for research purposes only and does not reflect standard clinical practice. Nonetheless, studies of this kind do establish that CBT has an enduring effect that lasts beyond the end of treatment. Current medical practice is moving in the direction of maintaining patients with a history of recurrence (the vast majority of all depressed patients) on medication indefinitely (Am. Psychiatric Assoc. 2000). In that context, it would be important to know if CBT's enduring effects extend to the prevention of recurrence.

There are reasons to think that the effects might prevent recurrence. In the Hollon et al. 2005 study, we continued to follow patients who survived without relapse for another year (months 13–24 following the end of acute treatment). Patients who survived the full year of continuation treatment without relapse were withdrawn from medications at that time and compared to patients with a history of prior exposure to CT. Given that all of these patients had gone a full 12 months without relapse following initial remission, they were assumed to be fully recovered from the treated episode and any subsequent return of symptoms was considered to represent the onset of a wholly new episode (recurrence). As suggested by the slopes of the curves in the right half of Figure 1 (months 13–24), recovered patients withdrawn from medications were more likely to experience a recurrence over that second year of follow-up than were patients with a history of prior exposure to CT (Hollon et al. 2005). Only 5 of 20 recovered patients with a history of prior

exposure to CT experienced a recurrence during that second year of naturalistic follow-up relative to 7 of 14 patients treated to recovery with medications alone. Taking attrition into account, the adjusted recurrence rates were 17.5% for prior CT versus 53.6% for prior continuation ADM, with a hazard ratio of 0.15 (meaning that prior CT reduced risk for recurrence relative to medication withdrawal by about 85%). Although larger studies are needed, these findings suggest that CT's enduring effect may extend beyond relapse to the prevention of recurrence. To the extent that this is true, it would mean that CT is not only curative (by virtue of bringing the treated episode to an end), but also possibly prophylactic (in the sense of forestalling the onset of wholly new episodes).

One important caveat must be mentioned. Such studies of enduring effects typically have relied on naturalistic follow-ups of acute treatment trials. Although patients in those trials initially were randomized to the different treatment conditions, subsequent attrition and nonresponse could have served to unbalance the groups with respect to preexistent patient characteristics. Differences in such characteristics (if they exist) could in turn be related to subsequent risk for relapse or recurrence. Thus, initial treatment could act as a "differential sieve" that systematically unbalances the groups with respect to underlying risk (Klein 1996). In the Hollon et al. 2005 study, only about 60% of the patients assigned to either condition both completed and responded to treatment and were therefore eligible to take part in the continuation phase. Losing nearly half the sample provides ample opportunities for differential retention to unbalance the groups; it is quite possible that patients who responded to CT were simply at lower risk for relapse for reasons unrelated to treatment than were patients who responded to medication treatment. There is little existing evidence that CT's enduring effect is an artifact of differential retention (responders to the different treatments rarely differ in meaningful ways), but there is nothing in the logic of the typical follow-up design that precludes the possibility.

The best way to reduce this risk to internal validity is to minimize the proportion of patients who fail to enter the period of risk. Attrition and nonresponse are inherent in any study, but there are design decisions that can be made to reduce their magnitude. For example, patients could be treated to criteria (remission or recovery) rather than for a fixed time prior to treatment termination. Similarly, the study design could be set to minimize the differences between conditions other than those directly relevant to the question of interest. For example, all patients might be medicated and psychotherapy provided for half on a random basis (rather than comparing the two single modalities directly) to minimize differences resulting solely from the provision of medications.

Such a strategy depends on the absence of interactions between drugs and psychotherapy. Two studies found that CBT's enduring effect was robust whether it is provided alone or in combination with medications (Blackburn et al. 1986, Evans et al. 1992), but a third found that relapse rates were higher when CT was provided in combination with medication relative to CT alone or CT plus placebo (Simons et al. 1986). Moreover, the existing combinatorial trials were all conducted with the older tricyclic medications; it remains to be seen if newer medications

with less problematic side effects and broader profiles of action will undermine CT's enduring effects. In a subsequent section, we discuss indications that such medications may sometimes interfere with the enduring effects of CBT in the treatment of anxiety.

There also are indications that CBT or related interventions may have enduring effects when provided after medications have been used to reduce acute distress. For example, Paykel and colleagues found that adding CT to medication treatment for partial responders not only helped resolve residual symptoms but also reduced risk for subsequent relapse after the end of the psychosocial treatment (Paykel et al. 1999). Even more interestingly, Fava and colleagues found that adding an enhanced version of CT, called "well-being therapy," to medications for remitted patients in continuation treatment reduced risk for recurrence following medication withdrawal (Fava et al. 1998). Finally, an innovative integration of acceptance and meditation called "mindfulness-based cognitive therapy" has been shown to reduce risk for relapse and recurrence following treatment termination in patients first treated to remission with medications (Teasdale et al. 2000).

There are even indications that CBT may have a preventive effect when provided to children and adolescents at risk (Gillham et al. 2000). Clarke and colleagues have conducted a pair of studies in adolescents at risk by virtue of having a parent with a history of depression; in both trials, training in skills designed to facilitate affective regulation reduced risk for subsequent diagnosable disorder (Clarke et al. 1995, 2001). Similarly, evidence has been found of preventive effects in nondepressed subjects selected on the basis of problematic cognitive style both in children (Jaycox et al. 1994) and college students (Seligman et al. 1999). By definition, preventive effects are enduring effects, since the benefits are obtained long after the intervention is over.

Other types of interventions simply have not been adequately tested. Dynamic psychotherapy has rarely been found to be efficacious with respect to acute treatment; although it is possible to have an enduring effect in the absence of any immediate effect, it is unlikely that such an effect would be detected. IPT has been shown to prevent relapse or recurrence when continued (Klerman et al. 1974) or maintained (Frank et al. 1990), but there is little evidence of any enduring effect once its use is terminated (Hollon et al. 2002). IPT was included in the National Institute of Mental Health Treatment of Depression Collaborative Research Program along with CT and medications, but medication treatment was extended for the first several months of the posttreatment follow-up (Shea et al. 1992). As described above, the program was one of only two studies to date not to find an enduring effect for CT, and it is not clear whether any such effect could have been detected given the methods used. The follow-up phase of an earlier acute treatment trial found no indication that prior IPT had any enduring effect relative to medication withdrawal, but that design relied on a single cross-sectional assessment that would likely have failed to detect the presence of enduring effects (Weissman et al. 1981). There are indications that more purely behavioral interventions also might have enduring effects, a point to which we return in a subsequent section.

In a disorder such as depression, patients who experience a relapse or recurrence following medication withdrawal are likely to seek additional treatment; if symptom return is not monitored in an ongoing fashion, the outcome of interest will likely be missed because subsequent treatment will reduce the distress before the next assessment. That is why we prefer to monitor symptom status in a continuous (albeit retrospective) fashion. Given that patients withdrawn from medications typically experience more symptom return, they are more likely to require additional treatment. Periodic assessments focused solely on current symptom levels typically fail to detect such differences because the effects of subsequent treatment tend to offset the very symptom return that led patients to get back on medications in the first place (Evans et al. 1992).

Mechanisms Underlying Enduring Change

How do cognitive and behavior therapies produce their enduring effects? Cognitive theory suggests that change in what people believe and the way they process information is the primary mechanism of change in CT (Beck 1991). Several studies have shown that thinking does change over the course of therapy; however, the kinds of "surface-level" automatic negative thoughts found in the stream of consciousness typically change as much in pharmacotherapy or other successful interventions as they do in CT (Imber et al. 1990, Simons et al. 1984). What does appear to change in a more specific fashion are the underlying beliefs and information-processing propensities often found in depression, such as core beliefs about the self or the way an individual explains the causes of negative life events. Such core beliefs and information-processing styles tend to lie dormant until activated by negative affect or external stress and serve as the stable cognitive predispositions in a larger diathesis-stress model of depression (Hollon et al. 1992b).

For example, in an earlier trial, we found that CT and medication treatment produced comparable rapid change in depression, with 90% of the symptom change occurring in the first six weeks treatment (Hollon et al. 1992a), but that patients treated to remission with CT were only half as likely to relapse following treatment termination as were patients treated to remission with medications (Evans et al. 1992). Change in "surface-level" automatic negative thoughts such as a sense of hopelessness was nonspecific with respect to treatment modality and mirrored the rapid rate of change shown by depressive symptoms, although it did predict subsequent change in depression to a greater extent in CT than in medication treatment (DeRubeis et al. 1990). At the same time, patients treated to remission with CT showed considerably greater change in underlying attribution style (the way they explained negative life events), and the bulk of that differential change occurred in the second half of treatment, well after the bulk of change in depression (Hollon et al. 1990). Moreover, this differential change in attribution style predicted the greater rate of relapse in the patients treated with medications alone relative to CT following treatment termination. Change in these more stable

information-processing proclivities mediated the enduring effects of CBT in a sample of at-risk young adults provided with a preventive intervention (Seligman et al. 1999). Taken together, these findings suggest that change in cognition mediates the enduring effect found for CBT, but it is change in stable cognitive predispositions and underlying information-processing proclivities that is key to prevention.

We do not yet have a good sense as to whether these changes reflect true accommodation in underlying cognitive predispositions or the acquisition of compensatory mechanisms, since existing measures are susceptible to either process (Barber & DeRubeis 1989). Anecdotal reports from patients suggest that it is more the latter, at least at first, as they describe needing to remind themselves to engage in formal cognitive restructuring techniques when they start to interpret negative life events in a problematic manner. Nonetheless, these same patients describe these capacities as becoming more automatic over time, such that they are less likely to jump to negative conclusions, a process more in keeping with the notion of accommodation. This area merits further investigation.

Teasdale and colleagues also reported that change in underlying information-processing proclivities mediated differential relapse, although in their trial it was becoming less extreme that was beneficial, not simply becoming less negative (Teasdale et al. 2001). That is, patients who became unrealistically positive were also at greater risk, a pattern that we have replicated in findings in our most recent trial (Hollon et al. 2005). Some patients appear to become unduly positive in CT, overshooting even normal controls, something we do not see in ADM. This unrealistic optimism appears to leave patients at elevated risk and produces a curvilinear relation between cognitive change and subsequent relapse that is quite distinct from the linear relation observed for ADM. It is unclear why some patients become unduly optimistic, although we suspect it reflects a triumph of wishful thinking over the more tedious process of reality testing. Whatever its source, it is clearly specific to CT, but it is not something that we have observed in earlier studies. This is something that needs to be addressed in future studies.

Finally, Tang & DeRubeis (1999) have observed that many patients treated with CT show "sudden gains" following a single session that account for the bulk of the change they show across the course of treatment. These sudden gains occur at different times for different patients, but tend to be preceded by cognitive change and followed by improved ratings of the therapeutic alliance. In effect, it is as if the patient suddenly "understands" that their thinking is unduly negative, rather than their personalities that are flawed or even their life situations that are to blame. Once they come to this realization, they seem to do a better job of managing their own affect and behavior. Patients who show sudden gains tend to get better faster and to stay better longer than do patients who show a more gradual pattern of response. At the same time, process studies indicate that attention to specific concrete beliefs and behaviors early in treatment leads to greater subsequent change in depression and higher ratings of the quality of the therapeutic relationship (DeRubeis & Feeley 1990, Feeley et al. 1999). Taken in aggregate, these findings suggest that patients are most likely to show enduring change when their therapists focus on specific

behavioral and cognitive strategies in a structured manner, but that this change is likely to emerge in a rapid and unpredictably idiosyncratic fashion across patients.

Enduring Effects in Behavior Therapy

Recent studies suggest that more purely behavioral interventions may also produce enduring effects. In a component analysis, Jacobson and colleagues found that the behavioral strategies used in the early stages of CT produced as much change as the complete treatment package when extended over the full course of therapy (Jacobson et al. 1996). Most critically for the current discussion, there was no evidence of differential risk for relapse following treatment termination (Gortner et al. 1998). This led to the articulation of a more fully realized contextual intervention, called "behavioral activation" (BA), that emphasizes the functional connection between behaviors and outcomes and eschews attention to the content of cognitions (Jacobson et al. 2001, Martell et al. 2001). In a recent trial, BA was found to be as efficacious as ADM with respect to acute treatment (Dimidjian et al. 2005) and as enduring as CT with respect to the prevention of subsequent relapse and recurrence (KS Dobson, SD Hollon, S Dimidjian, KB Schmaling, RJ Kohlenberg, R Gallop, S Rizvi, JK Gollan, DL Dunner, NS Jacobson, manuscript in preparation). Given that therapists pay little attention to thought content in BA (other than noting the role of rumination in maintaining behavioral avoidance), it is clear that direct efforts to address beliefs and information-processing proclivities may not be required to produce enduring change. However, it remains possible that these largely behavioral strategies work through underlying cognitive mechanisms to produce their enduring effects (Bandura 1977). Additional research will be required to resolve this issue. For now, the best that we can do is to note that there is good evidence for enduring effects in CT for depression (quite possibly mediated by changes in underlying cognitive predispositions) and promising indications that these enduring effects may also extend to more behavioral interventions through mechanisms yet unknown.

Prevention of Bipolar Disorder

Patients with bipolar disorder are at risk for episodes of mania or hypomania as well as depression. Genetic factors play a greater role in bipolar disorder than in unipolar depression, and the course is marked by frequent relapse and recurrence. Medication forms the core of treatment and most patients are maintained indefinitely on lithium or mood stabilizers to forestall symptom onset (especially mania), with antidepressants often added to deal with depressive symptoms (Am. Psychiatric Assoc. 2002).

Psychosocial interventions typically are used in an adjunctive fashion and it has only been in recent years that empirical studies have demonstrated their value. Teaching patients to detect prodromal signs and seek prompt medical help has been shown to reduce the frequency of onset of full manic episodes (Perry et al. 1999), and interpersonal and social rhythm therapy has been found to reduce the

frequency of depressive relapse (Malkoff-Schwartz et al. 1998). Family-focused treatment, designed to reduce stress and improve communication, has been found to reduce the frequency of mania and depression both during and after the end of treatment (Miklowitz et al. 2003). This represents one of the few instances in which enduring effects have been documented for any psychosocial intervention other than CBT.

Lam and colleagues found that adding CT to medication management over a six-month period reduced risk for subsequent relapse relative to medications alone for the rest of the year in bipolar patients not currently in episode (Lam et al. 2003). A subsequent follow-up found that this advantage was maintained over the next year and a half, but largely reflected differences that emerged during and shortly after treatment (Lam et al. 2005). Thus, although CT had a beneficial effect (in that patients did better during treatment when CT was added to medications), it is not clear that it had an enduring effect such that patients previously treated with CT were at any lower risk of relapse once its use was terminated than were patients receiving medication only (a direct comparison of the conditional probabilities of relapse for each specific interval following the termination of the psychosocial treatment would have been of interest).

However, survival analyses based on time to first relapse are not the best way to detect enduring effects when differences emerge during treatment, since high-risk patients are more likely to be retained (and thus remain at risk) by the more efficacious treatment (Hollon et al. 2002). Thus, it is possible that documented success of CT in preventing relapse during its initial application led to the differential retention of more high-risk patients going into the later months of the follow-up, thereby obscuring possible enduring effects. In this regard, it is of interest that patients previously treated with CT spent fewer months in episode during the extended follow-up than did patients treated with medication management alone, an index that would not be biased by differential retention. This suggests that CT with bipolar patients may have the same kind of enduring effect that has been found so often in unipolar depression.

PANIC AND THE ANXIETY DISORDERS

Is there evidence for enduring effects in other disorders? Such evidence does exist, especially with respect to panic and the anxiety disorders, although it is not as well documented as for depression and is largely limited to CBT (Hollon & Beck 2004). In this section, we review panic and the anxiety disorders.

Catastrophic Cognitions in Panic and Agoraphobia

As for depression, different theoretical models exist and each posits different interventions for panic disorder and agoraphobia. Biological models view panic as the consequence of the spontaneous discharge of neural centers deep in the brain

stem or limbic system and treat the symptoms with medications (Lydiard et al. 1996). Behavioral models regard panic attacks to be a conditioned response to internal or external cues that need to be extinguished via exposure; agoraphobia is regarded as reinforced avoidance behavior that needs to be suppressed through response prevention (Barlow & Lehman 1996). Cognitive models emphasize the role of catastrophic misinterpretations of benign bodily sensations and encourage patients to test the accuracy of their beliefs via inducing the physiological sensations that they most fear (Beck & Emery 1985, Clark 1986). Interventions based on all three models have been shown to be both specific and efficacious in the treatment of panic disorder (DeRubeis & Crits-Christoph 1998).

There are consistent indications that treatment effects achieved with the psychosocial interventions are more likely to endure following treatment termination than are those obtained with medications (Roth & Fonagy 2005). For example, Sharp and colleagues (1996) found that patients treated with CBT (alone or in combination with medication) were more likely to maintain gains at a six-month posttreatment follow-up than were patients treated with fluvoxamine alone. Loerch and colleagues (1999) found that adding CBT enhanced response relative to either moclobemide or placebo alone and that those patients in the latter two conditions were far more likely to seek additional treatment across a naturalistic follow-up. In perhaps the best of the early trials, Clark and colleagues (1994) found CT superior to either imipramine (an older tricyclic ADM) pharmacotherapy or applied relaxation in a sample of patients with panic disorder (and each superior to a wait-list control); across a subsequent six-month follow-up, only 5% of the CT patients relapsed following treatment termination, compared with 40% of the patients withdrawn from medications. Moreover, as specified by theory, CT produced a reduction in catastrophic cognitions that was greater than that of either of the other two active treatments, and the frequency of such beliefs at the end of treatment predicted subsequent risk for relapse following treatment termination.

Barlow and colleagues (2000) also found an enduring effect for prior CBT, as well as a cautionary note with regard to its combination with medication. In a multisite study, patients with panic disorder (with or without mild agoraphobia) were randomly assigned to three months of weekly acute treatment followed by six months of monthly maintenance treatment with either CBT or imipramine, each alone and in combination, or pill-placebo, again alone and in combination with CBT. Each single modality was superior to pill-placebo, with combined treatment somewhat better still by the end of maintenance treatment. Imipramine produced higher quality response among treatment completers, but CBT was more enduring. As shown in Figure 2, adjusted relapse rates were 8% for patients who responded to CBT alone (92% survival) versus 25% for responders to imipramine alone (75% survival). Curiously, adding active medication appeared to undermine the enduring effects of CBT, as 36% of the patients in the combined condition relapsed (64% survival). It is unlikely that purely psychological processes mediated this effect (such as misattributing treatment gains solely to medications), since only 4% of the patients treated with the combination of CBT plus pill-placebo relapsed

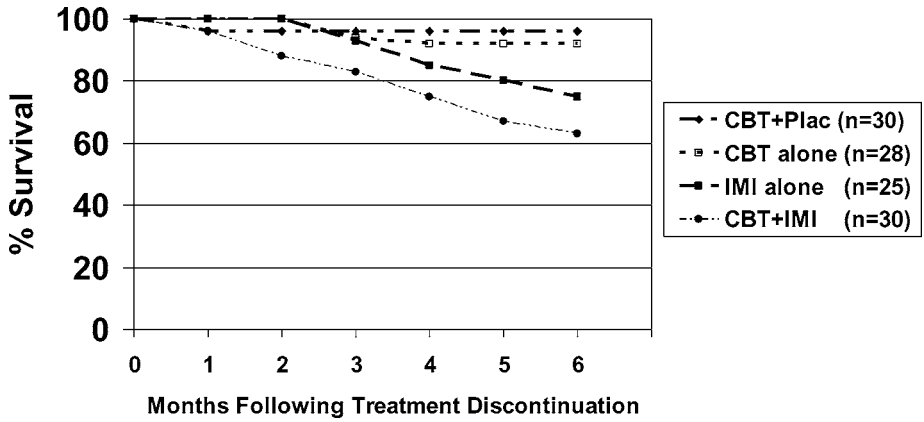

Figure 2 Proportion of panic patients meeting criteria for relapse following termination as a function of prior treatment condition. CBT + Plac patients received cognitive behavior therapy plus pill-placebo; CBT-alone patients received cognitive behavior therapy only; IMI-alone patients received imipramine pharmacotherapy only; CBT + IMI patients received cognitive behavior therapy plus imipramine pharmacotherapy. Adapted from Barlow et al. (2000).

(96% survival). It also is unlikely that this effect was an artifact of differential retention, since response rates were as high when CBT was combined with pill-placebo as with active medication.

This pattern of findings suggests that if adding medication suppresses CBT's enduring effect, it does so through largely pharmacological mechanisms. For example, it is possible that the presence of active medication acts to retard learning, either by suppressing interioceptive cues in a manner that slows the process of habituation (behavioral) or by reducing the opportunity for disconfirmation of catastrophic expectations (cognitive). That is, medication may operate through biological mechanisms that suppress the occurrence of panic for so long as it is taken, but that interfere with other learning-based mechanisms that would have produced more lasting change.

This is not the first time that the addition of medications has undermined the enduring effect of a psychosocial intervention. In an earlier trial, Marks and colleagues combined either exposure or relaxation with alprazolam (a high-potency benzodiazapine) or placebo in a factorial design. Agoraphobic patients treated with the combination of exposure plus alprazolam showed a higher rate of relapse than did patients treated with the combination of exposure plus placebo (Marks et al. 1993). It would appear that medication is more likely to interfere with the enduring effects of CBT for panic and agoraphobia than has been the case to date in the treatment of depression.

The precise nature of the mechanisms involved remains unclear, but it is possible that they will vary as a function of the nature of the disorder and the specific

medication. Disorders such as panic with rapid symptom onset may be more susceptible to interference effects than are disorders with greater temporal stability, such as depression. At the same time, patients are particularly likely to attribute change to fast-acting medications such as alprazolam, and any medication that produces rapid and transitory change in affective states is particularly likely to induce state-dependent learning. Moreover, medications with short half-lives are particularly likely to produce discontinuation effects, making them harder to withdraw and increasing the risk of relapse following termination. In fact, the sequential application of CBT has been used to facilitate withdrawal from high-potency benzodiazapines such as alprazolam, which can provoke rebound panic attacks following discontinuation (Bruce et al. 1999, Otto et al. 1993). It remains to be seen whether such a sequential strategy will prove useful in helping patients withdraw from newer antidepressants with short half-lives, such as venlafaxine or paroxetine, and whether those medications will interfere with CBT's enduring effect if provided in combination during active treatment.

There are few indications that either dynamic-eclectic or humanistic-experiential interventions are efficacious in the treatment of panic disorder, much less have enduring effects. More purely behavioral interventions tend to produce gains that endure (Arntz & Van den Hout 1996, Öst & Westling 1995), although enduring effects have not been clearly documented. Presence of agoraphobic avoidance predicts poorer overall response, and tests of enduring effects have been few (Bouchard et al. 1996). Nonetheless, it is clear that CBT has an enduring effect in the treatment of panic disorder. As in the treatment of depression, this effect is most evident in comparisons to drugs and represents one of the main advantages of CBT over medications.

Hypochondriasis and Concerns about Physical Illness

Patients with hypochondriasis believe they have a physical illness and take little comfort from medical reassurance. Although common in medical settings, the disorder has rarely been studied empirically and has long been thought to be impervious to treatment. Cognitive theory suggests that hypochondriasis is a consequence of an enduring tendency to misinterpret innocuous physical sensations as symptoms of a serious illness, much as in panic disorder, with the key difference being the perceived imminence of the medical catastrophe (Warwick & Salkovskis 1990). Patients in CT are encouraged to examine the evidence for their beliefs and to conduct behavioral experiments in which they induce symptoms by focusing attention on their body. Reassurance seeking and "body checking" are discouraged to reduce the operation of safety behaviors, and patients are encouraged to keep a daily record of negative thoughts and rational responses.

Recent studies suggest the efficacy of this approach. Warwick and colleagues (1996) found CT superior to a wait-list control after four months of treatment, with gains essentially maintained at a three-month follow-up. Clark and colleagues (1998) found CT superior to a wait-list control on all measures and better than

behavioral stress management on symptoms of hypochondriasis, with gains essentially maintained over a subsequent 12-month follow-up. Barsky & Ahern (2004) found that six sessions of CBT (similar but not identical to the approach described above) produced greater change in symptoms of hypochondriasis compared with usual medical care across a 12-month posttreatment follow-up.

Given that hypochondriasis has long been thought to be refractory to treatment, these findings are most promising; this is especially the case for indications that gains endure over time following treatment termination. Evidence for enduring effects would be even more compelling if prior CBT were to prove more stable over time than some alternative intervention of equal initial efficacy; however, with the possible exception of behavioral stress management, no other treatment (including medications) has yet been found to be efficacious in the treatment of hypochondriasis.

Generalized Anxiety Disorder and the Primacy of Worry

In recent years, cognitive symptoms like pervasive apprehension and worry have come to be seen as the core symptoms of generalized anxiety disorder (GAD) (Brown et al. 1994). CBT involving the combination of relaxation training and cognitive restructuring has been found to be both efficacious and specific in a number of comparisons to other interventions and control conditions, and applied relaxation has shown promise in a smaller number of trials (DeRubeis & Crits-Christoph 1998). More purely behavioral interventions based on exposure are difficult to implement, since there is often no clear external referent to target. Medication treatment is often problematic; minor tranquilizers can induce dependence and tend to lose potency with prolonged use, and antidepressants do nothing to reduce future risk (Nathan & Gorman 1998). More traditional forms of psychotherapy have long been touted, but rarely tested.

Treatment gains for CBT are generally well maintained over time, both in adults (Borkovec & Ruscio 2001) and in geriatric populations (Stanley et al. 2003). Differences favoring cognitive strategies sometimes have emerged following treatment termination relative to more purely behavioral interventions (Borkovec & Costello 1993), and Durham and colleagues (2003) recently reported that patients in CBT exhibited lower overall symptom severity and less interim treatment in the 8–14 years following treatment termination than did patients receiving either dynamic or pharmacological treatment.

Nonetheless, there is a general sense that more can be done with the treatment of GAD. For example, Borkovec and colleagues (2002) found that interpersonal difficulties remaining after treatment with CBT predicted poorer status across follow-up, and they called for the inclusion of strategies that targeted these problems. Similarly, Fava and colleagues (2005) found that adding strategies designed to promote a sense of well-being enhanced the efficacy of CBT, with gains maintained across a one-year follow-up. Finally, Ladouceur and colleagues (1999) have dropped relaxation training entirely to focus on more purely cognitive targets such

as intolerance of uncertainty and cognitive avoidance. A recent controlled trial found this more purely cognitive intervention superior to a delayed treatment control; 77% of all participants treated with CBT no longer met criteria for GAD following treatment (Ladouceur et al. 2000). In a subsequent trial, participants provided with group CBT did better than a wait-list control and continued to improve across a 24-month follow-up (Dugas et al. 2003). It remains to be seen how this approach will compare to more conventional renditions of CBT that incorporate more explicitly behavioral components, but treatment effects for each appear to be well maintained over time.

Interpersonal Anxiety and Social Phobia

Social phobia (also known as social anxiety disorder) tends to begin early in life and often follows a chronic course (Davidson et al. 1993). Social phobia involves an undue fear of evaluation by others and the accompanying desire to avoid situations in which scrutiny is anticipated. A number of pharmacological agents have been found to reduce distress, including the monoamine oxidase inhibitors and, more recently, the selective serotonin reuptake inhibitors (SSRIs) (Hidalgo et al. 2001). Behavioral approaches based on exposure to social situations (often supplemented with training in social skills) generally have been efficacious, although gains have not always been well maintained over time (DeRubeis & Crits-Christoph 1998). Cognitive approaches target beliefs regarding personal defects that could lead to ridicule or censure by others in social situations (Beck & Emery 1985), and a recent extension focuses on the elimination of safety behaviors that retard the disconfirmation of those beliefs (Wells et al. 1995).

Although the effects produced by exposure alone have not always been stable over time, they do appear to be more enduring than are those produced by medications. Blomhoff and colleagues (2001) found that sertraline (an SSRI) was superior to exposure therapy in a primary-care sample, but that patients treated with the psychosocial intervention continued to improve across a subsequent year-long follow-up, whereas those treated with sertraline (alone or in combination) tended to deteriorate after treatment termination (Haug et al. 2003). Similarly, a naturalistic study of long-term treatment outcomes in social phobia found that concomitant use of benzodiazepines during active treatment was associated with greater risk of relapse in patients treated with exposure (Fava et al. 2001). These reports are reminiscent of the findings in panic and agoraphobia that adding medication may undermine the enduring effects of CBT (Barlow et al. 2000, Marks et al. 1993). There are also indications that the addition of cognitive restructuring facilitates the maintenance of gains produced by exposure to social situations (e.g., Butler et al. 1984, Heimberg et al. 1993).

Perhaps the best evidence for the enduring effects of CBT in social phobia comes from a two-site comparison to medications that did a particularly nice job of controlling for allegiance across sites and conditions (Heimberg et al. 1998). In that trial, patients with social phobia were randomly assigned to cognitive

behavioral group therapy (CBGT) alone, phenelzine (a monoamine oxidase inhibitor) alone, pill-placebo, or educational-supportive group therapy. Both active treatments were superior to the two control conditions; phenelzine produced somewhat faster response than did CBGT, but rates of response were comparable by the end of acute treatment (12 weeks). Responders to the two active treatments were provided with six months of additional maintenance treatment and then tracked across a subsequent six-month interval following treatment termination. As shown in Figure 3, there was clear evidence for an enduring effect for the psychosocial intervention; none of the patients previously treated with CBGT relapsed following treatment termination versus 33% of the patients previously treated with phenelzine (100% versus 67% survival) (Liebowitz et al. 1999).

Clark and colleagues have suggested that patients with social phobia focus undue attention on their image of themselves in social situations and engage in safety behaviors (strategies designed to protect them from feared consequences) that make them appear less socially skilled and prevent them from learning that they can handle the feared encounters (Wells et al. 1995). Patients are videotaped engaging in various social situations, both with and without safety behaviors, and are then invited to watch both tapes and rate themselves on various characteristics. Patients typically find that they appear more relaxed and socially skilled when they drop their safety behaviors and that their internal images of themselves are more negative and self-derogatory than they actually appear. Moreover, patients show greater change in beliefs and greater reductions in anxiety when they drop their safety behaviors in exposure situations.

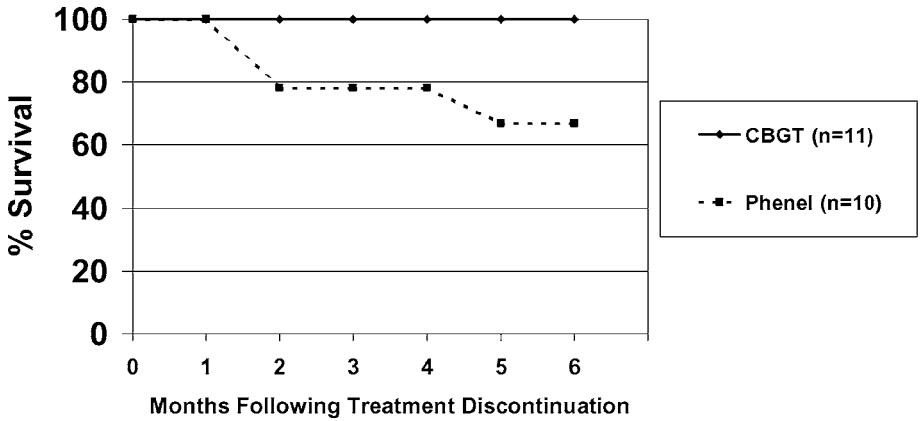

Figure 3 Relapse following successful treatment among patients with social phobia treated with cognitive behavioral group therapy (CBGT) or medications. CBGT patients were previously treated with cognitive behavioral group therapy; phenelzine patients previously treated with the monoamine oxidase inhibitor phenelzine. Adapted from Liebowitz et al. (1999).

Clark and colleagues have argued that targeting self-focused attention and encouraging patients to drop their safety behaviors during exposure can facilitate their capacity to learn from experience and hasten their response to treatment. A recent comparison suggests that this newer individual approach may be superior to more conventional CBGT, which had been done in groups to take advantage of the opportunities afforded for exposure to social situations (Stangier et al. 2003). In another recent comparison, Clark and colleagues found that patients with social phobia who were treated with individual CBT in the manner described above showed greater reductions in measures of social phobia across 16 weeks of active treatment than did patients treated with either fluoxetine or placebo. Moreover, the advantage observed for CBT relative to medication was essentially maintained across three months of booster treatment and was still evident at a 12-month follow-up following treatment termination (Clark et al. 2003). These findings, combined with those obtained by Liebowitz and colleagues (1999) with CBGT, suggest that CBT for social phobia has an enduring effect not found with medication.

Specific Phobias and the Perception of Danger

Specific phobias involve an intense fear of certain objects or situations and a corresponding desire to avoid being in their presence. Behavior theory suggests that phobias are established via traumatic conditioning and maintained by avoidance behaviors that prevent their extinction (Mowrer 1948). Behavioral interventions such as systematic desensitization and exposure plus response prevention are clearly efficacious and represent the current standard of treatment (DeRubeis & Crits-Christoph 1998). Gains typically are well maintained, although return of fear (spontaneous recovery) sometimes does occur, especially when contextual cues favor the retrieval of memories associated with fear acquisition rather than extinction (Bouton 1993). Being on a psychoactive substance during exposure appears to alter those cues (Mystkowski et al. 2003), and treatment has been shown to be more enduring when provided in the absence of medications (Marks et al. 1972).

Cognitive theory posits that individuals with specific phobias perceive greater danger or risk in the feared situation than do other people and suggests that such beliefs often are activated only in the presence of the feared object (Beck & Emery 1985). Öst and colleagues have had great success with single-session cures by adjusting the nature of the exposure to test the idiosyncratic beliefs expressed by patients as they approach the object of their fears (Hellström et al. 1996, Hellström & Öst 1995, Öst et al. 1997). Other groups have obtained similar results (Thom et al. 2000, Thorpe & Salkovskis 1997). Whether this approach will enhance the stability of change relative to more purely behavioral interventions remains to be seen, but it does appear to facilitate the rapidity of change.

Obsessive-Compulsive Disorder and Personal Responsibility

Obsessive-compulsive disorder (OCD) is characterized by obsessive thoughts and images that evoke anxiety and compulsive behaviors or ritualistic mental acts that serve to reduce distress. OCD tends to be a chronic recurrent disorder. Naturalistic data suggest that full remission is rare and relapse is common after partial remission (Eisen et al. 1999); nearly half of all patients will show a chronic course (Skoog & Skoog 1999). Comparison across studies indicates that patients with OCD show a lower rate of placebo response than do patients with other types of anxiety disorders (Huppert et al. 2004).

Exposure plus response prevention (ERP) is both efficacious and specific in the treatment of OCD (DeRubeis & Crits-Christoph 1998). Although it need not be the case, ERP for this disorder typically is presented as a test of the consequences of choosing not to act to undo the obsessions, making it a largely cognitive behavioral intervention. Pharmacotherapy with the serotonin reuptake inhibitor (SRI) clomipramine and the other selective SSRIs has also been shown to be efficacious (Eddy et al. 2004). Treatment response is often quite substantial for both approaches, with up to two-thirds of all patients showing clear improvement and about one-third of all patients showing full recovery. Nonetheless, symptoms tend to persist at moderate levels for most patients, and few controlled comparisons have tracked the maintenance of gains over extended periods.

Foa & Kozak (1996) reviewed studies that examined the long-term outcome of OCD patients treated with ERP and found that more than three-quarters of all patients had maintained response up to two-and-a-half years after treatment termination. By way of contrast, the available double-blind discontinuation medication trials show a different picture (Romano et al. 2001); relapse rates often are high and typically exceed those for continuation medication (Koran et al. 2002, Pato et al. 1988, Ravizza et al. 1996). Hembree and colleagues (2003) conducted a naturalistic follow-up of patients treated with ERP, serotonergic medications, or their combination. Although differences were not evident across the whole sample, patients previously treated with ERP had less severe symptoms than did patients who were not among those not currently on medications.

Foa and colleagues (2005) provided the first direct comparison of ERP versus medications in the treatment of OCD in a study that afforded an opportunity to evaluate the stability of response following treatment termination. In that trial, adult patients with OCD of at least one-year duration were randomly assigned to ERP or clomipramine pharmacotherapy, each alone and in combination, or a pill-placebo control. Acute treatment lasted for 12 weeks and was quite intense; exposure sessions were conducted daily for the first three weeks and dosage levels were pushed aggressively (as appropriate) to a maximum of 250 mg/day. At the end of treatment, all three active treatments were superior to pill-placebo, ERP was superior to medications alone, and adding medication did little to enhance response to ERP. Response rates among all assigned were 62% for ERP, 70% for combined treatment, 42% for clomipramine alone, and 8% for placebo.

Treatment responders were then monitored over a subsequent 12-week period following the end of active treatment. ERP was discontinued and patients who responded to clomipramine (alone or in combination) were tapered off medications over a four-week period. Relapse was defined as a return to pretreatment levels of severity or a clinical state that warranted resumption of treatment. As shown in Figure 4, responders to medications alone were more likely to relapse following treatment termination than were patients previously treated with ERP alone or in combination (Simpson et al. 2004). Among treatment responders, 45% of the medication-alone patients relapsed (55% survival) relative to 12% of the patients treated with ERP (88% survival). As for depression (but unlike panic disorder), there was no indication that adding medications during acute treatment did anything to undermine the enduring effect of CBT; the rate of survival without relapse following ERP alone was 89% versus 87% for the combination.

It is unlikely that differential retention could have accounted for these findings, since clomipramine alone produced a lower rate of response than ERP alone and did little to enhance response when provided in combination. For acute treatment to act like a "differential sieve," medication treatment would have had to enhance response selectively among high-risk patients, and there is just no indication that that was the case in this trial. Response to ERP was more complete than to medication alone, meaning that patients treated with clomipramine alone required less symptom return to meet criteria for relapse. Yet, this does not undermine the validity of

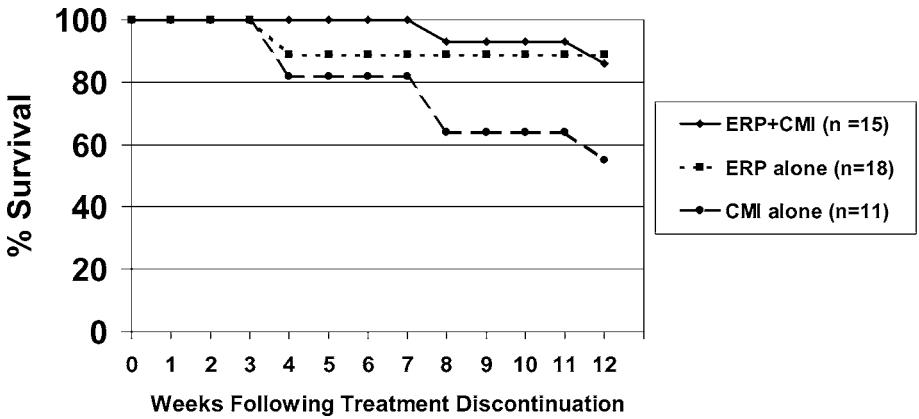

Figure 4 Time to relapse following successful treatment for obsessive-compulsive disorder with exposure plus response prevention (ERP) or clomipramine (CMI) pharmacotherapy (alone or in combination). ERP + CMI patients were previously treated with exposure plus response prevention plus clomipramine; ERP-alone patients were previously treated with exposure plus response prevention alone; CMI patients were previously treated with clomipramine alone. Adapted from Simpson et al. (2004), copyright 2004 by Wiley-Liss, Inc. Reprinted by permission.

the enduring effect (ERP may produce more complete remission than medications). Symptom severity at the end of treatment did predict subsequent risk differentially within the respective conditions; among the patients treated with ERP, relapse was largely confined to patients with more residual symptoms, whereas among the patients treated with medication alone even patients with fewer residual symptoms were at risk for relapse. This would be expected if medications suppressed symptom expression independent of individual differences in underlying risk.

The literature is mixed with respect to the relative benefits of ERP versus more purely cognitive approaches (Van Oppen et al. 1995, McLean et al. 2001, Vogel et al. 2004). Salkovskis (1999) suggests that people who are prone to OCD have exaggerated beliefs about personal responsibility and argues that making that a focus of treatment during exposure can enhance response and contribute to the stability of gains. The only trial to date to explore that approach found good maintenance of gains over a six-month follow-up, but did not provide comparisons to either more conventional ERP or medication treatment (Freeston et al. 1997). More studies are clearly needed comparing such different versions of CBT (and each to medication maintenance), but results to date suggest that CBT for OCD has an enduring effect not found for medications.

Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) involves the occurrence of a distinctive set of symptoms in response to traumatic events; symptoms include increased arousal, persistent avoidance, flashbacks and intrusive recollections, affective constriction, and a sense of interpersonal detachment. The nature of the trauma can be diverse, including sexual and other assaults, motor vehicle and other accidents, and combat trauma, with the last the most refractory to treatment (Bradley et al. 2005). Not everyone who experiences trauma develops PTSD, but for those who do, it often becomes chronic. Simple debriefing strategies applied in an unselective fashion immediately after trauma may actually increase the risk for developing PTSD (Mayou et al. 2000), but preventive interventions applied in a more selective fashion to targeted populations appear to reduce subsequent risk (Bryant et al. 1998, Foa et al. 1995).

Although PTSD tends to be a chronic condition that does not remit spontaneously once it is established, treatment with several different types of interventions appears to lead to large initial gains that are often well maintained over time (Bradley et al. 2005). Prolonged exposure (flooding), stress-inoculation training (combining relaxation training and controlled breathing with some limited cognitive restructuring), and cognitive reprocessing of the trauma have all been shown to be efficacious in the treatment of PTSD. Some studies suggest that prolonged exposure may have a more sustained effect than does stress-inoculation training (Foa et al. 1991), and that adding stress-inoculation training to prolonged exposure may undermine the enduring effects of the latter (Foa et al. 1999), but those indications are neither robust across trials nor consistent across research groups (Marks et al.

1998, Resick et al. 2002, Tarrier et al. 1999). Eye-movement desensitization and reprocessing continues to be controversial; although typically superior to control conditions (Marcus et al. 1997; Wilson et al. 1995, 1997), there is little evidence of specific efficacy and it sometimes is outperformed by other active interventions (Devilly & Spence 1999, Ironson et al. 2002, Taylor et al. 2003). ADM (especially the SSRIs) has been shown to be efficacious and is less likely to induce dependence than are minor tranquilizers or alcohol, but does nothing to reduce risk once its use is discontinued (Ballenger et al. 2004).

Resick and colleagues developed an approach called cognitive processing therapy that targets specific maladaptive beliefs related to safety, trust, and self-esteem (Resick & Schnicke 1992). In this approach, patients are encouraged to write detailed descriptions of the traumatic event and read them back to the therapist as a form of exposure, and the implications are then discussed. In a recent trial, cognitive processing therapy was found to be at least as efficacious and enduring as prolonged exposure and superior to a minimal treatment control; patients in the control condition showed comparable gains when provided subsequent active treatment (Resick et al. 2002).

Ehlers & Clark (2000) have argued that PTSD becomes persistent when individuals process trauma in a way that leads to a continued sense of current threat. As they describe, the paradox of PTSD from a cognitive perspective is that memories for prior events create a current state of anxiety, which usually implies a sense of impending threat. This sense of current threat is seen as a consequence of the combination of excessively negative appraisals of the implications of the prior trauma and a disturbance of autobiographical memory characterized by poor elaboration and perceptual priming. This model differs from earlier cognitive formulations by specifying more fully the processes contributing to the maintenance of distress and addressing the idiosyncratic nature of the appraisals made by different individuals. As such, it represents an evolution in theory that may well guide the development of the next generation of cognitive interventions.

In a recent trial, Ehlers and colleagues (2003) applied this approach to the treatment of a sample of motor vehicle accident survivors who met criteria for persistent PTSD. Potential participants were first asked to self-monitor symptoms for a three-week period, and those who fell below criteria for subsequent risk were excluded (about 12%). Those who did not recover with self-monitoring alone were then randomly assigned to treatment with this newly elaborated version of CT, a self-help booklet based on those same principles, or repeated assessments only. Treatment lasted for three months, with subsequent assessments at six and nine months following the start of treatment. Participants treated with CT were less likely to meet criteria for PTSD at the end of treatment than were participants provided with either self-help or assessment only (21% versus 79% and 72%, respectively). Although the sample as a whole continued to improve over time, CT retained its relative advantage over the other conditions; only 11% of the participants who received CT met criteria for PTSD at the nine-month follow-up relative to 61% of the self-help participants and 55% of the assessment-only controls.

These studies provide support for the notion that the effects of CBT endure in the treatment of PTSD. The absence of comparisons to efficacious medication treatments such as the SSRIs makes it hard to demonstrate this effect as persuasively as can be done for other disorders, but it is clear that the effects of treatment do not erode over time. Thus, the preponderance of evidence appears to suggest that CBT has an enduring effect in the treatment of PTSD.

CONCLUSIONS

CBT appears to have an enduring effect in the treatment of depression and the anxiety disorders that reduces risk for subsequent symptom return. This enduring effect is most evident in comparison to medication treatment, which appears to be largely palliative in nature, and represents one of the major advantages for CBT. Clear documentation exists relative to medication treatment with respect to depression, panic, social phobia, and OCD. Despite the absence of comparisons to prior medication treatment, there is also evidence of stability of gains for several of the other anxiety disorders (tests for enduring effects are largely lacking in other disorders). There are indications that adding medications may undermine the enduring effects of CBT in some instances; such interference could be a consequence of either pharmacological or psychological mechanisms and may vary as a function of medication and disorder. With the exception of family-focused treatment for bipolar disorder, there are few indications that other psychosocial interventions have enduring effects, although this possibility has rarely been explored. The nature of the underlying mechanisms remains to be determined, but CBT appears to have an enduring effect in the treatment of depression and the anxiety disorders that may preclude the need for extended medication treatment.

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LITERATURE CITED

Am. Psychiatric Assoc. 2000. Practice guideline for the treatment of patients with major depressive disorder (revision). *Am. J. Psychiatry* 157(Suppl. 4):1–45

Am. Psychiatric Assoc. 2002. Practice guideline for the treatment of patients with bipolar disorder (revised). *Am. J. Psychiatry* 159(Suppl. 4):1–50

Arntz A, Van den Hout M. 1996. Psychological treatments of panic disorder without agoraphobia: cognitive therapy versus applied relaxation. *Behav. Res. Ther.* 34:113–21

Ballenger JC, Davidson JR, Lecrubier Y, Nutt DJ, Marshall RD, et al. 2004. Consensus statement update on posttraumatic stress disorder from the International Consensus Group on Depression and Anxiety. *J. Clin. Psychiatry* 65(Suppl. 1):55–62

Bandura A. 1977. Self-efficacy: toward a unifying theory of behavioral change. *Psychol. Rev.* 84:181–215

Barber JP, DeRubeis RJ. 1989. On second thought: where the action is in cognitive therapy for depression. *Cogn. Ther. Res.* 13:441–57

Barlow DH, Gorman JM, Shear MK, Woods

SW. 2000. Cognitive-behavioral therapy, imipramine, or their combination for panic disorder: a randomized controlled trial. *JAMA* 283:2529–36

Barlow DH, Lehman CL. 1996. Advances in the psychosocial treatment of anxiety disorders. *Arch. Gen. Psychiatry* 53:727–35

Barsky AJ, Ahern DK. 2004. Cognitive behavior therapy for hypochondriasis: a randomized controlled trial. *JAMA* 291:1464–70

Beck AT. 1991. Cognitive therapy: a 30-year retrospective. *Am. Psychol*. 46:368–75

Beck AT, Emery G. 1985. *Anxiety Disorders and Phobias: A Cognitive Perspective*. New York: Basic Books

Beck AT, Rush AJ, Shaw BF, Emery G. 1979. *Cognitive Therapy of Depression*. New York: Guilford

Blackburn IM, Eunson KM, Bishop S. 1986. A two-year naturalistic follow-up of depressed patients treated with cognitive therapy, pharmacotherapy and a combination of both. *J. Affect. Disord.* 10:67–75

Blomhoff S, Haug TT, Hellström K, Holme I, Humble M, et al. 2001. Randomised controlled general practice trial of sertraline, exposure therapy and combined treatment in generalized social phobia. *Br. J. Psychiatry* 179:23–30

Borkovec TD, Costello E. 1993. Efficacy of applied relaxation and cognitive behavioral therapy in the treatment of generalized anxiety disorder. *J. Consult. Clin. Psychol.* 61: 611–19

Borkovec TD, Newman MG, Pincus AL, Lytle R. 2002. A component analysis of cognitive-behavioral therapy for generalized anxiety disorder and the role of interpersonal problems. *J. Consult. Clin. Psychol.* 70:288–98

Borkovec TD, Ruscio AM. 2001. Psychotherapy for generalized anxiety disorder. *J. Clin. Psychiatry* 62(Suppl. 11):37–42

Bouchard S, Gauthier J, LaBerge B, French D, Pelletier MH, Godbout C. 1996. Exposure versus cognitive restructuring in the treatment of panic disorder with agoraphobia. *Behav. Res. Ther.* 34:213–24

Bouton ME. 1993. Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychol. Bull.* 114:80–99

Bradley R, Greene J, Russ E, Dutra L, Westen D. 2005. A multidimensional meta-analysis of psychotherapy for PTSD. *Am. J. Psychiatry* 162:214–27

Brown TA, Barlow DH, Liebowitz MR. 1994. The empirical basis of generalized anxiety disorder. *Am. J. Psychiatry* 151:1272–80

Bruce TJ, Spiegel DA, Hegel MT. 1999. Cognitive-behavioral therapy helps prevent relapse and recurrence of panic disorder following alprazolam discontinuation: a long-term follow-up of the Peoria and Dartmouth studies. *J. Consult. Clin. Psychol.* 67:151–56

Bryant RA, Harvey AG, Dang ST, Sackville T, Basten C. 1998. Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. *J. Consult. Clin. Psychol.* 66:862–66

Butler G, Cullington A, Munby M, Amies P, Gelder M. 1984. Exposure and anxiety management in the treatment of social phobia. *J. Consult. Clin. Psychol.* 52:642–50

Clark DM. 1986. A cognitive approach to panic. *Behav. Res. Ther.* 24:461–70

Clark DM, Ehlers A, McManus F, Hackmann A, Fennell M, et al. 2003. Cognitive therapy versus fluoxetine in generalized social phobia: a randomized placebo-controlled trial. *J. Consult. Clin. Psychol.* 71:1058–67

Clark DM, Salkovskis PM, Hackmann A, Middleton H, Anastasiades P, Gelder M. 1994. A comparison of cognitive therapy, applied relaxation and imipramine in the treatment of panic disorder. *Br. J. Psychiatry* 164:759–69

Clark DM, Salkovskis PM, Hackmann A, Wells A, Fennell M, et al. 1998. Two psychological treatments for hypochondrias: a randomised controlled trial. *Br. J. Psychiatry* 173:218–25

Clarke GN, Hawkins W, Murphy M, Sheeber LB, Lewinsohn PM, Seeley JR. 1995. Targeted prevention of unipolar depressive disorder in an at-risk sample of high school adolescents: a randomized trial of a group cognitive intervention. *J. Am. Acad. Child Adolesc. Psychiatry* 34:312–21

Clarke GN, Hornbrook MC, Lynch F, Polen M, Gale J, et al. 2001. Offspring of depressed parents in a HMO: a randomized trial of a group cognitive intervention for preventing adolescent depressive disorder. *Arch. Gen. Psychiatry* 58:1127–34

Davidson JRT, Hughes DL, George LK, Blazer DG. 1993. The epidemiology of social phobia: findings from the Duke Epidemiological Catchment Area Study. *Psychol. Med.* 23: 709–18

DeRubeis RJ, Crits-Christoph P. 1998. Empirically supported individual and group psychological treatments for adult mental disorders. *J. Consult. Clin. Psychol.* 66:37–52

DeRubeis RJ, Evans MD, Hollon SD, Garvey MJ, Grove WM, Tuason VB. 1990. How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *J. Consult. Clin. Psychol.* 58:862–69

DeRubeis RJ, Feeley M. 1990. Determinants of change in cognitive therapy for depression. *Cogn. Ther. Res.* 14:469–82

DeRubeis RJ, Hollon SD, Amsterdam JD, Shelton RC, Young PR, et al. 2005. Cognitive therapy vs. medications in the treatment of moderate to severe depression. *Arch. Gen. Psychiatry* 62:409–16

Devilly GJ, Spence SH. 1999. The relative efficacy and treatment distress of EMDR and a cognitive-behavior trauma protocol in the amelioration of posttraumatic stress disorder. *J. Anxiety Disord.* 13:131–57

Dimidjian S, Hollon SD, Dobson KS, Schmaling KB, Kohlenberg RJ. 2005. Behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of major depression. *J. Consult. Clin. Psychol.* In press

Dugas MJ, Ladouceur R, Léger E, Freeston MH, Langlois F, et al. 2003. Group cognitive-behavioral therapy for generalized anxiety disorder: treatment outcome and long-term follow-up. *J. Consult. Clin. Psychol.* 71:821–25

Durham RC, Chambers JA, MacDonald RR, Power KG, Major K. 2003. Does cognitive-behaviour therapy influence the long-term outcome of generalized anxiety disorder? An 8–14-year follow-up of two clinical trials. *Psychol. Med.* 33:499–509

Eddy KT, Dutra L, Bradley R, Westen D. 2004. A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clin. Psychol. Rev.* 24:1011–30

Ehlers A, Clark DM. 2000. A cognitive model of posttraumatic stress disorder. *Behav. Res. Ther.* 38:319–45

Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M, et al. 2003. A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. *Arch. Gen. Psychiatry* 60:1024–32

Eisen JL, Goodman WK, Keller MB, Warshaw MG, DeMarco LM, et al. 1999. Patterns of remission and relapse in obsessive-compulsive disorder: a 2-year prospective study. *J. Clin. Psychiatry* 60:346–51

Evans MD, Hollon SD, DeRubeis RJ, Piasecki JM, Grove WM, et al. 1992. Differential relapse following cognitive therapy and pharmacotherapy for depression. *Arch. Gen. Psychiatry* 49:802–8

Fava GA, Grandi S, Rafanelli C, Ruini C, Conti S, Belluardo P. 2001. Long-term outcome of social phobia treated by exposure. *Psychol. Med.* 31:899–905

Fava GA, Rafanelli C, Grandi S, Conti S, Belluardo P. 1998. Prevention of recurrent depression with cognitive behavioral therapy. *Arch. Gen. Psychiatry* 55:816–20

Fava GA, Ruini C, Rafanelli C, Finos L, Salmaso L, et al. 2005. Well-being therapy of generalized anxiety disorder. *Psychother. Psychosom.* 74:26–30

Feeley M, DeRubeis RJ, Gelfand LA. 1999. The temporal relation of adherence and alliance to symptom change in cognitive therapy for depression. *J. Consult. Clin. Psychol.* 67:578–82

Foa EB, Dancu CV, Hembree EA, Jaycox LH, Meadows EA, Street GP. 1999. Comparison

of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *J. Consult. Clin. Psychol.* 67:194–200

Foa EB, Hearst-Ikeda D, Perry KJ. 1995. Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. *J. Consult. Clin. Psychol.* 63:948–55

Foa EB, Kozak MJ. 1996. Psychological treatment for obsessive-compulsive disorder. In *Long-Term Treatments of Anxiety Disorders*, ed. MR Mavissakalian, RF Prien, pp. 285–309. Washington, DC: Am. Psychiatric Press

Foa EB, Liebowitz MR, Kozak MJ, Davies S, Campeas R, et al. 2005. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *Am. J. Psychiatry* 162: 151–61

Foa EB, Rothbaum BO, Riggs DS, Murdock TB. 1991. Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. *J. Consult. Clin. Psychol.* 59:715–23

Frank E, Kupfer DJ, Perel JM, Cornes C, Jarrett DB, et al. 1990. Three-year outcomes for maintenance therapies in recurrent depression. *Arch. Gen. Psychiatry* 47:1093–99

Frank E, Prien RF, Jarrett RB, Keller MB, Kupfer DJ, et al. 1991. Conceptualization and rationale for consensus definitions of terms in major depressive disorder: remission, recovery, relapse, and recurrence. *Arch. Gen. Psychiatry* 48:851–55

Freeston MH, Ladouceur R, Gagnon F, Thibodeau N, Rheaume J, et al. 1997. Cognitive-behavioral treatment of obsessive thoughts: a controlled study. *J. Consult. Clin. Psychol.* 65:405–13

Gibbons RD, Hedeker D, Elkin I, Waternaux C, Kraemer HC, et al. 1993. Some conceptual and statistical issues in analysis of longitudinal psychiatric data: application to the NIMH Treatment of Depression Collaborative Research Program dataset. *Arch. Gen. Psychiatry* 50:739–50

Gillham JE, Shatte AJ, Freres DR. 2000. Preventing depression: a review of cognitive-behavioral and family interventions. *Appl. Prevent. Psychol.* 9:63–88

Gortner ET, Gollan JK, Dobson KS, Jacobson NS. 1998. Cognitive-behavioral treatment for depression: relapse prevention. *J. Consult. Clin. Psychol.* 66:377–84

Greenhouse JB, Stangl D, Bromberg J. 1989. An introduction to survival analysis: statistical methods for analysis of clinical trial data. *J. Consult. Clin. Psychol.* 57:536–44

Haug TT, Blomhoff S, Helstrøm IH, Holme I, Humble M, et al. 2003. Exposure therapy and sertraline in social phobia: 1-year follow-up of a randomised controlled trial. *Br. J. Psychiatry* 182:312–18

Heimberg RG, Liebowitz MR, Hope DA, Schneier FR, Holt CS, et al. 1998. Cognitive behavioral group therapy vs. phenelzine therapy for social phobia: 12-week outcome. *Arch. Gen. Psychiatry* 55:1133–41

Heimberg RG, Salzman DG, Holt CS, Blendell KA. 1993. Cognitive-behavioral group treatment for social phobia: effectiveness at five-year follow-up. *Cogn. Ther. Res.* 17:325–39

Hellström K, Fellenius J, Öst LG. 1996. One versus five sessions of applied tension in the treatment of blood phobia. *Behav. Res. Ther.* 34:101–12

Hellström K, Öst LG. 1995. One-session directed exposure vs. two forms of manual directed self-exposure in the treatment of spider phobia. *Behav. Res. Ther.* 33:959–65

Hembree EA, Riggs DS, Kozak MJ, Franklin ME, Foa EB. 2003. Long-term efficacy of exposure and ritual prevention therapy and serotonergic medications for obsessive-compulsive disorder. *CNS Spectr.* 8:363–71

Hidalgo RB, Barnett SD, Davidson JRT. 2001. Social anxiety disorder in review: two decades of progress. *Int. J. Neuropsychopharmacol.* 4:279–98

Hollon SD, Beck AT. 2004. Cognitive and cognitive-behavioral therapies. In *Garfield and Bergin's Handbook of Psychotherapy*

and Behavior Change: An Empirical Analysis, ed. MJ Lambert, pp. 447–92. New York: Wiley. 5th ed.

Hollon SD, DeRubeis RJ, Evans MD, Wiemer MJ, Garvey MJ, et al. 1992a. Cognitive therapy and pharmacotherapy for depression: singly and in combination. *Arch. Gen. Psychiatry* 49:774–81

Hollon SD, DeRubeis RJ, Seligman MEP. 1992b. Cognitive therapy and the prevention of depression. *Appl. Prevent. Psychol.* 1:89–95

Hollon SD, DeRubeis RJ, Shelton RC, Amsterdam JD, Salomon RM, et al. 2005. Prevention of relapse following cognitive therapy versus medications in moderate to severe depression. *Arch. Gen. Psychiatry* 62:417–22

Hollon SD, Evans MD, DeRubeis RJ. 1990. Cognitive mediation of relapse prevention following treatment for depression: implications of differential risk. In *Psychological Aspects of Depression*, ed. RE Ingram, pp. 117–36. New York: Plenum

Hollon SD, Thase ME, Markowitz JC. 2002. Treatment and prevention of depression. *Psychol. Sci. Public Interest* 3:39–77

Huppert JD, Schultz LT, Foa EB, Barlow DH, Davidson JRT, et al. 2004. Differential response to placebo among patients with social phobia, panic disorder, and obsessive-compulsive disorder. *Am. J. Psychiatry* 161: 1485–87

Imber SD, Pilkonis PA, Sotsky SM, Elkin I, Watkins JT, et al. 1990. Mode-specific effects among three treatments for depression. *J. Consult. Clin. Psychol.* 58:352–59

Ironson G, Freund B, Strauss JL, Williams J. 2002. Comparison of two treatments for traumatic stress: a community-based study of EMDR and prolonged exposure. *J. Clin. Psychol.* 58:113–28

Jacobson NS, Dobson KS, Truax PA, Addis ME, Koerner K, et al. 1996. A component analysis of cognitive-behavioral treatment for depression. *J. Consult. Clin. Psychol.* 64:295–304

Jacobson NS, Hollon SD. 1996. Prospects for future comparisons between drugs and psychotherapy: lessons from the CBT-versus-pharmacotherapy exchange. *J. Consult. Clin. Psychol.* 64:104–8

Jacobson NS, Martell CR, Dimidjian S. 2001. Behavioral activation treatment for depression: returning to contextual roots. *Clin. Psychol. Sci. Pract.* 8:255–70

Jaycox LH, Reivich KJ, Gillham J, Seligman MEP. 1994. Prevention of depressive symptoms in school children. *Behav. Res. Ther.* 32:801–16

Klein DF. 1996. Preventing hung juries about therapy studies. *J. Consult. Clin. Psychol.* 64:74–80

Klerman GL, DiMascio A, Weissman M, Prusoff B, Paykel ES. 1974. Treatment of depression by drugs and psychotherapy. *Am. J. Psychiatry* 131:186–91

Klerman GL, Weissman M, Rounsaville BJ, Chevron ES. 1984. *Interpersonal Psychotherapy of Depression*. New York: Basic Books

Koran LM, Hackett F, Rubin A, Wolkow R, Robinson D. 2002. Efficacy of sertraline in the long-term treatment of obsessive-compulsive disorder. *Am. J. Psychiatry* 159: 88–95

Kovacs M, Rush AT, Beck AT, Hollon SD. 1981. Depressed outpatients treated with cognitive therapy or pharmacotherapy: a one-year follow-up. *Arch. Gen. Psychiatry* 38: 33–39

Ladouceur R, Dugas MJ, Freeston MH, Leger E, Gagnon F, Thibodeau N. 2000. Efficacy of a cognitive-behavioral treatment for generalized anxiety disorder: evaluation in a controlled clinical trial. *J. Consult. Clin. Psychol.* 68:957–64

Ladouceur R, Dugas MJ, Freeston MH, Rheaume J, Blais F, et al. 1999. Specificity of generalized anxiety disorder symptoms and processes. *Behav. Ther.* 30:191–207

Lam DH, Hayward P, Watkins ER, Wright K, Sham P. 2005. Relapse prevention in patients with bipolar disorder: cognitive therapy outcome after 2 years. *Am. J. Psychiatry* 162:324–29

Lam DH, Watkins ER, Hayward P, Bright J, Wright K, et al. 2003. A randomized controlled study of cognitive therapy for relapse prevention for bipolar affective disorder: outcome of the first year. *Arch. Gen. Psychiatry* 60:145–52

Liebowitz MR, Heimberg RG, Schneier FR, Hope DA, Davies S, et al. 1999. Cognitive-behavioral group therapy versus phenelzine in social phobia: long-term outcome. *Depress. Anxiety* 10:89–98

Loerch B, Graf-Morgenstern M, Hautzinger M, Schlegel S, Hain C, et al. 1999. Randomized placebo-controlled trial of moclobemide, cognitive-behavioural therapy and their combination in panic disorder with agoraphobia. *Br. J. Psychiatry* 174:205–12

Lydiard RB, Brawman-Mintzer O, Ballenger JC. 1996. Recent development in the psychopharmacology of anxiety disorders. *J. Consult. Clin. Psychol.* 64:660–68

Malkoff-Schwartz S, Frank E, Anderson B, Sherrill JT, Siegel L, et al. 1998. Stressful life events and social rhythm disruption in the onset of manic and depressive bipolar episodes: a preliminary investigation. *Arch. Gen. Psychiatry* 55:702–7

Marcus SV, Marquis P, Sakal C. 1997. Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy* 34:307–15

Marks I, Lovell K, Noshirvani H, Livanou M, Thrasher S. 1998. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. *Arch. Gen. Psychiatry* 55:317–25

Marks IM, Swinson RP, Basoglu M, Kuch K, Noshirvani H, O'Sullivan G. 1993. Alprazolam and exposure alone and combined in panic disorder with agoraphobia: a controlled study in London and Toronto. *Br. J. Psychiatry* 162:776–87

Marks IM, Viswanathan R, Lipsedge MS, Gardner R. 1972. Enhanced relief of phobias following flooding during waning diazepam. *Br. J. Psychiatry* 121:493–505

Martell CR, Addis ME, Jacobson NS. 2001. *Depression in Context: Strategies for Guided Action*. New York: Norton

Mayou RA, Ehlers A, Hobbs M. 2000. Psychological debriefing for road traffic accident victims. *Br. J. Psychiatry* 176:589–93

McLean PD, Whittal ML, Thordarson DS, Taylor S, Sochting I, et al. 2001. Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *J. Consult. Clin. Psychol.* 69:205–14

Miklowitz DJ, George EL, Richards JA, Simoneau TL, Suddarth RL. 2003. A randomized study of family-focused psychoeducation and pharmacotherapy in the outpatient management of bipolar disorder. *Arch. Gen. Psychiatry* 60:904–12

Mowrer OH. 1948. Learning theory and the neurotic paradox. *Am. J. Orthopsychiatry* 18:571–610

Mystkowski JL, Mineka S, Vernon LL, Zinbarg RE. 2003. Changes in caffeine states enhance return of fear in spider phobia. *J. Consult. Clin. Psychol.* 71:243–50

Nathan PE, Gorman JM, eds. 1998. *A Guide to Treatments That Work*. New York: Oxford Univ. Press

Öst LG, Ferebee I, Furmark T. 1997. One-session group therapy of spider phobia: direct versus indirect treatments. *Behav. Res. Ther.* 35:721–32

Öst LG, Westling BE. 1995. Applied relaxation vs. cognitive behavior therapy in the treatment of panic disorder. *Behav. Res. Ther.* 33: 145–58

Otto MW, Pollack MH, Sachs GS, Reiter SR, Meltzer-Brody S, Rosenbaum JF. 1993. Discontinuation of benzodiazepine treatment: efficacy of cognitive-behavioral therapy for patients with panic disorder. *Am. J. Psychiatry* 150:1485–90

Pato MT, Zohar-Kadouch R, Zohar J, Murphy DL. 1988. Return of symptoms after discontinuation of clomipramine in patients with obsessive-compulsive disorder. *Am. J. Psychiatry* 145:1521–25

Paykel ES, Scott J, Teasdale JD, Johnson AL, Garland A, et al. 1999. Prevention of relapse

in residual depression by cognitive therapy. *Arch. Gen. Psychiatry* 56:829–35

Perlis RH, Nierenberg AA, Alpert JE, Pava J, Matthews JD. 2002. Effects of adding cognitive therapy to fluoxetine dose increase on risk of relapse and residual depressive symptoms in continuation treatment of major depressive disorder. *J. Clin. Psychopharmacol* 22:474–80

Perry A, Tarrier N, Morriss R, McCarthy E, Limb K. 1999. Randomised controlled trial of efficacy of teaching patients with bipolar disorder to identify early symptoms of relapse and obtain treatment. *Br. Med. J.* 318:149–53

Ravizza L, Barzega G, Bellino S, Bogetto F, Maina G. 1996. Drug treatment of obsessive-compulsive disorder (OCD): long-term trial with clomipramine and selective serotonin reuptake inhibitors (SSRIs). *Psychopharmacol. Bull.* 32:167–73

Resick PA, Nishith P, Weaver TL, Astin MC, Feuer CA. 2002. A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *J. Consult. Clin. Psychol.* 70:867–79

Resick PA, Schnicke MK. 1992. Cognitive processing therapy for sexual assault victims. *J. Consult. Clin. Psychol.* 60:748–56

Romano S, Goodman W, Tamura R, Gonzales J. 2001. Long-term treatment of obsessive-compulsive disorder after an acute response: a comparison of fluoxetine versus placebo. *J. Clin. Psychopharmacol.* 21:46–52

Roth A, Fonagy P. 2005. *What Works for Whom? A Critical Review of Psychotherapy Research*. New York: Guilford. 2nd ed.

Salkovskis PM. 1999. Understanding and treating obsessive-compulsive disorder. *Behav. Res. Ther.* 37(Suppl. 1):S29–52

Seligman MEP, Schulman P, DeRubeis RJ, Hollon SD. 1999. December 21. The prevention of depression and anxiety. *Prevent. Treat.* 2:Article 8. Retrieved July 4, 2002, from http://journals.apa.org/prevention/volume2/pre0020008a.html

Sharp DM, Power KG, Simpson RJ, Swanson V, Moodie E, et al. 1996. Fluvoxamine, placebo and cognitive behavior therapy used alone and in combination in the treatment of panic disorder and agoraphobia. *J. Anxiety Disord.* 10:219–42

Shea MT, Elkin I, Imber SD, Sotsky SM, Watkins JT, et al. 1992. Course of depressive symptoms over follow-up: findings from the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *Arch. Gen. Psychiatry* 49:782–87

Simons AD, Garfield SL, Murphy GE. 1984. The process of change in cognitive therapy and pharmacotherapy for depression. *Arch. Gen. Psychiatry* 41:45–51

Simons AD, Murphy GE, Levine JL, Wetzel RD. 1986. Cognitive therapy and pharmacotherapy for depression: sustained improvement over one year. *Arch. Gen. Psychiatry* 43:43–48

Simpson HB, Liebowitz MR, Foa EB, Kozak MJ, Schmidt AB, et al. 2004. Post-treatment effects of exposure therapy and clomipramine in obsessive-compulsive disorder. *Depress. Anxiety* 19:225–33

Skoog G, Skoog I. 1999. A 40-year follow-up of patients with obsessive-compulsive disorder. *Arch. Gen. Psychiatry* 56:121–27

Stangier U, Heidenreich T, Peitz M, Lauterbach W, Clark DM. 2003. Cognitive therapy for social phobia: individual versus group treatment. *Behav. Res. Ther.* 41:991–1007

Stanley MA, Beck JG, Novy DM, Averill PM, Swann AC, et al. 2003. Cognitive-behavioral treatment of late-life generalized anxiety disorder. *J. Consult. Clin. Psychol.* 71:309–19

Tang TZ, DeRubeis RJ. 1999. Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *J. Consult. Clin. Psychol.* 67:894–904

Tarrier N, Pilgrim H, Sommerfield C, Faragher B, Reynolds M, et al. 1999. A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. *J. Consult. Clin. Psychol.* 67:13–18

Taylor S, Thordarson DS, Maxfield L, Fedoroff IC, Lovell K, Ogrodniczuk J. 2003. Comparative efficacy, speed, and adverse effects of three PTSD treatments: exposure therapy, EMDR, and relaxation therapy. *J. Consult. Clin. Psychol.* 71:330–38

Teasdale JD, Scott J, Moore RG, Hayhurst H, Pope M, Paykel ES. 2001. How does cognitive therapy prevent relapse in residual depression: evidence from a controlled trial. *J. Consult. Clin. Psychol.* 69:347–57

Teasdale JD, Segal Z, Williams JMG, Ridgeway VA, Soulsby JM, Lau MA. 2000. Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *J. Consult. Clin. Psychol.* 68:615–23

Thom A, Sartory G, Hohren P. 2000. Comparison between one-session psychological treatment and benzodiazepine in dental phobia. *J. Consult. Clin. Psychol.* 68:378–87

Thorpe SJ, Salkovskis PM. 1997. The effect of one-session treatment for spider phobia on attentional bias and beliefs. *Br. J. Clin. Psychol.* 36:225–41

Van Oppen P, de Haan E, van Balkom A, Spinhoven P, Hoogduin K, van Dyck R. 1995. Cognitive therapy and exposure in vivo in the treatment of obsessive-compulsive disorder. *Behav. Res. Ther.* 33:379–90

Vogel PA, Stiles TC, Götestam KG. 2004. Adding cognitive therapy elements to exposure therapy for obsessive-compulsive disorder: a controlled study. *Behav. Cogn. Psychother.* 32:275–90

Warwick HMC, Clark DM, Cobb AM, Salkovskis PM. 1996. A controlled trial of cognitive-behavioural treatment of hypochondriasis. *Br. J. Psychiatry* 169:189–95

Warwick HMC, Salkovskis PM. 1990. Hypochondriasis. *Behav. Res. Ther.* 28:105–17

Weissman MM, Klerman GL, Prusoff B, Sholomskas D, Padian N. 1981. Depressed outpatients: results one year after treatment with drugs and/or interpersonal therapy. *Arch. Gen. Psychiatry* 38:51–55

Wells A, Clark DM, Salkovskis P, Ludgate J, Hackmann A, Gelder M. 1995. Social phobia: the role of in-situation safety behaviours in maintaining anxiety and negative beliefs. *Behav. Ther.* 26:153–61

Willett JB, Ayoub CC, Robinson D. 1991. Using growth modeling to examine systematic differences in growth: an example of change in functioning of families at risk of maladaptive parenting, child abuse, or neglect. *J. Consult. Clin. Psychol.* 59:38–47

Willett JB, Singer JD. 1993. Investigating onset, cessation, relapse, and recovery: Why you should, and how you can, use discrete-time survival analysis to examine event occurrence. *J. Consult. Clin. Psychol.* 61:952–65

Wilson SA, Becker LA, Tinker RH. 1995. Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *J. Consult. Clin. Psychol.* 63:928–37

Wilson SA, Becker LA, Tinker RH. 1997. Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. *J. Consult. Clin. Psychol.* 65:1047–56

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