Cognitive-Behavioral Therapy: Nature and Relation to Non-Cognitive Behavioral Therapy

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Since the introduction of Beck’s cognitive theory of emotional disorders, and their treatment with psychotherapy, cognitive-behavioral approaches have become the most extensively researched psychological treatment for a wide variety of disorders. Despite this, the relative contribution of cognitive to behavioral approaches to treatment are poorly understood and the mechanistic role of cognitive change in therapy is widely debated. We critically review this literature, focusing on the mechanistic role of cognitive change across cognitive and behavioral therapies for depressive and anxiety disorders.

Keywords: cognitive-behavioral therapy; cognitive theory; psychotherapy processes; depression; anxiety

The origin of cognitive-behavioral therapies (CBTs) as a family of interventions can be traced to the advent of behavioral treatments for psychopathology in the 1950s and, later, the so-called “cognitive revolution” of the 1950–1960s (Dobson, 2009). Consequently, CBTs blend techniques that are emphasized in behavioral therapies (BTs) and cognitive therapies (CTs). However, there remains skepticism regarding the relative contributions of CT strategies to BT strategies in promoting symptom change within the CBTs (Longmore & Worrell, 2007). Additionally, critics have asserted that changes in thinking are not mechanisms of change in CBTs (e.g., Kazdin, 2007), calling into question whether there is any kind of contribution of the “cognitive” in cognitive-behavioral therapy.

Despite debate regarding their active treatment components as well as working mechanisms, CBTs continue to be the most widely studied forms of therapy (Hofmann, Asmundson, & Beck, 2013). A uniquely appealing aspect of CBTs is that their theories of therapeutic change comport well with most modern conceptualizations of psychopathology. In this review, we attempt to reconcile skepticism regarding the relative contribution of CT strategies to BT, as well as the mechanisms that account for their efficacy. First, we provide a very brief historical overview of the origins of CBT and discuss the support for the cognitive vulnerability models to depression and anxiety disorders. We discuss methodological challenges in psychotherapy research that have impeded a more thorough understanding of the relative contributions of cognitive to behavioral techniques. We then focus most of our discussion on research on the cognitive mechanisms of change in CT, BT, and CBTs for depression and anxiety disorders.

We use the terms cognitive therapy (CT) and cognitive techniques to refer to behaviors therapists engage in that are targeted towards changing the content or process of thoughts, inferences, interpretations, cognitive biases, and cognitive schemas.¹

¹The terms “cognitive therapy” (CT) and “cognitive-behavioral therapy” (CBT) are often used interchangeably. We believe this is somewhat unfortunate in that it might be informative to reserve the term CT to a set of interventions within the broader family of CBTs that are more “purely” cognitive in nature. However, throughout the article, when we refer to findings in studies of CT or CBT, we are adhering to the label the study authors use. Additionally, we use CBTs, in plural, to refer to the family of cognitive-behavioral therapies.
These interventions can include Socratic questioning, examining the evidence for and against beliefs, cognitive restructuring, and adopting alternative core beliefs. We use the terms behavior therapy (BT) and behavioral techniques to refer to behaviors therapists engage in that are targeted towards a change in observable behavior, including in vivo exposure, imaginal exposure, and activity scheduling. We use cognitive-behavioral therapies in the plural (CBTs) to refer to the family of interventions to which CT and BT belong, and in the singular, CBT, to refer to a treatment package that combines cognitive and behavioral techniques. By cognitive change, we refer to changes in the content of thoughts, inferences, interpretations, and cognitive biases. By behavioral change, we refer to changes in behavior, such as increasing the frequency of selected behaviors (e.g., approaching feared stimuli, engaging with pleasurable activities) or decreasing the frequency of other behaviors (e.g., safety behaviors). We include in our paper a discussion of issues related to the conceptualization and measurement of cognitive vs. behavioral interventions as well as cognitive vs. behavioral mechanisms of change and conclude with a summary and with recommendations for future research.

Cognitive Therapy: Nature and Relation to Behavioral Therapy

Behavioral therapies emerged in the 1950s–1960s (O’Donohue & Noll, 1995). The behavioral models emphasized maladaptive learning and self-sustaining behaviors as key to the maintenance of psychopathology. This made behavioral change the obvious target of treatment, an approach that was in stark contrast to the previously dominant psychoanalytic models. Under psychoanalysis, pathological behavior was seen to reflect dysfunction in underlying psychic structures. Behavioral change was thus seen as surface-level “symptom reduction” that did not address underlying problems. BTs proved very effective, particularly in the treatment of phobias and more circumscribed states of anxiety. Principles of associative learning were used to account for the efficacy of these interventions. To the behaviorists, learning had a specific meaning: an overt change in behavior (e.g., approaching a previously avoided stimulus) in the absence of symptoms (e.g., without displaying the fear reaction). This definition avoided “mentalistic” terms. Although early behavioral models featured theoretical accounts focused on associative learning, nonassociative learning, including habituation, was also seen as important. Newer behavioral models also focus on inhibitory learning (Craske et al., 2008).

CT emerged in the context of the so-called cognitive revolution (Beck, 1991; O’Donohue, Ferguson, & Naugle, 2003) from the writings of Ellis (1962), who described a form of therapy known as rational-emotive therapy, and Beck (1963). The cognitive models of Ellis and Beck focused on inferential errors leading to maladaptive views of one’s self, world, and the future. According to Beck, cognitive biases and maladaptive cognitive content are the product of the activation of cognitive schemas that typically develop early in life. Unlike BTs, which were initially successful in specific phobias and circumscribed anxieties, CTs were focused on depressotypic presentations and more generalized anxiety. Early in his writing, Beck recognized that his cognitive theory of psychopathology, which gave a central role to cognition in the etiology of disorder, contrasted with behavioral theories of psychopathology. In his highly cited article, “Cognitive Therapy: Nature and Relation to Behavioral Therapy,” Beck (1970) described important differences between the theories that underlie BT and CT while recognizing areas of overlap in the performance of the therapies. Similarities include that both therapies deal with issues in the present, are symptom-focused, and require active therapist contribution.

Beck (1970) recognized differences between behavioral and cognitive approaches. He applied the principles of his then nascent cognitive theory to account for the mechanisms of action of systematic desensitization, a BT. He concluded that the cognitive model “provides a greater range of concepts for explaining psychopathology as well as the mode of action of therapy.” That is, Beck made a distinction between the nature of the therapeutic interventions (i.e., cognitive vs. behavioral) and their working mechanisms in providing a cognitive account of the effects of a behavioral intervention. Beck’s paper would become one of the early reflections on the relative contributions of cognitive to behavioral strategies and the relevant mechanisms of change. Although Beck has provided two updates to his cognitive model (Beck, 1996; Beck & Haigh, 2014), its basic tenets remain largely intact: that the distinction between different forms of psychopathology can be traced to differences in the locus of the cognitive pathology and that cognitive change, regardless of how this change is achieved, is integral to symptom change.

Cognitive Vulnerability to Depression and Anxiety

Basic research supports the notion that cognitive vulnerabilities confer risk to the onset and maintenance of psychopathology (see Mathews & MacLeod, 2005). Attentional biases to threatening
stimuli, along with overestimation of threat, have been implicated in the etiology of anxiety disorders (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van Ijzendoorn, 2007). Biases associated with depression include difficulties disengaging from negative material, sustained or symmetrical attention to negative, relative to positive, stimuli (Kircanski & Gotlib, 2015), negative biases in the appraisal of life events (Mehu & Scherer, 2015), symmetric memory for negative vs. neutral or positive information (Kircanski & Gotlib, 2015), and negative schemas about the self that foster maladaptive and negative thinking (Beck & Haigh, 2014).

Overall, existing research is supportive of cognitive vulnerability models of affective disorders. For example, cross-cultural research consistently suggests that, on average, healthy individuals have a bias towards optimistic thinking that is not found in individuals who are depressed and who, instead, have a bias towards more negative thinking (Mezulis, Abramson, Hyde, & Hankin, 2004). Similarly, in a meta-analytic review of 172 studies examining biases towards threatening stimuli, Bar-Haim et al. (2007) found that anxious participants are biased to attend to threatening stimuli, relative to nonanxious participants (d = 0.45). The causal role of these cognitive vulnerabilities, particularly in depression, has been questioned partly because most of the early research on this matter was correlational in nature (see Ingram et al., 2006). Findings from prospective studies, however, also support cognitive models. For example, daily fluctuations in negative automatic thoughts have been found to predict subsequent negative mood, even controlling for prior levels of automatic thoughts (Wenze, Gunthert, & Forand, 2007; Wenze et al., 2010). Negative dysfunctional attitudes also predict depressed mood following a stressor (Hankin, Abramson, Miller, & Haefel, 2004). In one study, participants who were classified as being at high cognitive risk were almost 7 times more likely to report a major depressive episode at 2.5 years follow-up, relative to those at low risk (Alloy et al., 2006).

Although prospective studies provide a stronger level of evidence for causality than correlational studies, findings from these studies are still subject to third variable confounds, making experimental designs preferable. Relatively few experiments manipulating cognitions and assessing the effects of the manipulation on mood have been conducted. The results of these experiments, however, are consistent with models of cognitive vulnerability (see Mathews & MacLeod, 2005). For example, in a series of experiments, Mathews and Mackintosh (2000) reported that inducing bias in the interpretation of ambiguous information as threatening leads to increases in state anxiety. In another study, MacLeod et al. (2002) manipulated attention to emotionally negative information. After a stressor task, participants who had their attention manipulated towards negatively valenced stimuli showed greater anxiety and depression than participants in the control group.

If cognitive biases increase the risk for depression and anxiety states, it follows that strategies that address these biases should result in a reduction of risk. This hypothesis has support in basic research on emotion regulation. Webb, Miles, and Sheeran (2012) conducted a meta-analysis of 306 comparisons of emotion regulation strategies. Strategies that focused on cognitive change were estimated to be the most consistently effective ways of regulating emotions (d = 0.36). Strategies aimed at helping individuals adopt more rational perspectives, as is encouraged in CT, were associated with the largest effect (d = 0.45). Providing even more support for cognitive theories, studies that examine the biological vulnerabilities to negative emotional states suggest that, at the phenomenological level, biological vulnerabilities render individuals more likely to experience negative emotional states by interfering with their abilities to engage in cognitive reappraisal strategies (Firk, Siep, & Markus, 2013; Lemogne et al., 2011).

More research is needed that characterizes more precisely the nature of the cognitive biases implicated in depression and anxiety, especially research that is experimental. The relationship between affective disorders and cognition is bidirectional, which must also be accounted for in theories of psychopathology. However, given the amount of evidence and the dearth of competing explanations, it can be safely asserted that the cognitive model is a valid characterization of the etiology of affective disorders. Thus, one would expect considerable support for the hypothesis that change in cognition mediates symptom change in the context of psychotherapy. Instead, the literature contains questions about whether “we need to challenge thoughts in cognitive behavior therapy?” (Longmore & Worrell, 2007) and assertions such as “whatever may be the basis of changes with CT, it does not seem to be the cognitions as originally proposed” (Kazdin, 2007). Why is this so?

It’s Complicated

Previously, we (Lorenzo-Luaces, German, & DeRubeis, 2015) have argued that disagreement among commentators (e.g., Kazdin, 2007; Longmore & Worrell, 2007) regarding the role of cognitive
change in promoting symptom change in psychotherapy for depression emerges from different assumptions regarding the inferences that can be drawn from treatment studies. Below we review some of these issues in psychotherapies for depression and anxiety.

**EXPERIMENTAL DESIGNS: ADDITIVE AND DISMANTLING STUDIES**

When two treatment packages are very different (e.g., psychoanalysis vs. exposure and response-prevention), it is easy and perhaps even appropriate to interpret findings from studies comparing treatment packages to reflect the relative efficacy of specific therapy procedures (e.g., analyzing transference vs. engaging in exposure). However, when assessing treatments, such as CBT or eye-movement desensitization and reprocessing (EMDR), which combine multiple active and overlapping elements, in this case exposure and cognitive restructuring (Tolin, 2014), it becomes more difficult to extrapolate conclusions about therapy procedures from outcome data.

In lieu of tightly controlled basic research, such as the research on emotion regulation strategies (Webb et al., 2012), researchers have used component studies as a way of addressing questions about the differential utility of treatment elements. These component studies are often referred to as if they represent a single class of study design, but there are at least two different types of study designs, additive and dismantling designs, that fall under this rubric. As described by Bell, Marcus, and Goodlad (2013), they address different kinds of questions. In additive component studies, in one condition a component is added to and compared with an already-existing, simpler treatment. Butler, Cullington, Munby, Amies, and Gelder (1984) provide an early example of such a study. They examined the value of adding anxiety management to exposure for social anxiety by comparing the combined treatment to exposure only as well as to exposure plus a nonspecific filler. Their findings suggested that adding anxiety management to exposure improved treatment outcomes. In dismantling designs, at least one component of a multicomponent treatment package is removed from the treatment and compared to the full treatment package or to the other components. For example, Foa, Steketee, Grayson, Turner, and Latimer (1984) dismantled exposure and response-prevention (ERP) and compared its effects with the effects of exposure only and response prevention only. Their findings suggested that ERP was superior to either of its single components and that, for contamination fears, exposure alone may be more effective than response-prevention alone. Bell et al. (2013) conducted a meta-analytic review of components studies and concluded that it is uncommon, in studies that have used dismantling designs, for one component of a treatment to outperform another. However, in studies that have used additive designs, adding one treatment component to another enhances positive therapeutic outcomes, particularly in the longer term.

A meta-analysis by Adams et al. (2015) addressed the contribution of CT to BT and did not find an added benefit of CT to BT and CBT packages. However, this meta-analysis did not differentiate between additive and dismantling designs. Although, on the face of it the component studies in the meta-analysis by Adams et al. seem like they provide very conclusive answers about the superiority or equipotency of CT and BT, component studies, as they have been conducted and interpreted, have been extremely problematic. Summarily reviewing some of the limitations of component studies, Bell et al. (2013) stated:

... Null results [in component studies] do not directly address the issue of specific versus common factors because there is no group that received only common treatment components. [...] Component designs may also underestimate the contributions of the component. Rehm (2009) suggested that because much improvement typically occurs in the early stages of therapy, whichever component is presented first will appear to be the most effective. Thus, the dismantled component (which is never introduced) is likely to appear unnecessary. Component studies are also likely to be statistically underpowered (Kazdin & Whitley, 2003) to detect the relatively small effect sizes that are likely to occur with these types of designs. [...] A two-group component study with a presumed effect size of .24 (half the treatment vs. placebo effect size) would require over 250 patients in each condition to have a power of .80. Even Kazdin and Whitley’s (2003) higher estimate of an effect size of .45 for additive design studies would require 78 patients in each condition. In contrast, the average sample size for the studies included in the present meta-analysis was 23 participants in each condition, which would require a large effect size of .84 to have a power of .80.

The component methodology evidenced a surge in popularity following a landmark study by Jacobson et al. (1996). Jacobson et al. conjectured that the full CBT for depression package could be divided into three components: (1) behavioral activation (BA); (2) challenging automatic thoughts (ATs); and (3) modifying core beliefs (CBs). To compare the relative efficacy of these procedures, and perceiving limitations in prior work suggesting that CT for depression was superior to BT (Shaw 1977), Jacobson et al. randomized participants to three conditions lasting a maximum of 20 sessions: (1) 100% BA; (2) a condition that could use all the elements of BA and could include AT work; and (3) a condition that could use all of the elements of
BA, work on ATs, with a required minimum of 8 sessions devoted to CB work. In this study, across various metrics, no statistically significant between-condition differences in outcome were reported. These findings have generally been misinterpreted as indicating that BA is the active component in CBT for depression and that the interventions provided in the cognitive components were inert, thus presenting a major challenge to Beck’s cognitive theory (Longmore & Worrell, 2007).

Data from assessments of therapists’ adherence speak to the construct validity of the experiment by Jacobson et al. (1996). Although, in terms of the absolute frequency with which techniques were conducted, behavioral work was nearly as frequent in the CB condition as they were in BA, the relative frequency of BA procedures was greater in BA than in AT and CB. In the follow-up analysis of the trial, Jacobson and colleagues went on further to note:

... by definition, participants in the BA condition received more BA than did those in the other treatment conditions. Although one might be tempted to infer from this study that cognitive interventions are nonessential, our study does not directly address the validity of such an interpretation. All we can conclude is that adding cognitive interventions to BA is no more effective than using that time to add more BA. (Gortner, Gollan, Dobson, & Jacobson, 1998, p. 381, emphasis added)

In other words, if the findings from Jacobson et al. (1996) are taken to mean that CT procedures are inert, a corollary emerges that was not tested in their design: that a BA condition that allowed only 6 or 7 sessions (one-third of the 20 sessions) should yield outcomes equivalent to a 20-session course of BA. Thus, per Gortner et al. (1998), the only hypotheses regarding behavioral treatment of depression that could have been tested with the study design were that BA is: (a) superior to, or (b) not inferior to, a cognitive-behavioral treatment. As there were no significant differences between the treatments, the most that can be said is that the BA condition, in the context of a study with low power, was not shown to be inferior to treatments that divided time between cognitive components and behavioral ones.

Very few dismantling studies have directly compared “pure” cognitive and behavioral interventions. The handful of studies that have compared purely behavioral (e.g., activity scheduling) to purely cognitive (e.g., cognitive restructuring) treatments for depression have tended to find little if any difference in the acute effects of cognitive versus behavioral treatments (Mazzucchelli, Kane, & Rees, 2009). In one study comparing BA to CT (Dimidjian et al., 2006), although there were no statistically significant differences between the two treatments, BA was more effective than CT or medications for individuals who were severely depressed. In the BA condition, 76% of more severely depressed participants met criteria for response or remission, compared to 48% of patients in CT and 49% in antidepressant medications. Among the less severely depressed patients, response rates on the BDI were 56% in CT, 60% in BA, and 40% in ADM. Coffman et al. (2007) identified patients from the Dimidjian et al. trial who exhibited a pattern of “extreme non-response” (ENR) in CT (approximately one-fourth of those assigned to CT) and noted that none of the patients assigned to BA evidenced an ENR. At baseline, the CT ENR patients were more severely depressed, evidenced more functional impairment, and reported more problems with their primary support group. They interpreted these findings to suggest that, relative to CT and medications, BA may be particularly effective for patients with severe depression that is accompanied by interpersonal dysfunction. However, it should be noted that the advantage of BA over CT dissipated entirely across the trial’s 2-year follow-up (Dobson et al., 2008). Moreover, to our knowledge, the findings of Dimidjian et al. (2006) have not been replicated. An attempt to replicate Coffman et al.’s ENR findings in a separate sample of depressed patients (Koenig, Jarrett, Gallop, Barrett, & Thase, 2014) treated with CT found a low (6%) rate of nonresponse, and severity, functional impairment, and interpersonal problems were not good predictors of nonresponse. Finally, Webb et al. (2013) found that the therapists in the Dimidjian et al. (2006) trial implemented CT with a relatively more behavioral than cognitive focus, compared to therapists from other CT trials.

The effect of cognitive change strategies in Beck’s C(B)T for depression has also been questioned, on the basis that much of symptom change occurs early in treatment (Ilardi & Craighead, 1994). However, it has been shown in several studies that CBT therapists use cognitive change techniques as early as session one (Braun, Strunk, Sasso, & Cooper, 2015; Conklin & Strunk, 2015; Strunk, Brotman, & DeRubeis, 2010). In fact, at least one therapy manual (i.e., Muñoz & Miranda, 1986) addresses cognitive change exclusively for several sessions before addressing behavior.

In anxiety disorders, among the studies included in the meta-analyses of Adams et al. (2015) and Bell et al. (2013), only eight studies (Barlow, Rapee, Brown, 1992; Borkovec, Newman, Pincus, & Lytle, 2002; Emmelkamp & Beens, 1991; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Mattick, Peters, & Clarke, 1989, Szymanski, & O’Donohue, 1995; White, Keenan, & Brooks, 1992; Williams,
Falbo, 1996) compared a purely cognitive treatment condition to a purely behavioral condition. Among these eight studies, we conducted a random effects meta-analysis (settings as per IntHout, Ioannidis, & Borm, 2014). There was no statistically significant difference in end-state primary outcomes between “pure” CT and “pure” BT (higher values indicate superiority for CT; \( g = 0.010 \) [95% CI: -0.203 to 0.222], \( SE = 0.090, t = 0.106, p = 0.919 \); see Fig. 1). There was minimal heterogeneity between effect sizes included in the meta-analysis (Cochrane’s \( Q = 3.371, df = 7, p = 0.849 \); 14.90% heterogeneity), consistent with an account that trial-level findings were generally equivocal, with few meaningful between-trial differences in effects. The results of these studies suggest that CTs can be as effective as BTs in the treatment of anxiety disorders. Although exposure-based treatments are considered the mainstay of CBTs for anxiety, other meta-analytic reviews also suggest that ERP, CBT, and CT are about equally effective across a range of anxiety disorders (Norton & Price, 2007; Ougrin, 2011). This led Arch and Craske (2008) to propose that cognitive restructuring is a form of exposure whose effects are possibly cognitively mediated. One exception to the pattern of equivalence in CT and BT is that, for OCD, ERP appears to be more effective than CT (Fisher & Wells, 2005; McLean et al., 2001; but see Ost, Havnen, Hansen, & Kvale, 2015). Another exception is that, in social anxiety, the CT component appears to add to the efficacy of exposure (Hofmann, 2004; Mayo-Wilson et al., 2014; Ougrin, 2011; but see Chambless & Gillis, 1993).

It has been suggested that, because with some anxiety disorders cognitive techniques may add little or nothing to the efficacy of BTs, exposure and other BTs are best conducted without the questioning of beliefs or the provision of other CT procedures (e.g., Arch & Craske, 2008). Indeed, a favored approach to the treatment of simple phobias has been and continues to be one that relies primarily or only on BT techniques (Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008; but see Choy, Fyer, & Lipsitz, 2007). Some BTs, like the ones focused on relaxation, do not even directly address feared stimuli which may be taken to call into question the need to engage in cognitive work. There are at least two things to keep in mind regarding these comparisons. First, there is no evidence that these therapies lead to greater symptom reduction than CT (e.g., Borkevec et al., 2002; Mayo-Wilson et al., 2014). Second, the fact that these therapies, which do not directly address thinking, are effective does not directly inform about their mechanisms. Recall that Beck (1970) accounted for the efficacy of

![FIGURE 1](image-url)  
**FIGURE 1** Meta-analytic plot of the comparative efficacy of “pure” CT compared to “pure” BT in anxiety disorder RCTs identified by Adams et al. (2015) and Bell et al. (2013). Positive values indicate a superiority of CT over BT. Hedge’s \( g \) was calculated as a between-groups effect size of the end-scores of a trial primary outcome measure, selected by RJD and JRK from a results-blinded list of trial outcomes prepared by LLL. No notable changes in effect size or statistical significance resulted from controlling for pre-treatment severity differences by meta-regressing the Hedge’s \( g \) of the pre-treatment score differences between treatments (analyses available upon request). Egger’s test did not detect the presence of a significantly asymmetrical funnel plot potentially indicative of publication bias (\( p = 0.169 \)), and Henmi and Copas’ (2010) test of publication bias proffered a similar between-groups effect estimate (\( g = 0.017 \)).

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behavorial therapies, giving the specific example of desensitization, in cognitive terms.

TECHNIQUES VS. MECHANISMS

It appears to be a frequent misunderstanding of comparative psychotherapy research, including component trials, that outcome studies inform about the mechanisms of treatments. Various authors have cautioned against conflating the efficacy of therapeutic procedures with their mechanisms of action (e.g., Hofmann, 2008; Lorenzo-Luaces et al., 2015), and the separation between therapeutic procedures and mechanisms has long been recognized (e.g., Beck, 1970; Foa & Rauch, 2004). Jacobson et al. (1996) explained it this way:

Of course, it is also possible that BA-focused treatments are more effective ways of changing the way people think than treatments that explicitly attempt to alter thinking. Perhaps the exposure to naturally reinforcing contingencies produces changes in thinking more effectively than the explicitly cognitive interventions do. (p. 303)

Although it may be tempting to assume that BT and CT produce symptom change via different theorized mechanisms (see DeRubeis, Brotman, & Gibbons, 2005), they may also do so by the same mechanisms (Hofmann, 2008). It is possible that both treatments work because they change thinking. A pattern of findings that would be consistent with this hypothesis is that both therapies change cognition and that the cognitive changes lead to symptom change irrespective of how the cognitive changes are achieved. This latter point is one of contention in the research literature because some have erroneously assumed that a cognitive theory of change implies that change in cognition leads to symptom change uniquely in CBTs (see Hollon, DeRubeis, & Evans, 1987). To the contrary, cognitive theories highlight the mechanistic role of cognition in psychopathology (Lorenzo-Luaces et al., 2015).

Given that findings from comparative outcome studies can, at best, provide food for thought about mechanisms of change, what is needed is more research on the psychological changes that account for symptom change in psychotherapy. In the context of component designs (e.g., Hofmann, 2004), as well as in other kinds of randomized comparisons, investigations of the mediation of the effects of psychotherapy promise to advance the understanding of the workings of psychotherapy.

TEMPORALITY

One of the greatest challenge to our understanding of how BT and CT work is that most studies that explore these questions have been unable to rule out reverse causality. Inferences about causality rest on the ascertainment of the correct temporal relation of the criterion and predictor variables. One must have confidence that change on the predictor variable preceded the change in the criterion. This has sometimes been interpreted to mean that pre-post changes in an outcome measure, regressed on an index of early change, establishes temporal precedence. However, it frequently is the case that substantial portions of pre to post changes in both proposed mediator and the outcome occur early in treatment, making early measurement of the mediator variable a crucial step in establishing causality. Changes in the criterion variable must be assessed subsequent to the assessment of change in the mediator if the aim is to rule out reverse causality. Only a small minority of tests of relations between symptom changes and proposed mediators of those changes has conformed to this pattern.

THIRD VARIABLE CONFOUNDS

Even in studies with the temporal features that allow reverse causality to be ruled out, third variable confounds can limit the inferences that can be made. Third variable confounds can never be entirely ruled out because the multitude of variables that may be confounding the relation between a therapeutic procedure, a mechanism, and symptom change are unknown and potentially unknowable. With observational studies the best one can do is to test for the most plausible confounds, using the best available measures of the potentially confounding construct. Experimental designs can protect against many of the third variable confounds, but unobserved variables may still act as proxies for the purported mediating construct. Yet, as noted by Kazdin (2007), most of the experimental designs in psychotherapy are manipulations of the therapy, not of the proposed mediator.

In the study of change in psychotherapy, the domains most commonly hypothesized to account for symptom change have been: common factors, especially the therapeutic alliance; cognitive change; and behavioral changes. Ideally, a study that was attempting to address questions about causality in psychotherapy would include measures or manipulations of these phenomena. Given that the literature is replete with studies that measure only one or two of these variables, any conclusion that can be drawn about common factors, cognitive change, and behavioral change is necessarily tentative.

STATISTICS AND MEDIATION

Another conceptual hurdle in the understanding of the relationship between therapeutic procedures,
cognitive change, and symptom change regards the role of statistical analyses. Statistical tests cannot determine causality. It does not matter how robust the test’s statistic is or whether the test is conducted in the context of so-called “causal modeling.” The crucial matter is whether the structure of the data, which is dependent on the assessment schedule, meets the assumptions of causal models. In addition, plausible third variables confounds must be ruled out. Thus, a series of carefully planned single-case studies, in which mediators are experimentally manipulated, and temporal order and third variable issues are addressed, may provide stronger evidence concerning the causal status of a mediator than would the use of more seemingly sophisticated data analyses developed in a large sample (if there is overlap in the time periods referenced in the mediator and outcome assessments).

Furthermore, an assumption in the writing of several commentators is that mediational tests are the sine qua non for establishing causality (see Hundt, Mignogna, Underhill, & Cully, 2013; Kazdin, 2007). As it relates to cognitive change procedures and cognitive change, in the mediation framework proposed by Baron and Kenny (1986), evidence concerning cognitive change as a mediator of symptom change is given by answers to the following questions:

1. Are cognitive change procedures more efficacious than (at least some) other procedures?
2. Do cognitive change procedures generate more cognitive change than those other procedures?
3. Does superiority in cognitive change account for superior symptom change in the context of cognitive change procedures vs. those other procedures?

This framework for conducting tests of mediation is widely accepted, but can easily be misapplied. Two change procedures can result in equivalent magnitudes of changes both on the outcome variable and on a putative mediator variable. For example, when cognitive and behavioral techniques produce equivalent outcomes, they may do so because they are equally effective at changing cognitions (or behavior). A study may find that cognitive and behavioral techniques are equally effective in changing symptoms, cognition, and behavior. However, a traditional mediational analysis will not be informative unless a condition is included in the design and analysis that produces less change in the mediator(s) and the symptoms because there would be no effect to mediate. As regards the role of cognitive change in producing symptom change, there are four guiding questions:

1. Are cognitive change procedures more efficacious in reducing symptoms than other procedures?
2. Do cognitive change procedures generate more cognitive change than other procedures?
3. Does cognitive change lead to symptom change?
4. Is cognitive change a specific predictor of symptom change only in the context of cognitive change interventions?

Although some writers have assumed that the cognitive change model implies that the answer to all of these questions should be affirmative (e.g., Longmore & Worrell, 2007), we have argued that only the third question is a test of the cognitive theory of change (Lorenzo-Luaces et al., 2015). In agreement with Jacobson et al. (1996) it is even possible that a noncognitive procedure will produce more cognitive change than an explicit cognitive focus. The question is whether cognitive change, once it has been produced, leads to symptom change.

**Behavioral Change Strategies, Cognitive Change, and Symptom Change**

Existing evidence largely supports cognitive changes as mechanisms of change in cognitive, behavioral, and cognitive behavioral therapies. Most of this research has been conducted in the context of social anxiety, panic disorder (Smits et al., 2012), and depression (Lorenzo-Luaces et al., 2015). Below, we review evidence for the role of cognitive change in symptom change in CT, BT, and CBT.

**Social Anxiety**

Cognitive models of social anxiety highlight the etiological role of cognitive biases in the perceived likelihood that negative social events will occur (Smits, Rosenfield, McDonald, & Telch, 2006), as well as in the overestimation of costs associated with these events (Clark & Wells, 1995). Moreover, it has been suggested that individuals with social anxiety tend to believe they are less socially desirable than they actually are (Moscovitch, 2009).

The proposed cognitive mediators of outcomes in social anxiety co-vary with symptom change in CBTs (Hofmann, 2008; Wilson & Rapee, 2005). For example, Boden et al. (2012) reported that changes in maladaptive interpersonal beliefs fully accounted for changes in social anxiety over the course of CBT for social anxiety. Hoffart, Borge, Sexton, and Clark (2009) explored four cognitive-
behavioral and four interpersonal processes of change in CBT and interpersonal psychotherapy (IPT) for social phobia. The lone IPT variable that predicted symptom change, perceived acceptance by others, is arguably a cognitive construct. By contrast, change in each of the four cognitive-behavioral variables—self-focus, estimated probability of negative social events, estimated cost of negative social events, and safety behaviors—predicted changes in social anxiety. Changes in positive, but not negative, self-view have been reported to co-vary with the reduction of social anxiety symptoms following acute CBT and over a 1-year follow-up (Goldin et al., 2013). Changes in self-focused attention have also been reported to account for treatment outcomes in individual CT and in group-based CBT (Hedman et al., 2013), whereas change in negative and positive self-statements predict outcomes in ACT and CBT (Niles et al., 2014).

Thus, a variety of cognitive constructs have shown associations with symptom improvement in CT, CBT, IPT, and ACT. An issue with many of these studies, however, is that the measurement of the predictor and criterion variables is contemporaneous. In one study that accounted for the temporal order of change in a mediator and change in outcome, Goldin et al. (2014) reported that the success (but not the frequency of use) of cognitive reappraisal strategies predicted subsequent decreases in social anxiety symptom. Decreases in social anxiety did not predict the successful use of cognitive reappraisal strategies. This study is encouraging in suggesting that cognitive process variables predict symptom change in social anxiety. However, given the large number of cognitive constructs that have been reported to co-vary with outcomes in social anxiety, it is likely that at least some of them are products rather than predictors of symptom change. Illustrating this point, Smits et al. (2006) found that changes in probability biases for negative social events predicted changes in social anxiety. However, after symptoms improved, there was a reduction in patients’ estimates of the costs attributed to negative social events. This pattern of results suggests that whereas changes in probability biases were causally related to symptom change, changes in cost estimates were the consequence of symptom change. These findings were replicated successfully and tested against the therapeutic alliance as a competing predictor of change (Calamaras, Tully, Tone, Price, & Anderson, 2015). In this study, the alliance was not found to predict symptom change.

PANIC
In cognitive models of panic disorder, catastrophic misinterpretations of interoceptive cues are implicated in the etiology and maintenance of the pathology (Clark, 1986). Anxiety sensitivity, the attribution of negative somatic, cognitive, and social consequences to anxiety, has been specifically reported to render individuals vulnerable to panic disorder (Reiss, 1991). Additionally, panic self-efficacy, the perception of one’s ability to cope with panic attacks, has also been implicated in the maintenance of the disorder (Casey, Oei, & Newcombe, 2004).

In one study, fear of bodily sensations and anxiety sensitivity, jointly “fear of fear,” were reported to mediate the superiority of exposure relative to a wait-list control (Smits, Powers, Cho, & Telch, 2004). Casey, Oei, Newcombe, and Kenardy (2004) reported that changes in catastrophic misinterpretations, as well as changes in self-efficacy, co-varied with symptom change in CBT. Others, however, have reported that changes in self-efficacy, but not in catastrophic beliefs, correlate with symptom change (Fentz et al., 2013; Hoffart, 1995).

Attending to the temporal order of cognitive and symptom change, Teachman, Marker, and colleagues have reported that cognitive change precedes and predicts symptom change in CBT for panic (Teachman, Marker, & Clerkin, 2010; Teachman, Marker, & Smith-Janik, 2008). In one study, changes in automatic panic associations predicted changes in symptom severity (Teachman et al., 2008). In another study, changes in catastrophic misinterpretations predicted subsequent change in overall symptom severity, panic attack frequency, panic apprehension, and avoidance behavior (Teachman et al., 2010). Gallagher et al. (2013) also provided evidence for the temporal precedence of changes in anxiety sensitivity and also found changes in self-efficacy to precede symptom change. These authors observed that overall change in anxiety sensitivity was greater than change in self-efficacy, and that changes in self-efficacy occurred later in treatment than changes in anxiety sensitivity. Taken together, these studies support the mediational roles of anxiety sensitivity, catastrophic misinterpretations, and panic self-efficacy as cognitive mediators of treatment effects in CBTs for panic disorder (Sandin et al., 2015).

PTSD
Current theories of posttraumatic disorder (PTSD) highlight the causal role of associations between threatening (unconditioned fear stimuli) and non-threatening (i.e., conditioned) stimuli in fear structures of traumatic memories (Cahill & Foa, 2007). Additionally, trauma-related cognitions about the self, others, and the world—most commonly measured with the Post-Traumatic Cognitions Inventory (PCTI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999)—
have also been implicated in the etiology and maintenance of the disorder.

In prolonged exposure (PE), changes in negative cognitions about the self have been reported to co-vary with changes in PTSD symptoms, whether or not the intervention includes cognitive restructur- ing (Fo&Rauch, 2004). In a sample of patients with a severe mental illness comorbid with PTSD, changes in posttraumatic cognitions were found to mediate the superiority of CBT relative to treatment as usual (Mueser et al., 2008). Research attending to the temporality of cognitive change and symptom change suggests that these findings do not reflect an epiphenomenal status for the cognitive changes. In patients who received trauma-focused CT for PTSD, weekly changes in trauma-related cognitions predicted subsequent reduction in symp- toms (Kleim et al., 2013). Similarly, using data from a sample of patients who received PE, Zalta et al. (2014) reported that session-to-session changes in trauma-related cognitions predicted subsequent changes in PTSD symptoms, but not the other way around. Thus, there is support for the cognitive model of symptom change in treatments for PTSD, although more research, with tests of additional measures of trauma-relevant cognition, would help advance our understanding of how treatments for PTSD work.

**OCD**

Cognitive theories of obsessive–compulsive disor- der (OCD) highlight the role of various cognitive variables. Overly attaching significance to one’s thoughts is central to Rachman’s influential cogni- tive theory (Rachman, 1997). Intolerance of uncer- tainty, overestimation of threat, the belief that thoughts should be controlled, inflated sense of responsibility, and perfectionism have also been implicated (Obsessive Compulsive Cognitions Working Group, 2003), with disagreement among the authors as to which cognitions are key to the etiology of OCD (Grayson, 2010; Gwilliam, Wells, & Cartwright-Hatton, 2004).

There are few studies that have explored medi- ators of change in psychotherapy for OCD, a surprising state of affairs given the number of purported cognitive mediators. Interestingly, many of the studies that have been conducted have ade- quately addressed the temporal relation between the purported mediators and outcomes, with mixed results. Woody, Whittal, and McLean (2011) reported that mediational tests conducted in a pre-post fashion suggested that the superiority of CBT relative to stress training were mediated by changes in negative cognitions. However, when these authors utilized the full session-by-session data available to them, changes in cognition were actually predicted by changes in symptoms rather than vice versa. An almost identical pattern of results was reported by Olatunji et al. (2013): it was symptom change that preceded and predicted changes in cognition and in avoidance behavior. Interestingly, changes in depression preceded chang- es in OCD symptoms. In a recent trial, Wilhelm, Berman, Keshaviah, Schwartz, and Steketee (2015) reported that, in CT for OCD, changes in perfec- tionism and certainty beliefs predicted subsequent change. These authors also reported that changes in maladaptive schemas related to dependency and incompetence predicted symptom improvement.

Taken together, these studies do not provide strong support for a causal role of any cognitive change variable in OCD treatment. An alternative mediator to cognitive change—such as behavioral change—has not successfully emerged as a predictor of change. It is possible that a cognitive mediator accounts for change in OCD but it not well captured by existing measures. The findings that changes in depression precede changes in OCD, along with the findings that changes in thoughts of dependency and incompetence predict symptom change, may be taken to suggest that cognitions that have been more typically thought of as depressotypic may func- tion as one of the mechanisms of change in OCD treatments.

**MAJOR DEPRESSION**

Cognitive models of major depression highlight the role of negatively biased cognitive processes (e.g., overgeneral memory style) and content (e.g., negative schemata, dysfunctional attitudes), as well as deficiencies in the ability to use cognitive reappraisal to modulate negative moods. This liter- ature has been reviewed elsewhere (see Lorenzo- Luaces et al., 2015). Contemporaneous associations between cognitive change and symptom change during treatments for depression have been observed in numerous investigations (e.g., Cristea et al., 2015; Hundt et al., 2013). In a recent meta-analysis, for example, Cristea et al. (2015) reported a very strong correlation between symptom change and cognitive change in treatments for depression ($r = 0.77$). Although these findings are consistent with the proposal that cognitive change produces symptom change, they are also consistent with the converse. Support for cognitive mediation models has been obtained in studies that have attempted to address reverse causality by modeling subsequent change in depression using prior cognitive change (Forman et al., 2012; Segal et al., 1999; Segal et al., 2006; Strunk, DeRubeis, Chiu, & Alvarez, 2007; Tang & DeRubeis, 1999; Tang, DeRubeis, Beberman, &
Pham, 2005). Although these studies provide general support for cognitive theories of change in symptoms, no one variable has emerged as the clear predictor of symptom change in therapy. For example, Strunk et al. (2007) found that patients’ competence in behavioral strategies, automatic thoughts, and core schematic work predicted a lower risk of relapse.

Given how heterogeneous depression is, it is likely that patients differentially benefit from different interventions. Adding support for this hypothesis, Sasso et al. (2015) found that more anxious patients and those with less recurrent depression benefitted more therapists’ use of behavioral strategies than cognitive ones. By contrast, Keefe, Webb, and DeRubeis (2016) found that patients with depression and personality disorders benefitted from a focus on maladaptive core beliefs but did not experience benefit from other techniques such as a focus on automatic thoughts and behavioral change.

**Generalized Anxiety and Specific Phobias**

There have been relatively few investigations of the mediation of change in treatments of generalized anxiety disorder (GAD) and specific phobias. Current etiological models of GAD highlight the causal roles of intolerance of uncertainty, positive beliefs about worry, and the avoidance of emotional/internal experiences (including emotional contrasts). Few studies have explored these constructs as mediators or explanatory variables in CBTs for GAD, although they each have some support in basic research (see Newman, Llera, Erickson, Przeworski, & Castonguay, 2013). Goldman et al. (2007) reported that decreases in intolerance of uncertainty preceded changes in worry over the course of treatment. Similarly, Bomyea et al. (2015) reported that changes in intolerance of uncertainty preceded and predicted change in GAD symptoms.

Cognitive models of specific phobias highlight the role of exaggerated judgments of dangerousness, disgust, unpredictability, and uncontrollability associated with feared stimuli (see Armfield, 2006). Additionally, these models highlight the role of low expectations for the capacity to cope with the consequences of coming into contact with the feared stimuli, which may include the ability to tolerate uncomfortable physiological sensations. In one study examining behavioral exposures vs. behavioral experiments designed to test maladaptive beliefs, both treatments showed substantial symptom and cognitive change, and cognitive change was associated with symptom levels at posttreatment and follow-up (Raes et al., 2011).

**Cognitive Specificity**

There is much excitement regarding the possibility that different treatments could work via different hypothesized mechanism. Statistical tests that reveal the moderation of a predictive effect (e.g., tests of moderated mediation) may be taken as evidence for this. Although the interpretation of mediators and predictors within a treatment has been widely discussed, moderated mediation and related findings have received relatively less attention and are consequently less well-understood (Gelfand & DeRubeis, 2015). Various findings of this nature, however, have been reported (e.g., Cottraux et al., 2001; Hedman et al., 2013; Niles et al., 2014). Here we focus on the interpretation of moderated relations between cognitive change and symptom change across treatments for panic disorder.

Arntz (2002) reported equal amounts of change in catastrophic interpretations following CT and BT for panic. However, posttreatment beliefs correlated with severity of symptoms at a follow-up in CT but not in BT. In a sample of patients receiving CBT, imipramine, or combined treatment, analyses suggested that changes in panic-related cognitions, which were equal across conditions, co-varied with changes in panic severity in CBT or combined treatment but not imipramine monotherapy (Hofmann et al., 2007). In a comparisons of capnometry-assisted respiratory training (CART) versus training in CT strategies, Meuret, Rosenfield, Seidel, Bhaskara, and Hofmann (2010) found support for the therapy-specific mediators. In CART, reduced carbon dioxide in the bloodstream from hyperventilation (hypocapnia), but not cognitive reappraisal, predicted symptom change. In cognitive training, the opposite pattern was obtained; cognitive reappraisal, but not hypocapnia, predicted symptom change.

Thus, in several studies of treatments for panic disorder, a “non-cognitive” intervention produced similar amounts of cognitive and symptom change relative to an explicitly cognitive intervention, yet the relation of cognitive change to symptom change was only found in the cognitive intervention. It is difficult to reconcile these findings with cognitive theories of panic, or cognitive theories of psychopathology more broadly, because these models assume that changes in cognitive variables are mechanisms of symptom change across treatment modalities (Lorenzo-Luaces et al., 2013). One possibility is that the cognitive change variables examined play a causal role only in interactions with another variable that is present only in the treatments in which cognitive change was predictive of symptom change (see DeRubeis et al., 1990). For example, catastrophic misinterpretations of
panic, in conjunction with increased self-efficacy, might mediate changes in both CT and respiratory training. A further possibility is that the treatments intervene at different stages of the pathological process. For example, one account of panic disorder could be that hyperventilation leads to hypocapnia, which produces dizziness and discomfort, which is misinterpreted, resulting in more severe panic symptoms. Breathing retraining might intervene “early” in this cycle, whereas cognitive interventions address the appraisal of bodily sensations have already been experienced. In such a case, the cognitive changes observed in breathing retraining are only a product of reduced symptoms.

Distinguishing Cognition and Behavior

Thus far in this review we have used “cognition,” “behavior,” and their adjectival forms to reflect the way previous authors have used them in discussions of therapeutic techniques or mechanisms. However, clear, consensually-agreed-upon definitions of these terms do not exist. In some cases, therapeutic procedures or mechanisms are easy to characterize as cognitive or behavioral. For example, searching for evidence for or against a belief in the context of therapy is readily seen as a cognitive procedure, just as engaging in in vivo exposure following a habituation rationale is easily seen as behavioral. However, cognitive techniques may be used to facilitate exposure (Arch & Craske, 2008), and cognitive therapists use behavioral techniques to facilitate tests of patients’ beliefs (McManus, Van Doorn, & Yiend, 2012). Thus, it behooves investigators to specify what counts as cognitive or behavioral vs. what does not. Similarly, cognitive and behavioral mechanisms of therapy must be operationalized in a way that allows for falsification. Arguing that all behavioral changes must, by definition, entail cognitive changes, or that all cognition is behavior, is unlikely to lead to more valid models of psychopathology.

A related issue to the conceptualization of cognition and behavior is their measurement. Measuring cognitive and behavioral activity objectively in the context of psychotherapy is a difficult enterprise. Although self-reported measures are the most convenient way of measuring cognitive or behavioral change, they are subject to various biases that have been well-articulated in the research literature. It remains to be ascertained whether self-report measures are more valid ways of ascertaining cognitive change than behavioral change or vice-versa. Moreover, as avoidance behaviors and maladaptive cognitions are symptoms of psychopathology, assessments of both behaviors and cognitions are difficult to disentangle, conceptually and practically, from symptom measures.

Recommendations for Future Research

Although the existing literature allows one to draw conclusions regarding the status of cognitive and behavioral techniques, as well as the mediational role of cognitive change, more research is clearly needed, particularly research employing experimental methods. Dismantling studies and randomized comparative trials only infrequently are complemented by mediational analyses that could help to clarify what drives change in the interventions. It will be important to conduct more experiments where interventions are compared and mediational hypotheses are tested. As Kazdin (2007) has noted, however, it is important for research to test the effects of the manipulations of mechanisms, rather than continuing a focus on the manipulation of techniques or packages of techniques.

Researchers who conduct work on mediators of change in nonexperimental contexts should ensure that third variables and competing mediators are measured. Given that behavioral change, cognitive change, and common factors are the most frequently cited mediators of symptom change, it will be important to measure and model these variables concurrently. Moreover, it might also be important to measure multiple aspects of a given construct. For example, in the behavioral domain, a reduction in safety behaviors along with behavioral exposures are both purported mechanisms of symptom change. In the cognitive domain, the multiplicity of cognitive variables that have been implicated should also be measured, particularly for disorders like depression and OCD, where multiple cognitive vulnerabilities are hypothesized to be at play. Additionally, the role of emotions and emotional arousal, whether by themselves or in interactions with other variables, needs to be explored. Although non-CBT treatments have traditionally focused more on emotional experiences than CBTs (Whelton, 2004), there is evidence that clients’ in-session emotional involvement relates to outcomes, at least in CT for depression (Castonguay, Goldfried, Wiser, Raue, & Hayes, 1996). Hayes and colleagues (Grosse Holtforth et al., 2012; Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005; Hayes et al., 2007) have proposed, and found evidence for, a model of change in depression treatment where behavioral interventions foster emotional arousal that, when combined with cognitive processing, leads to symptom change. These findings, which can be conceptualized using the concept of cognitive-affective schemata (Beck & Haigh, 2014), warrant further exploration.

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Mechanisms of Change: Cognitive and Behavioral Treatments

The suggestion that behavioral exposure is superior to cognitive restructuring alone for OCD and that cognitive restructuring adds to behavioral exposures for social anxiety may be dismissed, given that these are not universal observations. However, it is noteworthy that in a disorder in which cognitive procedures appear to be especially potent—social anxiety—strong evidence has been obtained for cognitive change as a mechanism of symptom change (see McMillan & Lee, 2010). By contrast, in a disorder in which cognitive procedures appear to be less potent (OCD), there is little if any evidence for a cognitive mechanism of symptom change. (These observations, however, should not be taken to imply that efficacy findings allow one to make claims about causal mechanisms.) Much is made and little is done about the potential for knowledge of mediator variables to inform treatment research, but the existing data on the cognitive mediators in mood and anxiety disorders could be used to inform treatment. For example, in the context of treatment for social phobia, treatment could be targeted especially to the perception that negative social events are likely to occur, as opposed to treating symptoms or functioning as the targets.

In broad terms, research supports cognitive vulnerability models of depression and anxiety disorders. Additionally, cognitive changes appear to correlate with and predict symptom change in therapy for depressive and anxiety disorders. Given this state of the literature, it behooves researchers to characterize the exact nature of the cognitive vulnerability to psychopathology, as well as the nature of the cognitive variables implicated in symptom change. Often the differences between different cognitive vulnerability theories lie in the stage of information processing that they focus on. For example, whereas some investigators have emphasized individual differences in the propensity to attend to negative stimuli (MacLeod et al., 2002), others have focused on individual differences in interpretation of life events (Abramson, Metalsky, & Alloy, 1989). The latest characterization of the cognitive model provided by Beck attempts to represent a more integrative picture of the cognitive vulnerabilities to psychopathology (Beck & Haigh, 2014), highlighting the roles of attention, memory, interpretative biases, emotional processing, and cognitive schemata in the development of psychopathology. However, it may be that vulnerabilities in different stages of information processing lead to different types of psychopathology. Reviewing the literature on cognitive biases and cognitive bias modification, Hallion and Ruscio (2011) asserted that:

anxiety is associated with biases in the early, automatic stages as well as the later, strategic stages of attention (Bar-Haim et al., 2007), whereas depression is associated with [cognitive] biases only in the later stages. (e.g., Gotlib et al., 2004; Joormann, 2004)

Temporal precedence of cognitive change has been easier to establish in anxiety disorders than in depressive disorders. It is possible that this reflects the temporal precedence of cognitive biases in anxiety relative to depression, thought this question merits further exploration.

Conclusions

As has been noted extensively (DeRubeis et al., 2005; Haaga, 2007; Kazdin, 2007), characterizing the process of change in psychotherapy is extremely challenging. Within a therapeutic orientation, a given therapeutic package will encourage a set of therapeutic procedures, such as enhancing motivation for behavioral change, some of which may overlap with those of therapeutic packages, within or outside of that orientation. Factors common to different interventions may also drive symptom change, confounding the relation between specific procedures and improvement. Furthermore, a given procedure might not affect the psychological mechanism(s) it is intended to target, and might instead affect a mechanism that is not its intended target.

As noted by Beck (1991), a psychotherapeutic approach is not a dissociated collection of techniques, but rather a set of procedures that follow from “a comprehensive theory of psychopathology that articulates with the structure of psychotherapy” (p. 368). Thus, productive considerations of the effectiveness or validity of a procedure or mechanism should be linked to the theory of the disorder or pathological process in question. Critiques of a cognitive model of the development, maintenance, and resolution of psychopathology will advance the discussion insofar as they are accompanied by an alternative theory, along with proposals, and evidence, regarding alternative noncognitive mechanisms of change. In our view, oft-cited critiques of the theoretical model that underlie CBTs (Kazdin, 2007; Hayes, 2004; Longmore & Worrell, 2007) have employed oversimplifications of the CBT model. The available data across the sciences supports cognitive models (Beck & Haigh, 2014; Hofmann et al., 2013) in suggesting that some individuals are more likely than others to be exposed to negative environmental stimuli, less likely to be exposed to positive stimuli, or more likely to
attend preferentially to negative information. In the contexts of stress, these individuals are more likely to experience distress and less likely to engage the kinds of reappraisal and coping strategies that would produce naturalistic recovery. Cognitive and behavioral therapies represent an attempt to activate these resources. The distinctions between behavioral and cognitive change strategies and procedures are, to some degree, artificial inasmuch as CT procedures are often used before and after behavioral exercises, and vice-versa. Moreover, the relative efficacy of these procedures, even when considered in specific contexts, does not provide dispositive evidence concerning mechanisms. To date, the most parsimonious account of change produced in psychotherapy is one that invokes the importance of changes in cognitive systems or contents. Although the research literature is limited in its ability to provide strong confirmations of the causal hypotheses embedded in cognitive theories of change, a compelling, evidence-based competing theory of change has yet to emerge.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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