

## Mediators and Mechanisms of Psychotherapy: Evaluating Criteria for Causality

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Although the efficacy of psychotherapy for treating many disorders has been demonstrated in randomized clinical trials (Ekers, Richards, & Gilbody, 2008; Hofman & Smits, 2008; Hoglend, 1999), little is known about the mechanisms through which psychotherapy achieves its effects (Kazdin, 2005). The purpose of this review is to examine the state of research on the mechanisms of psychotherapy. First, a definition of mechanism is presented and the importance of studying mechanisms in psychotherapy is discussed. Then, methods of demonstrating mediation in empirical studies are reviewed along with selected areas of empirical research on mechanisms of cognitive therapy. Finally, suggestions for future directions in studying mechanisms are offered.

A mechanism is defined as a step or series of steps in a process through which change is produced (Johansson & Hoglend, 2007). Each intermediate step in a process of change can also be thought of as a mediator—a mediator is an intervening variable that is causally related to the mechanism of change (Doss & Atkins, 2006). This definition of mediation is crucial to research on how psychotherapy works, as research in the aim of elucidating psychotherapy mechanisms is primarily concerned with identifying mediators of change. In psychotherapy research, a mediator is a construct that explains why therapy results in symptom improvement. Mediation must therefore take place during the course of psychotherapy and before symptom change.

It is important to distinguish mediation from causality. Logically, causality is a necessary condition for mediation; only when something has a causal effect can the intervening mediating pathway be established. A common misconception in psychotherapy research until recently was that establishing causal effects gives information about possible mediators of those effects (Kazdin, 2007). For example, if cognitive therapy were shown to be effective, it may have been assumed to work through cognitive change. However, this line of reasoning is faulty. Knowing *that therapy works* is not in any way informative of *the way in which therapy works*—based on efficacy research alone, it is likely to be difficult to draw any conclusions regarding the mechanism(s) of change without measurement of potential mediators. In the example of cognitive therapy, there is a chance that affective change (or any variable, no matter how implausible) may actually explain why cognitive therapy was shown to be effective. Mediators must be evaluated on their own terms, rather than being inferred from the modality of therapy.

It is important to study the mechanisms of psychotherapy for at least four reasons. The first reason is based on the extremely high proliferation of different types of psychotherapy. It is highly unlikely that all of these types of therapy work in unique ways; therefore, identifying common mediators across psychotherapies may add parsimony to the varied theories of what happens when psychotherapy works (Johansson & Hoglend, 2007). Second, identifying mediators may advance the understanding of differential responses to treatment. Even in the most successful clinical trials, only 60% of depressed patients show clinically significant symptom improvement (DeRubeis, Tang, & Beck, 2001). Third, identifying mediators may maximize treatment effectiveness (Laurenceau, Hayes, & Feldman, 2007). Psychotherapies contain multiple components, and yet each component may not be causally related to the mechanism by which therapy works. For example, although Cognitive Behavioral Therapy (CBT) includes both cognitive and behavioral components, it is possible that only cognitive or only behavioral components are causally related to change depending on the disorder being treated. Identifying mechanisms may allow therapists to focus on only those aspects that are actively contributing to the patient's improvement, making therapy much more efficient and economical. Knowledge about how therapy works also allows for generalization from clinical trials. A common criticism of randomized clinical trials (RCTs), a clinical experiment in which participants are randomly allocated to receive different forms of treatment, is that the various constraints and artificialities imposed on participants prevent the results from transferring to "real world" psychotherapy (Doss & Atkins, 2006). If it was known why a specific psychotherapy worked, this knowledge would be potentially transferrable across a wide range of community settings. Fourth, there is the potential for benefits outside the realm of psychotherapy. Specifically, knowing about the process by which maladaptive functioning changes into adaptive functioning may reflect upon the processes underlying different forms of psychopathology and healthy functioning in general.

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### Criteria for Mediation

Causality may be logically identified when an experiment utilizes random assignment of participants. Therefore, RCTs by definition are able to establish that psychotherapy causes symptom improvement. However, there is no comparable method to show that a possible mediator is responsible for the effects of psychotherapy. This is because the variables that may be responsible for change are left to vary freely rather than being randomly assigned.

The lack of a definitive method for showing that mediators are part of a causal mechanism presents a difficult hurdle for achieving certainty regarding potential mediators. Numerous criteria have been proposed that supposedly strengthen the inferences one can make about whether a putative mediator is actually a part of the mechanism of psychotherapy (Doss & Atkins, 2006; Johansson & Hoglend, 2007; Kazdin, 2007; Nock, 2007; Weersing, 2006). However, the myriad criteria have not been reviewed in a systematic way. It is important to organize these criteria in order to establish a method for evaluating the degree of support for a given mediator as a link in the causal chain between psychotherapy and treatment outcome.

There are two important clarifications to make before discussing the criteria for mediation. First, because RCTs are the gold standard in psychotherapy research, much of the discussion of criteria below centers on research conducted in the context of an RCT; however, with the exception of Criterion 4 (that the mediator was discovered in the context of an RCT), each of the criteria may be applied to non-RCT designs. Second, the distinction between *full mediation* (in which a mediator completely accounts for the association between treatment and outcome) and *partial mediation* (in which a mediator accounts for only part of the association between treatment and outcome) is an important issue but is outside the scope of this paper. The discussion focuses on mediation in the general sense—that is, whether a particular mediator is part of the causal chain linking treatment to outcome.

The first criterion, temporal precedence, is a necessary condition for mediation (i.e., the mediator must occur temporally before symptom change). This criterion had not been explicitly recognized by psychotherapy research until recently, as it only just became common practice to measure mediator variables and symptom changes at multiple time points in psychotherapy studies (Hayes, Laurenceau, Feldman, Straus, & Cardaciatto, 2007; Laurenceau et al., 2007). It is a logical flaw to claim as a potential mediator any changes that occurred after symptoms have already changed.

The most commonly cited evidence used to support mediation are statistical criteria (Baron & Kenny, 1986); see Figure 1 for a depiction of this model as applied to psychotherapy). Each path in the figure signifies a criterion that must be met in order for mediation to be achieved. Path “a” indicates that the relationship between treatment and outcome must be statistically significant. Path “b” indicates

that the relationship between the treatment and mediator must be significant, and path “c” indicates the relationship between the mediator and outcome must be significant. Path “d” is shown as a dotted line, symbolizing that the relationship between treatment and outcome should be significantly reduced when taking the mediator into account. More recent statistical mediation models (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002) have relaxed the Baron and Kenny model by excluding path “d” from the mediation model in order to maximize power to detect significant mediation effects. There are various other statistics that assess mediation (Fritz & MacKinnon, 2007); however, distinguishing between those statistics is beyond the scope of this paper. The point of establishing statistical mediation is that it lessens the possibility that a potential mediator went unidentified due to Type II error alone. For example, consider a case in which multiple mediators (A, B, and C) are examined in the same study. If mediator A meets statistical criteria and mediators B and C do not, then it is *probable* that mediators B and C failed to meet statistical significance because they are less causally implicated in the mechanism of psychotherapy (relative to mediator A), rather than for power reasons alone. Establishing statistical mediation in the context of an RCT strengthens the case for the potential mediator being examined. This is because, if random assignment was correctly implemented, pre-existing conditions cannot account for the effects of the mediator. However, it is still entirely possible that a different variable that changed as a result of therapy accounts for the effects of the mediator being examined. In addition to statistical criteria, there are a number of other criteria that can be used to evaluate the likelihood that a potential mediator is a causal component of therapy. These additional criteria are helpful in ruling out or at least reducing alternative possibilities, thereby strengthening inferences that can be made about potential mediators.

For example, RCTs can examine whether mediators meet a *dose-response* criterion; this means that, the more change in the mediator, the more change in the outcome. Dose-response increases confidence in the conclusion that mediators systematically relate to outcome across the range of possible outcomes; mediators that do not meet the dose-response criterion may only be relevant or related to outcomes in a very narrow range. For example, if a moderate amount of cognitive change is associated with moderate symptom improvement, and a high amount of cognitive change is associated with high symptom improvement, the range over which cognitive change is relevant to the outcome would be greater than if all levels of cognitive change were associated with moderate symptom change only.

The *specificity* of mediators may also be examined. Specificity refers to the fidelity of a mediator to a particular form of psychotherapy. For example, if cognitive change met criteria for statistical mediation in cognitive therapy but not in behavioral therapy, then cognitive change would have higher specificity than if it were shown to mediate the effects of both

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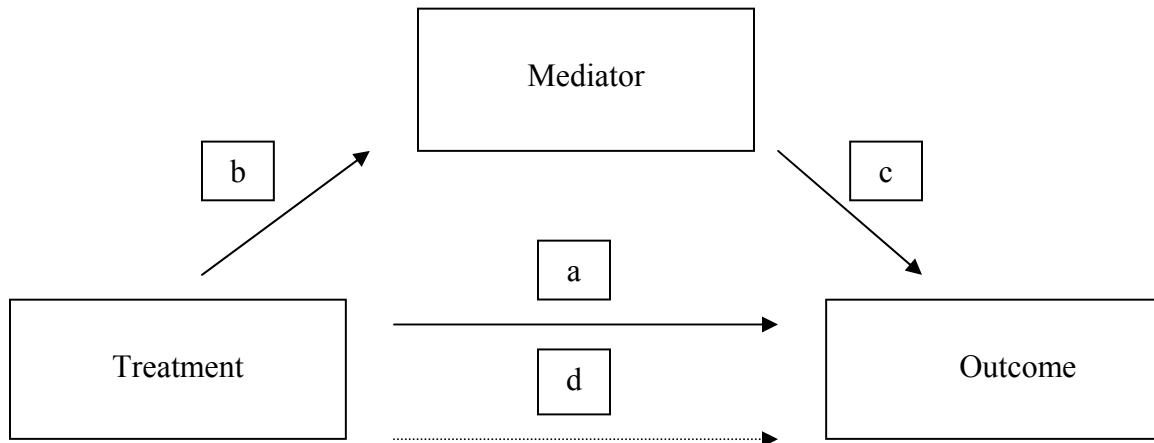


Figure 1. The mediation model of Baron and Kenny (1986) as applied to psychotherapy.

Note. Path “a” indicates that the relationship between treatment and outcome must be statistically significant. Path “b” indicates that the relationship between the treatment and mediator must be significant, and path “c” indicates the relationship between the mediator and outcome must be significant. Path “d” is shown as a dotted line, symbolizing that the path between treatment and outcome should be significantly reduced when taking the mediator into account.

cognitive therapy and behavioral therapy. Specificity says more about the psychotherapy in question than the mediator per se. It is possible that all psychotherapies work through the same or similar mechanisms—in this extreme case, there would be no specificity for mediators even though the mediator would be completely valid. However, if a particular brand of psychotherapy were not tied to a specific mediator, then the validity of the unique components of the psychotherapy could be called into question.

If multiple RCTs for the same psychotherapy found evidence supporting a particular mediator, then the mediator would have high *consistency*. High consistency reduces the possibility that the mediator in question met the statistical definition of mediation due to Type I error. Similarly, *converging evidence*—evidence obtained across a variety of methodologies in a variety of settings—increases confidence in the conclusion that the mediator is not simply an artifact of the context of a particular RCT. Converging evidence may be obtained through lab studies, field studies, and even studies examining different species. For example, suppose that exposure therapy for spider phobia was thought to reduce phobic symptoms by reducing autonomic arousal. Then, evidence that both individuals without clinically significant phobic symptoms as well as rats exhibit diminished autonomic responses to various potentially fear-inducing stimuli after undergoing exposure therapy would constitute converging evidence supporting reduced autonomic arousal as a mediator.

Finally, *plausibility* (the degree to which a mediator makes intuitive sense) has been suggested as another criterion that may increase the confidence that can be placed in a mediator (Kazdin, 2007). However, one difficulty in evaluating a mediator’s plausibility is that there may be

disagreement in regard to whether a proposed mediator is a plausible explanation of change. In general, potential mediators that are recognized as valid constructs have more plausibility compared to potential mediators that are not firmly established as valid scientific constructs. For example, the empirical evidence for repressed memories (Davis & Loftus, 2009) is not as strong as the empirical evidence for cognitive biases (Hallion & Ruscio, 2011). Thus, the idea that reductions in cognitive bias mediated symptom improvement in a trial would be inherently more plausible than the idea that therapy worked by activating repressed memories. Examining mediators that have been well established within other empirical contexts reduces the likelihood that the potential mediator is not a valid scientific construct.

It is important to note that the criteria intended to bolster the case for mediators only lessens the likelihood of alternative explanations (or rules out alternative explanations) instead of providing evidence in favor of mediation effects. However, evidence that some potential mediators meet the aforementioned criteria to a greater extent than other potential mediators does carry some very important implications. Specifically, practical decisions can be informed by taking into account the cumulative evidence for or against any possible mediator. When deciding which psychotherapies should be widely disseminated or which components of psychotherapies should be emphasized, decisions that are normally best made by examining efficacy research may be well-served by also taking into account mechanism research. Based on the rationale that potential mediators meeting criteria are less likely to be the result of extraneous explanations compared to potential mediators not meeting criteria, interventions based on mediators meeting criteria may have a greater likelihood of success. However, this idea

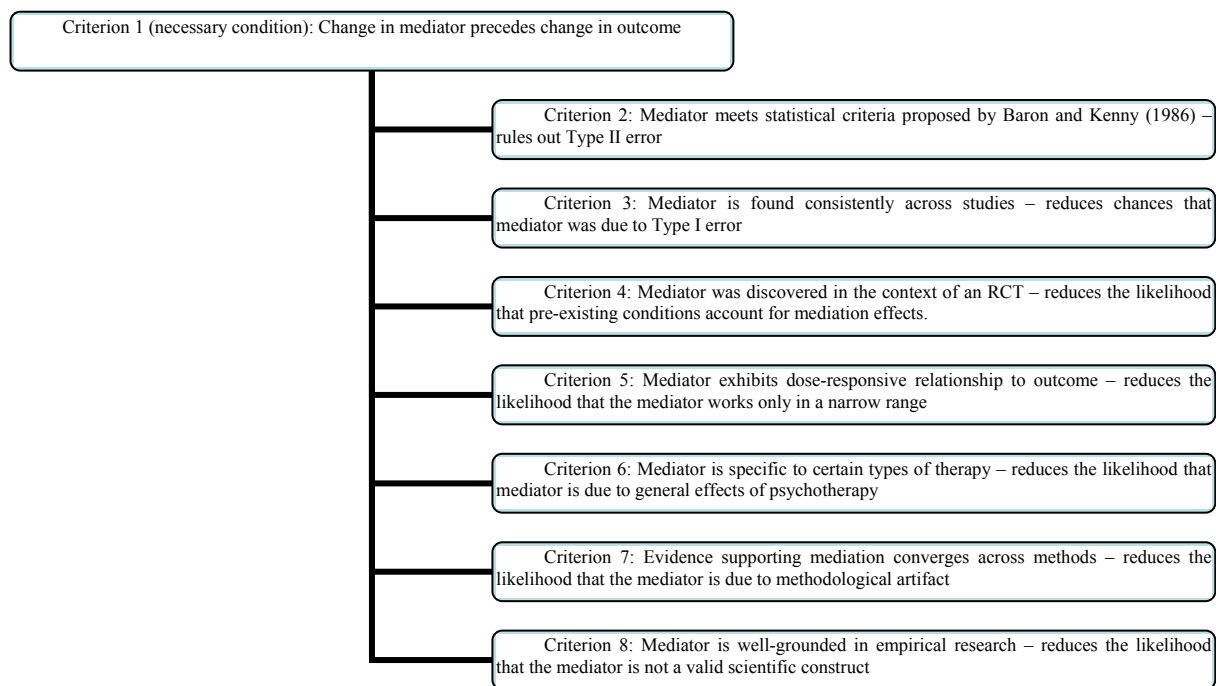


Figure 2. This figure shows how achieving criteria for mediation reduces the likelihood that mediation effects are due to extraneous explanations.

is currently only speculation. Examination of interventions based on potential mediators meeting higher numbers of criteria versus interventions that are not based on this evidence could clarify whether using the additional criteria to evaluate mediation is indeed practically useful.

In addition to the possibility that a potential mediator is actually part of the causal mechanism, evidence to support mediation may be due to a variety of alternative explanations (e.g., statistical artifacts or methodological artifacts). If a mediator meets many of the criteria above, the likelihood that the mediator can be explained by such alternative explanations is decreased. Thus, more confidence can be placed in mediators that meet additional criteria than potential mediators that do not meet said criteria (see Figure 2 for a schematic summary).

### Evaluating Mediation in RCTs: Common Statistical and Methodological Pitfalls

As discussed above, various methods may be used to obtain evidence for mediation. However the primary evidence for mediation should be garnered within the context of RCTs because RCTs can establish causality, a necessary condition for mediation. The most common way to evaluate mediation in RCTs is to use the statistical criteria for mediation proposed by Baron and Kenny (1986). Although these criteria are rather straightforward, there are some subtleties involved in applying them to RCTs that have led to confusion in some research studies. First, when examining the relationship between treatment and outcome (path “a”),

evidence that multiple treatments are equally effective does not prohibit testing for mediation. If any treatment is shown to be significantly related to the outcome, then path “a” is said to be established for that treatment. Second, if a significant path exists between a treatment and mediator, then path “b” is said to be established. Third, a non-significant relationship between mediator and outcome (path “c”) across all participants does not necessarily preclude the possibility that mediation occurred. This is because path “c” may only be significant within the treatment group that improved significantly on the outcome variable. Tests for treatment-by-mediator interactions should be used to detect this possibility. These considerations apply regardless of the particular statistical modeling technique being used to evaluate mediation (e.g., multiple regression, multilevel-modeling, or structural equation modeling) as they each deal with the same fundamental assumptions of mediation.

Even if all statistical criteria for mediation are met, many existing studies purporting to have found mediation effects suffer from methodological limitations related to the timing of mechanism and outcome assessments. As a result, these authors often conclude erroneously that mediation occurred without having determined that the mediator changed before outcome variable changed. Examples of flawed designs are enumerated in Table 1, along with an empirical example of each design variation. One example of flawed methodology includes studies that assess mediators and outcomes at two time points only, prior to the experimental manipulation and after the experimental manipulation. One study used this type of design to find evidence suggesting that CBT helped to

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Table 1

*Examples of flawed designs commonly used for studying mediation effects*

Study	Disorder Studied	Mediator Assessment			Outcome Assessment			Limitation
		Pre	During	Post	Pre	During	Post	
Smits, Powers, Cho, and Telch (2004)	Panic Disorder	Y	N	Y	Y	N	Y	Cannot determine whether the mediator changed prior to the outcome due to lack of assessments during the study
Hofmann (2004)	Social Phobia	N	Y	N	Y	N	Y	Cannot determine whether mediator changed during the study due to only assessing mediator one time
Wilson, Fairburn, Agras, Walsh, and Kraemer, H. (2002)	Bulimia Nervosa	Y	Y	Y	Y	N	Y	Although it is possible to determine that change in the mediator preceded the final outcome assessment, it is not possible to establish that the mediator changed before the outcome because the outcome was not assessed during the study

*Note.* “Y” indicates that the mediator or outcome is measured at the time specified by the column label: “Pre” = prior to treatment, “During” = during treatment, and “Post” = following treatment.

decrease panic symptoms through the mechanism of reducing “fear of fear,” or feelings of anxiety related to physical symptoms (Smits, Powers, Cho, & Telch, 2004). This study consisted of 130 participants who were referred to the study by mental health professionals for treatment of panic disorder. Ninety participants were randomized to the treatment condition and 40 to a waitlist condition. “Fear of fear,” assessed by the Anxiety Sensitivity Index (Peterson & Reiss, 1987) met all of Baron and Kenny’s (1986) criteria for mediation; however, as “fear of fear” was only measured concurrently with panic symptoms, there is no evidence to make the claim that “fear of fear” changed before panic symptoms.

Failing to include pre-treatment measures of mediators also limits the conclusions that can be drawn from a study. For example, one study presented evidence suggesting that cognitive behavioral group therapy reduced symptoms of social phobia through the putative mediator of patients’ appraisals of hypothetical events associated with social phobia (Hofmann, 2004). This study consisted of 90 participants who were randomly selected from among individuals seeking outpatient treatment at a university anxiety clinic. Participants were randomly assigned to receive either cognitive behavioral group therapy or exposure therapy, or were assigned to a waitlist control condition.

Although judgments of events, as assessed by the Social Cost Questionnaire (Foa, Franklin, Perry, & Herbert, 1996) were reported during the study and thus before the post-treatment assessment of social phobia, the lack of a baseline assessment of the mediator prohibits conclusions about whether the mediator changed during treatment.

Designs that do not measure outcome variables during the study are also flawed. One study showed that the effects of CBT and interpersonal psychotherapy (IPT) on symptoms of bulimia nervosa were statistically mediated by changes in dietary restraint (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). This study consisted of 220 participants diagnosed with bulimia nervosa and was conducted on an outpatient basis at two university treatment centers. Participants were randomly assigned to either the CBT or IPT conditions. The study’s design was able to establish that the mediator of dietary restraint (as assessed by the Eating Disorder Examination Questionnaire; Fairburn & Beglin, 1994) changed in response to treatment, and that the mediator changed during the treatment and thus before the post-treatment outcome assessment. However, as no assessments of bulimia symptoms were made during the study, the possibility still exists that those symptoms changed prior to the mediator of dietary restraint. It is worth noting that a substantial body of research ostensibly supporting the

meditational effects of therapeutic alliance (i.e., the degree to which a patient and therapist collaborate, agree on goals, and form a personal bond; Henry, Strupp, Schacht, & Gaston, 1994) has been largely discredited due to failure to measure symptom change during studies (DeRubeis et al., 2001). When symptom change has been included in assessments during the course of treatment, therapeutic alliance has not emerged as a significant predictor of post-treatment symptom change (DeRubeis & Feeley, 1990; Tang & DeRubeis, 1999).

To avoid the pitfalls of these studies, the best possible design would include assessments of potential mediators and outcome variables before and after the treatment is implemented, and also at multiple time points while the treatment is being implemented.

### **Cognitive Therapy for Depression Works Through Cognitive Change**

The methodological limitations described above have the consequence of preventing the accumulation of knowledge about how psychotherapy works. However, one exception is the study of cognitive therapy for depression. This section describes how various studies are converging on the possibility that cognitive therapy works through producing cognitive change, and that cognitive change mediates not only response to treatment but also reduction of relapse rates. Examples of studies providing evidence in support of the criterion for mediation are presented below (see Figure 2 for a list of each criterion identified by number).

The first direct evidence for mediation by cognitive change was provided by Tang and DeRubeis (1999). In this study, cognitive therapy was found to be effective for treating depression in the context of an RCT (thus establishing causality and meeting Criterion 4, that the research was conducted in the context of an RCT). Importantly, symptoms of depression as well as cognitive change were monitored at multiple time points throughout the course of treatment. Cognitive change was assessed with a well-validated measure of cognition in depression, the Patient Cognitive Change Scale (Tang, DeRubeis, Beberman, & Pham, 2005), suggesting that the proposed mediator is a valid scientific construct (Criterion 8). When considered together, two noteworthy findings of this study make a strong case that cognitive change mediated the effects of therapy. First, individuals who experienced “sudden gains,” or large reductions in depressive symptoms from one session to the next, were much more likely to improve than individuals who did not experience sudden gains (meeting the statistical criterion from Criterion 2). Second, the session in which sudden gains occurred was likely to have been preceded by cognitive change in the previous session (Criterion 1), but not during a control session (i.e., two sessions prior to sudden gains). These findings were subsequently replicated in an independent sample (Tang et al., 2005), providing some evidence that cognitive change meets the consistency criteria for mediation (Criterion 3). Additionally, there is some evidence that cognitive change meets specificity criteria

(Criterion 6), as cognitive change has not been found to mediate the effects of Supportive-Expressive therapy (Andrusyna, Luborsky, Pham, & Tang, 2006) on depression. Encouraging findings are also occurring in the domain of relapse prevention. It was recently found that lower rates of relapse in depressed patients receiving cognitive therapy were predicted by sudden gains (Tang, DeRubeis, Hollon, Amsterdam, & Shelton, 2007) as well as by competence in cognitive techniques emphasized during cognitive therapy (Strunk, DeRubeis, Chiu, & Alvarez, 2007). These findings provide more evidence for consistency, and they also suggest that cognitive change is durable and robust rather than a transient effect of cognitive therapy. Thus, cognitive change does reasonably well (meeting six of eight criteria) when evaluated on the criteria of mediation reviewed in this paper. Future research should test for a dose-response relationship (Criterion 5) and use additional measures of cognitive change (Criterion 7).

### **Conclusions and Future Directions**

Research on the mechanisms of psychotherapy is still in its earliest stages. Although much has been written on the ways in which psychotherapy might work, little empirical research has investigated the process of psychotherapy, and a high percentage of this research was plagued by serious methodological flaws. Therefore, there is an urgent need for mechanism research that is conducted with appropriate methodological rigor and that establishes treatment efficacy before evaluating mechanisms. While this area holds great promise for future researchers, moving forward with blind enthusiasm (by not establishing treatment efficacy first) would be just as detrimental to progress as the methodological pitfalls described in this paper.

One way that mechanism research could have a significant impact is by finding some common ground across the vast number of psychotherapies in existence. New psychotherapies are developed frequently, each laying claim to a somewhat unique theoretical basis. Thus, the theoretical landscape of psychotherapy is likely much more complex than it needs to be, which has the important practical implication of making treatment choices seem overwhelming and impossible to navigate for clients. Mechanism research has the potential to reveal the processes that are shared between different therapies. This development would increase parsimony and would hopefully lead to the incorporation of techniques relevant to the ways in which psychotherapy works across its disparate forms. Mechanism research incorporating multiple efficacious psychotherapies and multiple putative mediators in the same RCT may be especially helpful for identifying mediators that are common across psychotherapies. The identification of mediators also has the potential to distill treatments down to their essential components, which might result in reductions to the time and cost to train therapists as well as to disseminate psychotherapy. Mechanism research may thus increase the efficiency of psychotherapy. Perhaps the most important

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benefit of increased efficiency is that more economical therapies increase the availability of psychotherapy, especially to underserved populations (Kazdin & Blase, 2011).

Another suggestion for future research is made specifically for investigating cognitive change. Cognitive change has a special status among potential mediators due to the increasing evidence which suggests that cognitive change mediates the effects of cognitive therapy on depressive symptom reduction. Future efforts to further validate this mediator may focus on meeting the various criteria described earlier (e.g., dose-response and consistency). Meeting these criteria may strengthen confidence in the conclusion that cognitive change truly is involved in the causal pathway from cognitive therapy to symptom reduction. Perhaps most important would be research that examines the “convergence of evidence” criterion by investigating whether cognitive therapy works through cognitive change across various disorders. If this were the case, the validity of applying cognitive change techniques across disorders would be strengthened. The studies summarized in Table 1 (Hofmann, 2004; Smits et al., 2004; Wilson et al., 2002) each investigated potential mediators of the effects of cognitive therapy on different disorders (panic disorder, social phobia, and bulimia nervosa); however, these studies included methodological flaws. Thus, new research taking multiple assessments of potential mediators and outcomes is needed across the realm of psychopathologies for which cognitive therapy has proven effective.

Any mediator may be evaluated based on the criteria proposed in this paper, and the degree to which a mediator meets those criteria could serve as a useful basis for making judgments about whether the mediator should be considered a causal mechanism. In summary, it is necessary for changes in mediators to precede changes in outcomes (Criterion 1). Establishing that a mediator meets the statistical criteria proposed by Baron and Kenny (1986) rules out the possibility of Type II error (Criterion 2). Finding evidence for a mediator across studies reduces the chances that the any individual study committed a Type I error (Criterion 3). If the mediator was discovered in the context of an RCT, it is less likely that pre-existing conditions could have accounted for mediation effects (Criterion 4). Evidence for a dose-response relationship between a mediator and outcome reduces the likelihood that the mediator works only in a narrow set of circumstances (Criterion 5). Showing that a mediator is specific to certain type of therapy reduces the likelihood that the mediator is due to general effects of psychotherapy (Criterion 6). Finding that mediation converges across methodologies reduces the likelihood that the mediator is due to methodological artifact (Criterion 7). Finally, examining potential mediators that have been well established within other empirical contexts reduces the likelihood that the mediator is not a valid scientific construct (Criterion 8). These criteria for mediation have the potential to clarify the scientific status of potential mediators

by serving as a framework in which research on mediation in psychotherapy may be systematically organized.

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