Do we need to challenge thoughts in cognitive behavior therapy?

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Abstract

Cognitive behavior therapy (CBT) emphasizes the primacy of cognition in mediating psychological disorder. It aims to alleviate distress by modifying cognitive content and process, realigning thinking with reality. Recently, various authors have questioned the need for CBT therapists to use logico-rational strategies to directly challenge maladaptive thoughts. Hayes [Hayes, S.C. (2004). Acceptance and commitment therapy and the new behavior therapies. In S.C. Hayes, V.M. Follette, & M.M. Linehan (Eds.), *Mindfulness and acceptance: Expanding the cognitive behavioral tradition.* (pp. 1–29). New York: Guilford] has identified three empirical anomalies in the research literature. Firstly, treatment component analyzes have failed to show that cognitive interventions provide significant added value to the therapy. Secondly, CBT treatments have been associated with a rapid symptomatic improvement prior to the introduction of specific cognitive interventions. Thirdly, there is a paucity of data that changes in cognitive mediators instigate symptomatic change. This paper critically reviews the empirical literature that addresses these significant challenges to CBT. A comprehensive review of component studies finds little evidence that specific cognitive interventions significantly increase the effectiveness of the therapy. Although evidence for the early rapid response phenomenon is lacking, there is little empirical support for the role of cognitive change as causal in the symptomatic improvements achieved in CBT. These findings are discussed with reference to the key question: Do we need to challenge thoughts in CBT?
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1. Introduction

In his 1995 paper, David A. Clark set out to define the principles that distinguish cognitive therapy from other therapeutic approaches:

"The therapist and patient collaborate to identify distorted cognitions, which are derived from maladaptive beliefs or assumptions. These cognitions and beliefs are subjected to *logical analysis* and *empirical hypothesis-testing* which leads individuals to realign their thinking with reality." (Clark, 1995; p. 155, emphases in original).

Clark, in common with other leading cognitive therapists including Aaron T. Beck (Beck, 1970; DeRubeis, Tang, & Beck, 2001), asserts that a fundamental postulate of the cognitive model of psychopathology is that cognitive change is central to treating psychological disorder, stating that "all therapies work by altering dysfunctional cognitions, either directly or indirectly" (p. 158). Hence, modification of maladaptive cognition is both the process by which cognitive therapy is effective, as well as the mechanism of change in psychotherapy more generally.

In line with this fundamental postulate, the authors of treatment manuals for cognitive behavior therapy (CBT) invariably describe techniques for modifying the meaning of thoughts (e.g.,Beck, Rush, Shaw, & Emery 1979; Beck, 1995). Hackmann, (1997), in common with these authors, draws attention to specific techniques for challenging the meaning of dysfunctional thoughts on the basis of their internal logic: for example, evaluating the evidence for and against a thought using written thought records, eliciting more realistic thoughts and looking for evidence of distorted thinking.

As well as distinguishing CBT theory, this emphasis on working with dysfunctional cognitions defines what cognitive therapists actually do in their therapy sessions. Blagys and Hilsenroth (2002) conducted a review of the psychotherapy process literature and found that evaluating, challenging and modifying thoughts was one of the hallmarks that distinguished CBT practice from that of other therapies.

However, is the direct, explicit modification of maladaptive cognitions a necessary or sufficient intervention in CBT? Hayes (2004) identified three "empirical anomalies" in the CBT outcome literature. First, component analyzes do not show that cognitive interventions provide added value to the therapy. Second, CBT treatment is often associated with a rapid, early improvement in symptoms that most likely occurs before the implementation of any distinctive cognitive techniques. Third, measured changes in cognitive mediators (the thoughts and beliefs held by the cognitive model to underpin disorder) do not seem to precede changes in symptoms. In the same vein, Orsillo, Roemer, Lerner, and Tull (2004) note that a problem in evaluating the mechanisms of change in cognitive behavior therapy is that CBT is "a general label for a variety of techniques, any of which may actually be the active ingredient of treatment" (p. 71).

The logical, "rationalist" approach to modifying cognition has also been a subject to critical reappraisal at a theoretical level by researchers proposing that multi-level cognitive architectures provide a more accurate description of human cognition. For example, Brewin, in his recent M.B. Shapiro Award Lecture (Lawson, 2005), questions the proposition that challenging thoughts leads to changes in feelings and behaviors. Drawing on the findings of cognitive science, he proposes that human cognition comprises multiple memory systems and knowledge stores, not all of which are open to introspection. Further, he suggests that these multiple systems give rise to multiple self-representations. He concludes that therapy is better employed in a constructivist strengthening of more helpful representations, rather than the logico-deductive challenging of unhelpful representations. Likewise, Teasdale (1997) contrasts "propositional" meanings (which are semantic and declarative) with "implicational" meanings (implicit, holistic meanings which reflect the "felt sense" of experience and are closely linked to emotion). For Teasdale (1997) implicational beliefs "do not have a specific truth value that can be assessed" (p. 152). Therefore, therapy should focus on changing the client's "actual way of *being*" (p. 150) rather than aiming at logically challenging beliefs. For Teasdale, there is little value in

exposing the logical flaws in the client's thought processes: to do so is merely to focus on semantic, declarative meanings without engaging emotional processes.

These are provocative critiques. CBT is rightly proud of its tradition as an empirically based therapy. It seeks to modify and improve interventions on the basis of their demonstrable effectiveness. This paper investigates the "empirical anomalies" identified by Hayes (2004) in order to determine whether they call into question some of the fundamental tenets of the therapy. It will examine what the research literature tells us about the value of the cognitive change procedures that form an explicit part of the therapy, as well as the evidence pertaining to the implicit role of cognitive modification as the mediating mechanism of symptomatic improvement. To what extent does effectiveness of CBT depend upon direct cognitive interventions? Does therapeutic change rely on cognitive change? In short, do we need to challenge thoughts in CBT?

2. CBT component analysis studies

Studies which attempt to split the therapeutic elements comprising CBT and deliver separate components of the therapy either to different groups (between-subjects designs) or to the same participants in sequence (within-subjects designs) provide useful evidence about the effectiveness of specific CBT interventions. A literature search for such studies was undertaken using the PsychInfo and Medline databases, limited to publications in English since 1980. References from studies elicited by this method were also followed up. A summary of the relevant CBT component analysis studies is given in Table 1.

2.1. Component analysis studies for depression

Perhaps the most significant component analysis study to have examined the active elements of CBT is that of Jacobson et al. (1996). Indeed, this particular paper is cited by Hayes (2004) to support his conclusion that the cognitive components of CBT do not actually add to the effectiveness of the treatment.

Table 1
CBT component analysis studies

Study	Disorder/number of participants	Components examined	Findings
Jacobson et al. (1996)	Depression *N*=152	BA v AT v CT	No significant difference in outcome between conditions at termination or 6 month follow-up.
Gortner et al. (1998)	Depression *N*=137	BA v AT v CT	2-year follow-up of above study (Jacobson et al., 1996). No significant difference in relapse rates between conditions.
Dimidjian et al. (2003)	Depression *N*=500	BA v CT v medication v pill placebo	At termination: BA=Medication. BA/Medication> CT. CT=Pill placebo.
Zettle and Hayes (1987)	Depression *N*=12	Cognitive restructuring v behavioral homework	No significant difference in outcome between conditions.
Jarrett and Nelson (1987)	Depression *N*=37	Logical analysis v hypothesis testing	No significant difference in outcome between conditions.
Borkovec et al. (2002)	Generalized anxiety disorder *N*=69	CT v SCD v CBT	No significant difference in outcome between conditions at termination and 6, 12, and 24-month follow-up.
Tarrier et al. (1999)	Post traumatic stress disorder *N*=72	IE v CT	No significant difference in outcome between conditions at termination.
Lovell, Marks, Noshirvani, Thrasher and Livanou (2001)	Post traumatic stress disorder *N*=77	Exposure v cognitive restructuring v combined	No significant difference in outcome between conditions at termination and 1, 3 and 6 month follow-up.
Emmelkamp et al. (1985)	Social phobia *N*=34	RET v SIT v exposure	No significant difference in outcome between conditions.
Mattick et al. (1989)	Social phobia *N*=43	Exposure v cognitive restructuring v combined	No significant difference in outcome between conditions at termination or 3 month follow-up.
van Oppen et al. (1995)	OCD *N*=71	CT v ERP	No significant difference in outcome between conditions.
Emmelkamp and Beens (1991)	OCD *N*=21	RET v ERP	No significant difference in outcome between conditions.
de Haan et al. (1997)	OCD *N*=99	CT v ERP	No significant difference in outcome between conditions.

AT = Automatic Thoughts; BA = Behavioral Activation; CBT = Cognitive Behavior Therapy; CT = Cognitive Therapy; ERP = Exposure and Response Prevention; IE = Imaginal Exposure; RET = Rational Emotive Therapy; SCD = Self Control Desensitisation; SIT = Self-Instructional Training.

The participants in the Jacobson et al. (1996) study comprised 152 people who met the Diagnostic and Statistical Manual — Third Edition (Revised) criteria for major depression (DSM-IIIR; American Psychiatric Association, 1987). They were randomly allocated to one of three treatment conditions: Behavioral Activation (BA), Automatic Thoughts (AT), and Cognitive Therapy (CT). Treatment protocols were produced for each of the conditions. The main elements of each treatment are described as follows:

2.1.1. Behavioral activation

- Monitoring daily activities and assessing the pleasure and mastery involved.
- Assigning new daily activities to increase pleasure and mastery.
- Imaginal rehearsal of activities before they are undertaken.
- Problem solving any obstacles to undertaking new activities.
- Interventions to address social skills deficits such as assertiveness and communication training.

2.1.2. Automatic thoughts

This treatment condition comprised the above elements of the activation condition, plus techniques designed to assess and modify negative automatic thoughts as follows:

- Identifying automatic thoughts arising in-session.
- Use of Daily Thought Records.
- Examining evidence for and against automatic thoughts.
- Examining attributional biases in the way participants assess their successes and failures.
- Homework assignments in which participants assess the validity of their negative interpretations.

2.1.3. Cognitive therapy

This condition comprised the elements of the above two conditions, plus techniques designed to modify dysfunctional assumptions and core beliefs as follows:

- Using the 'downward arrow' technique and discussion to identify core beliefs.
- Identifying the advantages and disadvantages of core beliefs and assumptions; identifying alternatives.
- Homework exploring the operation of core beliefs and assumptions and applying alternative beliefs.

Treatment comprised 20 sessions. For all three conditions, treatment was provided by four trained cognitive therapists with a range of 8 to 12 years post-qualification experience, all of whom had experience in at least one previous clinical trial for cognitive therapy. Indeed, Jacobson and Gortner (2000) note that the therapists in the trial all had an allegiance to CT. Protocol adherence in the treatment conditions (for example, ensuring that AT techniques did not 'leak' into the BA treatment condition) was monitored by randomly selecting tapes of 20% of the sessions and blind rating them for their use of all the techniques from the three conditions. Adherence proved to be high: for example, in the BA condition, BA interventions were rated as ten times more prevalent than AT or CT interventions combined.

Outcome, as measured by the Beck Depression Inventory (BDI; Beck et al., 1979) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967), showed no significant differences between the conditions, either at the conclusion of treatment or at 6-month follow-up. For example, in the BA condition ($n = 56$) mean BDI pre-treatment was 29.3, which had reduced to 9.1 post-treatment. In the AT condition ($n = 43$), mean BDI was 29.1 pre-treatment, compared with 9.3 post-treatment, while for the CT condition ($n = 50$), the comparable figures were 29.8 and 10.3, respectively. Gortner, Gollan, Jacobson and Dobson (1998) provide two-year follow-up data for the study's participants, which show no significant differences between treatment conditions in terms of relapse rates. Therefore, it was concluded that behavioral activation alone was as effective in treating depression as BA combined with cognitive interventions.

Dobson and Khatri, (2000) consider that the Jacobson et al. (1996) study has potentially serious implications for both the theory and practice of CBT for depression. In practical terms, behavioral activation is simpler and more cost effective, both in the training of therapists and delivery to patients. Further, they suggest that efficacy of behavioral

interventions in the trial must lead to doubt regarding the significance of cognitive factors in the etiology and maintenance of depression.

However, findings of the Jacobson et al. (1996) study need to be qualified in several respects. Despite being a component study, the treatment conditions remained fairly broad and complex. It could be argued that the BA condition contained cognitive elements in its protocol: for example, the imaginal rehearsal of activities and problem solving of obstacles may entail the modification of dysfunctional thoughts and assumptions. In addition, it should be borne in mind that the AT and CT conditions were not *less* effective than BA, but rather equally effective — a finding that would not be expected if cognition played no role in the maintenance of depression.

The findings of Jacobson et al. run so radically counter to the CBT paradigm, that Jacobson and Gortner (2000) report they gave rise to questions in the research community regarding the quality of the cognitive therapy provided in the study. However, they point out that the recovery rates in the study (about 60% of participants in each condition no longer meeting diagnostic criteria at termination) compare favorably with those of previous outcome studies of CBT for depression. Therefore, the case at issue is not that CT performed poorly, but rather that BA performed so well. Despite this, it was generally recognized that such potentially important results require replication. Therefore a further research program–the University of Washington Treatment for Depression Study– was instigated.

This new study has been running since 1997. The results have not yet been formally subjected to peer review and journal publication. However, some details of the design are given in Jacobson and Gortner (2000). Rather than being conceptualized as component research comparing the elements of CBT, the University of Washington study conceives of behavioral activation as a separate treatment in its own right. The aim of BA for depression is to increase access to environmental "anti-depressant reinforcers." Jacobson and Gortner give examples of what this might mean in practice: "People who have lost their jobs are aided in finding employment. Those who have lost their friends or lovers are aided in finding new people. Those with relationships that have gone sour are aided in fixing them." (Jacobson & Gortner 2000, p. 113). BA is compared in the new study with CT, pharmacotherapy and pill placebo conditions. According to Jacobson and Gortner, the three active treatment conditions are being administered and supervised by advocates of each approach who are experts in their field. Jacobson and Gortner state the intention to enter 500 participants into the trial.

Members of the study's research team have presented some of the acute phase results at conferences (Dimidjian et al., 2003). These findings are also alluded to in Martell, Addis and Dimidjian (2004). Here, it is stated that BA proved as effective as antidepressant medication, and that both produced superior outcomes to cognitive therapy, which was no more effective than the pill placebo condition (Martell et al., 2004, p. 155). Given that the Washington University study purports to be the largest outpatient therapy trial for depression yet undertaken, these would appear to be perplexing results for the proponents of cognitive therapy as a treatment for depression. However, putting aside the comparison with BA, the Washington results would seem to contradict many previous studies which have shown CT to be equally effective as pharmacotherapy as a treatment for moderate depression (e.g.,Hollon et al., 1992; Murphy, Simons, Wetzel, & Lustman, 1984) and severe depression (DeRubeis, Gelfand, Tang, & Simons, 1999; Hollon & DeRubeis, 2004). Therefore, it will be necessary to wait for the publication of the study's data before its full implications can be assessed.

Two studies have sought to compare the components of CBT for depression by delivering them not to separate groups of participants, but instead to the same participants in separate phases of treatment. In the first such study, Zettle and Hayes (1987) compared cognitive restructuring with behavioral homework for 12 people with depression. Time series analyzes revealed no superiority for either element. Further, the order in which the components were introduced did not influence effectiveness. Unfortunately, difficulty in interpreting these results comes from Zettle and Hayes' decision to incorporate an initial treatment component — "distancing," which they describe as helping participants to recognize that depressogenic beliefs are hypotheses rather than facts, through the use of similes and reattribution techniques. For all participants, distancing came before the behavioral and cognitive components were introduced. Therefore, the "distancing" intervention (which is ostensibly cognitive) may have blunted the impact of the study's overtly cognitive phase of treatment. The second study, reported in Jarrett and Nelson (1987), compared the effectiveness of logically analysing dysfunctional thoughts with that of behavioral experiments in 37 depressed subjects in group treatment across 12 sessions. Once again, there was no difference in outcome between the two classes of intervention.

2.2. Component analysis studies of anxiety disorders

In the light of Jacobson et al's findings, Dobson and Khatri, (2000) called for component analysis research into the active elements of CBT for disorders other than depression. In particular, they noted that such research should address whether the cognitive components of CBT contributed to the efficacy of treatment for the anxiety disorders, above and beyond exposure to anxiety-provoking stimuli. There is a scattering of studies in the research literature that allow such a comparison. These will now be considered.

Borkovec and Costello (1993) had previously found that a CBT package comprising applied relaxation, self-control desensitisation and brief cognitive therapy proved an effective treatment for generalized anxiety disorder (GAD). Consequently, Borkovec, Newman, Pincus and Lytle (2002) set out to find the active component of this package by comparing cognitive therapy (CT) with self-control desensitisation (SCD) and a condition in which the two elements were combined (CBT). What is illuminating about this study is that the component treatment conditions were relatively "pure." The CT condition examined worry thoughts through logical analysis, examination of evidence and probability estimates, labelling logical errors, decatastrophizing and identifying alternative thoughts and beliefs. The SCD condition comprised progressive relaxation training and the imaginal rehearsal of anxiety-provoking situations in order to establish cued relaxation. For Borkovec et al., the CT condition targeted the cognitive response system, while the SCD condition aimed at ameliorating physiological responsiveness.

Sixty-nine participants meeting diagnostic criteria for GAD received 14 sessions of treatment. At termination and at a 2-year follow-up, all the treatment conditions had proven to be equally effective on a comprehensive battery of anxiety measures. From this result, Borkovec et al. concluded that "targeting some response processes in therapy for a sufficiently long period of time might therefore affect all of the other processes involved in the maintenance of anxiety" (p. 296). Therefore, this study provides evidence that challenging thoughts is potentially effective for GAD, but no more effective than a desensitisation procedure, and certainly not a necessary element of treatment. Corollary evidence to this effect is provided by Öst and Breitholtz (2000), who compared applied relaxation to cognitive therapy for GAD (the CT condition incorporated behavioral experiments — the study was not intended as a component analysis comparing cognitive with behavioral elements of treatment) and found no difference between outcomes on any measure for their 36 participants, either at the termination of treatment or a 1-year follow-up.

Two component studies of posttraumatic stress disorder (PTSD) have sought to separate exposure from cognitive interventions in order to specify the active element of treatment. Tarrier et al. (1999) compared the effectiveness of imaginal exposure and cognitive therapy in a between-subjects design with 72 participants who had been diagnosed on the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990). The cognitive therapy condition involved looking at beliefs regarding the meaning of the trauma event and the attributions made following it. Therapists in this condition avoided discussion of the trauma itself in order to prevent inadvertent exposure, although they had access to trauma narratives from the assessment phase of the study. The aim of the CT condition was to focus on the interpretation of the event rather than the event itself. In contrast, the imaginal exposure condition involved describing the trauma event as if it was happening, while attempting to visualise it. This condition precluded interventions aimed at cognitive restructuring. Participants received 16 sessions of therapy, and there were no significant differences on outcome measures between the two conditions at the termination of treatment or 6-month follow-up, with one exception: although both conditions provided equal levels of clinical improvement, a significantly greater minority of participants experienced a worsening of symptoms at the termination of imaginal exposure than CT (9 participants in the IE condition, compared with 3 in the CT condition). This difference was no longer evident by the 6-month follow-up.

Lovell et al. (2001) also compared imaginal exposure with cognitive restructuring for PTSD, but incorporated a treatment condition that combined these elements. They hypothesised that the different treatment conditions might impact differently on the specific symptom clusters of PTSD, with exposure having greatest impact on anxiety-based symptoms such as re-experiencing and avoidance, while cognitive restructuring would produce greater change in appraisal-related features such as numbing, detachment and guilt. At termination, there were no significant differences between the treatment conditions, either in terms of overall outcome or differential impact on specific symptoms.

Two studies have compared the effectiveness of purely cognitive interventions with that of exposure alone in the treatment of social phobia. Emmelkamp, Mersch, Vissia and van der Helm (1985) evaluated rational emotive therapy (RET) against self-instructional training and exposure *in vivo* for 34 participants in a between-subjects design. After six 2 1/2 h group treatment sessions, there were no significant differences between the RET and

exposure groups on a range of outcome measures. Mattick, Peters and Clarke (1989) compared exposure *in vivo* with cognitive restructuring and a condition that combined the two treatments. Treatments were administered in a group format. Again, there were no significant differences between the treatment conditions in terms of endstate functioning or clinical improvement, either at the termination of treatment or at 3-month follow-up, although the group receiving cognitive restructuring alone showed significantly greater gains on two subscales of the Fear Questionnaire (Marks & Mathews, 1979).

More commonly, studies of social phobia have compared full CBT treatment protocols with exposure alone. Three have reported superior outcomes for CBT. Firstly, Butler, Cullington, Munby, Amies and Gelder (1984) compared self-directed exposure with exposure plus anxiety management (AM). The AM package comprised relaxation and distraction techniques along with rational self-talk. Addition of AM produced a significantly superior outcome on measures of social avoidance. However, the heterogeneity of the AM treatment, along with the extreme brevity of the cognitive component (participants received just 2 1/2 h of AM training in total), makes it impossible to identify the active element of treatment. Secondly, Mattick and Peters (1988) compared group CBT with exposure *in vivo*. CBT participants performed significantly better on a behavioral test involving exposure to situations derived from individually constructed fear hierarchies. Further, 48% of the exposure group reported ongoing avoidance of phobic situations at a 3-month follow-up, compared with 14% of the CBT group. However, using precisely the same treatment protocols, therapists and measures, the study by Mattick et al. (1989) failed to replicate these findings. Finally, Hofmann (2004) compared group CBT and exposure *in vivo*, finding that the CBT group scored significantly lower on the Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989) at a 6-month follow-up. However, Hofmann notes the limited scope of the exposure exercises in his study: "Although participants feared numerous social situations, this intervention focused primarily on the patients' public speaking anxiety" (Hofmann, 2004, p. 393). Sub-optimal exposure, delivered in a group format, may have prevented the generalization of gains to other phobic situations. For the CBT group, however, participants considered the full range of their individual fears, and it is possible that this may have allowed a more creative, self-directed use of exposure (or lessening of avoidance) after the termination of active treatment.

Recognizing the methodological shortcomings of many studies in this area, Feske and Chambless, (1995) conducted a meta-analysis of data from fifteen studies of CBT and exposure treatments for social phobia. They concluded that "exposure with and without cognitive modification are equally effective in the treatment of social phobia" (Feske & Chambless, 1995, p. 712).

Three treatment studies for obsessive compulsive disorder (OCD) allow for a comparison of the effectiveness of behavioral and cognitive interventions. For these studies, the behavioral intervention in each case has been exposure and response prevention (ERP), which involves exposure to the obsessional thought and resisting the performance of the accompanying compulsive ritual. van Oppen et al. (1995) followed a treatment protocol that involved participants receiving either "pure" cognitive therapy (without behavioral experimentation) or ERP for six sessions, before behavioral experiments were added to the CT condition. Measures taken at the sixth session showed no significant differences between the two conditions. Likewise, both Emmelkamp and Beens, (1991) and de Haan et al. (1997) compared purely cognitive interventions with ERP for randomly assigned OCD sufferers. For both studies, there was no significant difference in outcome between the two conditions.

Studies that compare full CBT packages with ERP alone are also illuminating with regards to whether adding cognitive interventions improves the effectiveness of ERP. In the van Oppen et al. (1995) study described above, behavioral experiments involving exposure were added to the pure CT condition after 6 weeks of treatment, and this produced a slight superiority for the full CBT package as compared with the pure ERP condition at the termination of treatment. Vogel, Stiles, and Götestam (2004) compared ERP combined with relaxation training (as an attention placebo control) with ERP combined with CT. They found no significant difference in the treatment outcome for those who completed treatment, but they did find a significantly lower dropout rate for those receiving the ERP plus CT combination. In another study, McLean et al. (2001) compared CBT to ERP in the group treatment of OCD. They found that ERP produced significantly superior outcomes: for example, at 3 month follow-up, 13% of patients treated with CBT were recovered, compared with 45% of patients treated with ERP alone. More recently, the same research group has compared CBT to ERP delivered on an individual basis, finding no significant difference in outcome (Whittal, Thordarson, & McLean, 2005). Clark (2004) concludes "At this time there is no evidence that adding cognitive interventions to ERP […] is clinically more effective than ERP alone for a heterogeneous sample of patients with OCD." (p. 275).

2.3. Summary of component analysis studies

Generally, it can be noted that there is only a limited number of component analysis studies that seek to specify the active elements of CBT. Further, there is a virtual absence of multiple baseline studies comparing the effectiveness of behavioral and cognitive interventions. This is somewhat surprising given the volume of studies that exists on the effectiveness of CBT as a whole, the effort expended on specifying the particular cognitive distortions that may be implicated in particular disorders, and the emphasis that the cognitive model of psychopathology places on the role of cognition in mediating distress. From the component analysis studies that have been examined here, however, it is possible to conclude that, for a range of clinical problems, specifically cognitive interventions do not produce superior outcomes to the behavioral components of CBT. In studies examining depression, behavioral activation alone is equally as effective as behavioral activation combined with cognitive interventions. In studies examining the anxiety disorders, exposure-based interventions are of comparable efficacy to techniques that focus on examining thoughts.

What conclusions are possible in light of these empirical findings? First, it could be argued that there simply have not been a sufficient number of studies of sufficient quality undertaken to allow the true potency of cognitive interventions to be demonstrated. That, as more is learned regarding the specific cognitive distortions and cognitive processes underlying specific disorders, it will become increasingly possible to measure and target these cognitions, improving the overall efficacy of the therapy. However, the obvious counterargument is that until the added value of cognitive interventions is empirically demonstrated, it would be wise to maintain a scientific neutrality: "we have no evidence it works better," is preferable to "we know it works better, but haven't quite managed to show it yet." Second, it could be argued that the relentless equality of outcome between conditions in the above studies provides evidence that common, non-specific therapy factors underpin clinical improvement. Third, reiterating the conclusion drawn by Borkovec et al. (2002), it could be hypothesised that the different components of CBT examined in the above studies work on different systems. According to Lang's 'three systems' model, (Lang, 1985; 1988) there are physiological, behavioral, and cognitive aspects to psychological problems. Producing change in one system will induct change in the other two. For example, for depression, behavioral activation works directly on the behavioral system, but produces change in the cognitive and physiological systems; likewise, cognitive interventions produce change in the cognitive system, but also lead to corresponding changes in the behavioral and physiological systems. If this were the case, then it would not be surprising that component studies show equal outcomes for behavioral and cognitive interventions.

3. Behavioral experiments

Looking merely at component studies, it would be possible to conclude that we do not need to challenge thoughts in CBT: that explicitly cognitive interventions provide little or no added value to behavioral ones. However, it could be argued that cognitive interventions work best precisely when they are *combined* with behavioral aspects of the therapy. In other words, that the CBT whole is distinctly greater than the sum of its parts. Nowhere might this synergy better be illustrated than in the use of behavioral experiments.

Behavioral experiments (BEs) are planned, experiential activities designed to test the validity of beliefs. For Bennett-Levy et al. (2004) they are a form of collaborative empiricism aimed at achieving cognitive change: "BEs in cognitive therapy are primarily a means of checking the validity of thoughts, perceptions, and beliefs, and/or constructing new operating principles and beliefs." (p. 11). They contrast this cognitive change mechanism with behavior therapy's emphasis on exposure and habituation. Indeed, for Bennett-Levy et al., behavioral experiments confirm the cognitive primacy hypothesis.

Bennett-Levy (2003) compared the effectiveness of automatic thought records (ATRs) with BEs for three groups of student participants undertaking cognitive therapy training courses ($N = 27$), who practiced these techniques on themselves. Participants rated the effectiveness of each technique on a scale from 1 to 10, where 1 = no effect and 10 = very strong effect, for three outcomes: a) increased awareness of internal processes b) belief change, and c) behavior change. Bennett-Levy also gathered qualitative data on how participants experienced the techniques from written reflections, interviews and group reflection. It was found that there was no difference between ATRs and BEs in terms of increased awareness of internal processes. However, in terms of belief change and behavior change, BEs were rated as significantly more effective than ATRs. A grounded theory analysis of the qualitative data revealed that participants felt BEs involved greater emotion, anxiety and impact on "emotional belief." Bennett-Levy concludes that this is in line with Teasdale's (1997) Interacting Cognitive Subsystems (ICS) model. Namely, ATRs operate on the propositional

code, with its emphasis on declarative truth value; while BEs impact more on the holistic implicational code, with its emphasis on "heart level," emotional belief.

However, there are problems with Bennett-Levy (2003) study. Most notably, his participants were students studying cognitive therapy. They were a self-selecting sample with a presumed allegiance to cognitive therapy and knowledge of its theory and methods. Further, they were not a clinical sample. Extrapolating the findings to clinical groups is difficult, because such groups may struggle with a higher order of dysfunctional beliefs and patterns of avoidance.

Bennett-Levy aligns his explanation for the effectiveness of BEs with Teasdale's (1997) ICS model. However, Teasdale's views differ from those of Bennett-Levy et al. (2004) in terms of the role afforded to cognitive change:

"it may not be sufficient simply to gather data *about* experience and to evaluate beliefs against this evidence. Rather, it may be necessary to arrange for actual experiences in which new or modified models are created" (Teasdale, 1997, p. 150; emphasis in original).

For Teasdale, the value of BEs is that they represent "enactive procedures" that activate different schematic models. They change the patient's "actual way of *being*" (p. 150) rather than providing evidence which, rationally considered, leads to belief change.

It remains a moot point whether behavioral experiments need to challenge thoughts in order to be effective. It is equally plausible that cognitive change follows behavioral change, rather than driving it. Studies testing these alternative hypotheses could potentially compare the effectiveness of full BEs (with their emphasis on identifying and challenging beliefs), with targeted behavioral exposure. In the absence of such research, it is open to question whether behavioral experiments gain anything from prioritising cognitive change.

4. The rapid early response debate

As noted in the introduction, one of the "empirical anomalies" identified by Hayes (2004) as casting doubt on the need for cognitive interventions in CBT is that "Clinical improvement in CBT often occurs before the presumptively key features have been adequately implemented" (Hayes 2004, p. 4). This phenomenon has been termed "rapid early response".

Ilardi and Craighead (1994) reviewed data from eight studies of the efficacy of CBT for depression. They found that in 7 out of the 8 studies, between 60% and 70% of the total improvement patients experienced across the course of therapy happened within the first 4 weeks. They hypothesized that initial improvement was unlikely to be explained by cognitive modification techniques, and concluded instead that non-specific factors mediated the majority of the improvement seen in CBT.

However, Tang and DeRubeis (1999) re-examined the data reviewed by Ilardi and Craighead. They found that in 7 out of the 8 studies, the therapy actually comprised two sessions per week within the initial 4-week period. Therefore, patients were receiving a dose of eight sessions of therapy within that time — an amount easily sufficient to allow the introduction of cognitive modification techniques and see resulting improvements. In addition, Tang and DeRubeis separated out the data for those who responded well to treatment and those who did not. This was done for two of the original eight studies, where full data were available. Responders were defined as those whose BDI score at the completion of treatment was less than 10; non-responders were those with a BDI of 10 or more. Separating the data in this way demonstrated that, within the first 4 weeks of treatment, in one study responders experienced 56% of their total improvement after 53% of their therapy sessions, and in the other study 43% of their total improvement after 42% of their therapy sessions. On the other hand, non-responders experienced almost 100% of their total improvement in the first 4 weeks. Thus, those for whom the therapy worked well experienced a steady improvement across the course of treatment, while those for whom it did not work so well experienced initial gains which then levelled off.

Ilardi and Craighead (1999) replied to the criticisms of Tang and DeRubeis by noting that in their original study, both responders and non-responders still showed a rapid early response to treatment. Further, they maintain that even though there were two treatment sessions per week, 4 weeks of treatment still constitutes too short a time frame for patients to absorb and respond to cognitive modification techniques. Wilson (1999) asserts that early rapid response may be a general phenomenon, applying to conditions other than depression. For example, Wilson et al. (1999) found that by session five of treatment, people with bulimia nervosa showed a 76% improvement in their frequency of binge eating.

On balance, evidence for the early rapid response phenomenon is not compelling. Tang and DeRubeis note that eight sessions is sufficient to allow for cognitive modification techniques to be introduced. Indeed, Fennell and Teasdale (1987) state that introducing patients to the cognitive rationale and providing initial homework tasks may be sufficient to produce substantial improvement in some cases. When the number of sessions is accounted for instead of the number of weeks, then the early response to therapy is not greatly disproportionate to the dose of sessions received. Of course, the lack of evidence for an early rapid response in cognitive therapy is not in itself evidence *for* the effectiveness of cognitive procedures.

5. Cognitive mediation

The final "empirical anomaly" in the CBT research literature alluded to by Hayes (2004) is that "Changes in cognitive mediators often fail to explain the impact of CBT" (p. 4). Indeed, Ilardi and Craighead (1999) note that "the status of targeted cognitive modification as the *sine qua non* of patient improvement in CBT remains in doubt" (p. 298). Here, it is necessary to return to the distinction between cognitive intervention as *procedure* in the practise of CBT, and cognitive change as the *mechanism* underpinning symptom improvement in the CBT model of psychopathology. According to the CBT model, cognitive interventions work through changing underlying cognitive structures. However, unless their effects were demonstrated to be mediated by changes in such underlying structures, there would remain the possibility that they work through other means. Likewise, impact of behavioral interventions, although not directly targeting dysfunctional beliefs, could theoretically work by altering cognitions. Hence, status of cognition as a mediating mechanism is a matter for separate empirical enquiry from the effectiveness of the CBT interventions aimed at changing these cognitions.

Comparison studies show that cognitive change can be a product of other therapeutic treatments, apart from CBT. Not only can cognitive change occur as a result of other treatments, but also the degree of change can be equal to that produced by CBT. For example, McManus, Clark, and Hackmann (2000) studied outcomes for 23 participants with social phobia. They received either cognitive therapy or pharmacotherapy with instructions for self-administered exposure. Belief change for the key cognitions thought to underlie social phobia was measured by the Social Probability and Cost Questionnaire (SPCQ; modified from Foa, Franklin, Perry, & Herbert, 1996). It was found that by the termination of treatment, there were equally significant reductions in participants' estimations of the probability and cost of negative social events in both treatment conditions. Likewise, Simons, Garfield and Murphy (1984) studied both symptom and cognitive change for 28 depressed outpatients who received either cognitive therapy or pharmacotherapy. Cognitive measures included the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1979) and the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980). Both CT and pharmacotherapy produced similar levels of change on the cognitive as well as the symptom measures. As a result, Simons et al. concluded that cognitive change was a *part* of the improvement seen in treatment, rather than the primary *cause* of improvement. These findings are reinforced by a meta-analysis conducted by Oei and Free (1995) that concluded that cognitive change is no greater as a result of CBT than as a result of drug treatment or other therapies.

Hollon and DeRubeis (2004) acknowledge these findings. However, they note that "the relevant question is not whether change in cognition is specific to cognitive therapy, but whether the pattern of change over time is consistent with causal agency." (p. 56). They cite a study by DeRubeis et al. (1990) that tracked symptomatic and cognitive changes during the treatment of depression with either cognitive therapy or pharmacotherapy. In this study, changes in cognition for those treated with CT predicted changes in symptoms, while this was not the case for those who received pharmacotherapy. Hollon and DeRubeis (2004) state that this finding supports the conclusion that cognitive change was the *mechanism* of change in the CT condition, but the *consequence* of change for those receiving pharmacotherapy. Tang and DeRubeis (1999) have also examined the phenomenon of the "sudden gain" that some patients make during cognitive therapy for depression. By examining individual data for three patients across two studies, they found that sudden decreases in depressive symptoms (as measured by the BDI) were often preceded in the previous therapy session by patients verbalizing a change in at least one important belief. Reviewing this evidence as a whole, Hollon and DeRubeis (2004) conclude that "there are indications that theoretically specific ingredients drive the change in depression by virtue of inducing change in existing beliefs and information-processing strategies" (p. 57).

However, is this conclusion warranted? In the original DeRubeis et al. (1990) study, four measures of cognition were used: the Automatic Thoughts Questionnaire (ATQ), the Hopelessness Scale (HS; Beck, Weissman, Lester & Trexler, 1974), the Dysfunctional Attitudes Scale (DAS) and the Attributional Style Questionnaire (ASQ; Seligman, Abramson,

Semmel, & von Baeyer, 1979). DeRubeis et al. compared participants who completed a 12-week course of CT (including data from participants who received CT *plus* imipramine) (*n* = 32) with participants who had received 12 weeks of pharmacotherapy alone (*n* = 32). Measures of cognition and depression were taken pretreatment, after 6 weeks (midtreatment) and at 12 weeks (post-treatment). Both groups showed significant and comparable improvements in depression at the termination of treatment. DeRubeis et al. also found that for both the CT and pharmacotherapy groups, there was significant improvement on all four cognitive measures between pretreatment and midtreatment. Further, there were no significant differences between the groups in the degree of improvement on any of the cognitive measures. Therefore, pharmacotherapy appears as effective as cognitive therapy in producing cognitive change. The key finding of DeRubeis et al., however, is that for participants receiving CT, greater midtreatment changes on the ASQ and DAS were predictive of lower posttreatment depression scores. From this, they conclude that cognitive change is instrumental to the amelioration of depression for those receiving CT, while being epiphenomenal to the improvement of those receiving drug treatment alone.

This begs the question as to why equally significant changes in cognitive content for the pharmacotherapy group failed to predict how well participants fared. Regardless of the treatment modality, the cognitive model would hold that "Alterations in the content of the person's underlying cognitive structures affect his or her affective state and behavioral pattern" (Beck et al., 1979, p. 8). DeRubeis et al. are aware of this anomaly. They hypothesise that measured cognitive changes in the CT group are indicative of changes at a deeper level: namely, the use of active cognitive and behavioral problem solving strategies. However, if such strategies are the true "theoretically specific ingredients" driving change in CT for depression, then they are neither specified nor measured in the study. Indeed, DeRubeis et al. state that "the hypothesis we offer is speculative and not, in its specific form, supported by the present findings" (p. 867).

Jacobson et al. (1996), as part of their component study of behavioral and cognitive interventions for depression, measured changes in putative cognitive mediators using the Automatic Thoughts Questionnaire (ATQ), and the Expanded Attributional Style Questionnaire (EASQ; Peterson & Villanova, 1988). They attempted to establish if there was a correlation between early cognitive change on these measures and late change in depression symptom scores. In contrast to the findings of DeRubeis et al., they did not find a causal, temporal link between changes in cognitive mediators and later changes in symptom scores for the AT (automatic thoughts) or CT (cognitive therapy) treatment conditions. However, they did find that change on two subscales of the EASQ (stable and global attributional style) correlated with later improvements in depression for patients treated in the BA (behavioral activation) condition. The study of Jacobson et al. (1996), using similar measures and temporal analyzes to the study of DeRubeis et al., failed to demonstrate evidence for cognitive mediation.

Burns and Spangler (2001) also examined the causal relationship between cognitive change and symptom change in depression and anxiety using structural equation modelling. Dysfunctional attitudes for 521 outpatients with depression and anxiety were measured using the Burns Perfectionism Scale (PS; Burns, 1980) and the Burns Interpersonal Attitude Scale (BIAS; Persons, Burns, Perloff, & Miranda, 1993). They found that although dysfunctional attitudes were correlated with changes in depression and anxiety, there was no causal effect linking belief change with symptom change.

Finally, Hollon and DeRubeis's assertion that cognitive change is the cause of symptom change in cognitive therapy but the consequence of symptom change in pharmacotherapy is not borne out by the temporal relationship between data from the Simons et al. (1984) study. Data illustrating this point is given in Table 2.

Table 2
Changes in cognitive variables and BDI score (after (Simons et al., 1984)

Measure	Condition (*N*=28)	Pretreatment	Week 4	Week 8	Termination
BDI}	CT	29.8	17.4	13.5	9.3
	Pharmacotherapy	28.6	16.7	10.3	8.6
ATQ}	CT	111	84	71	62
	Pharmacotherapy	105	81	61	62
DAS}	CT	160	146	124	111
	Pharmacotherapy	157	139	128	128

CT = cognitive therapy; BDI = Beck Depression Inventory; ATQ = Automatic Thoughts Questionnaire; DAS = Dysfunctional Attitudes Scale.

It can be seen from Table 2 that there is a similar gradient of change in cognitive variables and symptoms of depression for both the cognitive therapy and pharmacotherapy conditions. Indeed, if anything, changes on cognitive measures occur earlier in the drug treatment condition relative to changes in BDI scores. The opposite temporal pattern might be expected if cognitive change were driving symptom change in CT, but a consequence of symptom change in pharmacotherapy.

Teasdale et al. (2001) report findings from the Cambridge and Newcastle trial of cognitive therapy for residual depression. Here, 158 patients were randomized to receive antidepressant medication and clinical management either alone or together with 18 sessions of cognitive therapy. A battery of cognitive measures was administered in order to see if cognitive change mediated relapse rates. It was found that an *extreme responding style* on these questionnaires predicted early relapse, rather than the specific content of cognitions. Teasdale et al. take this to be indicative of a "black and white" thinking style that both mediates relapse and responds differentially to treatment with CT. However, the clarity of these findings is somewhat compromised by the fact that a non-depressed control group produced *more* extreme positive responses on the same questionnaires.

In summary, the evidence that cognitive variables mediate therapeutic change in CBT is somewhat limited. The key findings by DeRubeis et al. (1990) have not been replicated. Although Tang and DeRubeis (1999) present data on "sudden gains" in therapy following belief change, this was only for three patients. The finding of Teasdale et al. (2001) of a mediating cognitive style (extreme responding) does not fully differentiate recovered depressives from non-clinical controls. Meanwhile, a variety of studies has shown that cognitive change is an outcome of other treatments, to the same extent as in CBT. Jacobson et al. (1996) suggest that difficulty identifying cognitive mediators of therapeutic change may reflect the poor quality of measures of beliefs and attitudes. Nevertheless, an important element of the rationale for cognitive interventions–that changes in cognition mediate therapeutic change in CBT–currently lacks empirical support.

6. Conclusion

This paper has attempted to answer an important question at the heart of CBT theory and practice: do we need to challenge thoughts in CBT? In essence, this question concerns the logical, rationalist methods used to challenge dysfunctional thoughts and beliefs. Nor is the question an arbitrary one. Proponents of a new generation of behavioral therapies propose that the rational challenging of thoughts is superfluous (Hayes, Follette, & Linehan 2004). Likewise, those who claim that human cognition comprises multiple processes, stores and codes seek new mechanisms by which to achieve change at these various levels (Segal, Williams, & Teasdale, 2002). These theoretical developments and others like them have led to an increased emphasis on behavioral change, constructivism and attentional control — the so-called "Third Wave" of CBT. At the same time, they have led to a decreased emphasis on the rational challenge of the content of thoughts.

This paper has attempted an empirical rather than a theoretical examination of the status of cognitive interventions in CBT. A comprehensive review of component research into the active elements of CBT was undertaken. This examined studies both of depression and the anxiety disorders. The review showed that, almost without exception, component studies found no difference in effectiveness between the cognitive and behavioral elements of CBT. Nor did cognitive interventions provide "added value" to behavioral interventions. Taken together, these studies provide a substantial body of research showing that cognitive interventions are not a necessary component of the therapy. While it could be the case that non-specific therapy factors or blunt outcome measures explain these findings, a further possibility is that interventions are effective when they help people to switch "modes"(Beck, 1996) or "schematic models" (Teasdale, 1997). More precisely, psychological states comprise interacting cognitive, affective, behavioral and physiological elements. Any treatment which effectively targets one of these systems may lead to a change in all of them (Borkovec et al., 2002).

It is possible that component studies are flawed because in seeking to dismantle the separate parts of CBT, they neutralise what makes it effective: the *interaction* of cognitive and behavioral techniques. Behavioral experiments were considered from this perspective. While Bennett-Levy (2003) compared the effectiveness of behavioral experiments to that of purely cognitive interventions, the key comparison will come when their efficacy is measured against purely behavioral interventions. This will elucidate whether their emphasis on cognitive hypothesis testing is justified, and in turn whether there is an interaction effect between the behavioral and cognitive elements.

Another doubt cast upon the validity of cognitive interventions in CBT is the finding that the therapy produces a rapid early improvement in symptoms; one which putatively precedes the application of cognitive techniques. The research on this issue was reviewed and it was found that evidence for such an effect was lacking.

The issue of cognitive mediation was then examined. If, as Clark (1995) asserts, CBT works by modifying dysfunctional cognitions and realigning thinking with reality, then it should be possible to show cognitive change mediating symptomatic improvement over the course of therapy. Evidence pertaining to this hypothesis was reviewed. It was shown that there is currently little empirical support for cognitive mediation.

Taken together, these findings reveal a worrying lack of empirical support for some of the fundamental tenets of CBT. There is a paucity of evidence that cognitive interventions forming the core procedural aspects of CBT are differentially effective in reducing distress. Further, there is a lack of evidence that their effectiveness, such as it is, is mediated cognitively. Given this state of affairs, it is somewhat surprising that there is not more research on these issues. Suggestions for further research might include the following:

a) Studies comparing cognitive measures and symptom measures throughout the course of therapy would allow a more complete examination of temporal changes in beliefs and cognitive mediation. Development of new and better measures of the beliefs thought to underlie particular disorders would enhance this research.
b) Multiple baseline studies of the cognitive and behavioral elements of CBT.
c) Further component studies comparing CBT with its behavioral and cognitive elements in order to test specific hypotheses (for example, *Do behavioral experiments work by testing beliefs, or through exposure?*).

CBT has always stressed its status as an empirically grounded therapy. A multitude of studies has shown it to be an effective treatment for a range of psychological disorders. Nothing should detract from this achievement. However, it is important for the development of CBT that it keeps sight of its empirical roots. Anomalous findings and gaps in knowledge represent an opportunity to clarify mechanisms of change and build better, more effective therapeutic interventions. This paper is an invitation to renewed curiosity in line with these principles.

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