

# Conceptual and Design Essentials for Evaluating Mechanisms of Change

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**Background:** Considerable progress has been made toward the development of evidence-based treatments for a wide range of psychological disorders; however, little is known about the mechanisms through which these treatments actually lead to clinical change. Although the use of traditional randomized controlled treatment designs and tests of statistical mediation have significantly advanced understanding of psychological treatments, they are insufficient to test mechanisms of change.

**Method:** This article outlines the conceptual and methodological requirements for evaluating mechanisms of change, highlights the importance of such a focus, and offers specific recommendations for research aimed at elucidating change mechanisms.

**Results and Conclusions:** Conceptualizing and conducting studies that test mechanisms of change requires substantial modifications to traditional research designs, but doing so will significantly enhance scientific understanding as well as the efficiency and effectiveness of clinical interventions.

**Key Words:** Mechanism of Change, Mediator, Treatment, Psychotherapy.

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THE ULTIMATE GOALS of clinical science are (1) to develop methods that decrease pathology and return individuals to healthy, adaptive functioning, (2) to elucidate the processes through which these methods have their effect, and (3) to identify the conditions that influence the efficacy of these methods. These goals are shared by clinical scientists working across various disciplines (e.g., psychology, psychiatry, oncology, dermatology) using a wide range of measurement methods (e.g., behavioral observations, interviews, neuroimaging techniques), and they have been highlighted as major research priorities by federal funding agencies (Zerhouni, 2003, 2005).

Recent advances toward the first of these 3 goals have been impressive. Guidelines for evaluating the evidence supporting the efficacy of specific treatments have been developed (Chambless and Hollon, 1998; NICE, 2005) and many efficacious treatments identified (Kazdin and Weisz, 2003; Nathan and Gorman, 2002). Unfortunately, much less attention has been focused on identifying the mechanisms through which treatments have their effects or the factors that moderate treatment outcomes (Kazdin, 2000a; Nock, 2003). The primary purpose of this article is to highlight the importance of studying mechanisms of change and to delineate the concep-

tual and design essentials for evaluating the processes through which clinical change occurs.

In this paper, I first expand on prior work defining mechanisms of change and clarify how mechanisms differ from closely related concepts such as statistical mediators of change (Kazdin and Nock, 2003). Next, I outline the 8 methodological criteria for demonstrating the operation of mechanisms of change and highlight the significance of this work for both scientific and clinical endeavors. Finally, I suggest key questions to be considered by researchers designing studies of mechanisms of change in psychological treatments, and end by making recommendations for future research in this important area.

## TERMS AND DEFINITIONS: CORRELATES, CAUSES, MEDIATORS, AND MECHANISMS

Given inconsistencies in the terms and definitions that have been used in prior work in this area, it is important to begin by defining key concepts and describing how they relate to each other and fit into the broader scientific context. At the most basic level, scientists are interested in understanding how constructs are related, or in demonstrating *covariation* or *correlation* among variables ( $A \leftrightarrow B$ ). Typically, there is interest in obtaining a deeper understanding of the nature of such relations, such as the direction or dependence of observed relations. If it can be shown that A is related and temporally prior to B, then A is considered a *predictor* of, or *risk factor* for, B (Kraemer et al., 1997, 2001).

Knowledge of risk factors is important for understanding how psychopathology develops and for guiding the focus of prevention and intervention programs. Notably, however, not all risk factors are malleable (e.g., sex, ethnicity), and when

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they are, changing a risk factor does not always lead to change in the outcome of interest. For instance, early onset androgenetic alopecia (i.e., hair loss) has been shown to be a risk factor for coronary heart disease (Matilainen et al., 2001); however, dispensing toupees and hair replacement kits do not decrease the risk of heart disease. This distinction is well captured in the popular acknowledgment that ‘correlation does not equal causation.’

Although the necessary conditions for demonstrating causation have been debated for centuries (Aristotle, 384–322 BCE; Hume, 1739; Popper, 1972), there is general scientific consensus that true experiments are necessary to demonstrate a causal relation between variables. For instance, using a randomized controlled trial (RCT) one can demonstrate that careful manipulation of an independent variable (i.e., assignment to treatment condition) leads to or *causes* change in a dependent variable (Nock et al., in press). The demonstration of causal relations has obvious implications for intervention research; however, showing that a treatment causes change does not in itself illuminate how this change occurred.

There has been increasing interest by treatment researchers in moving beyond knowing *that* a causal relationship exists to understanding *how* change in one variable causes change in another. A *mechanism of change* refers to the process or series of events through which one variable leads to or causes change in another variable. The difference between cause and mechanism is conveyed in the familiar distinction between *internal* and *construct* validity (Cook and Campbell, 1979; Kazdin, 2003; Shadish et al., 2001). The former refers to the extent to which one has identified *what* caused change, while the latter to the extent to which one has identified *how* the change occurred.

Over the past 2 decades, researchers have developed useful methods for testing whether a proposed mechanism can statistically explain the relationship between an independent and dependent variable. To show such a relation, one must demonstrate that an independent variable (A) is associated with a dependent variable (B); that A is associated with the proposed mechanism (M); that M is associated with B; and when A and M are both covaried with B, M continues to be associated with B but the relationship between A and B is diminished. This pattern of relationships provides evidence that A is associated with B through its relation with M (Baron and Kenny, 1986; Holmbeck, 1997; MacKinnon et al., 2002).

This approach has been incredibly valuable in elucidating how different variables are related statistically. However, while statistical mediation is necessary to support the operation of a mechanism of change, it does not provide sufficient evidence for such a relation. Indeed, just as correlation does not equal causation, mediation does not equal mechanism. This paper describes the more rigorous methodological requirements for demonstrating the processes through which an independent variable causes change in a dependent variable. As such, I use the term *statistical mediator* to denote when requirements for statistical mediation have been satisfied, and reserve the term *mechanism of change* for instances

in which additional criteria have been met. These criteria are outlined below.

## REQUIREMENTS FOR DEMONSTRATING MECHANISMS OF CHANGE

The requirements for demonstrating the operation of a mechanism of change integrate the criteria for showing statistical mediation with those for inferring causal relations. The criteria for demonstrating causality have been outlined previously (Hill, 1965; Kenny, 1979; Schlesselman, 1982), yet discussion of these conditions has been absent from psychological science in general, and intervention research in particular. I outline these criteria here and highlight how, when used in concert with the standards for demonstrating mediation, they provide evidence for the operation of mechanisms of change.

### *Strong Association*

Showing a correlation among variables is the most fundamental requirement for demonstrating the operation of a mechanism of change. Consistent with the criteria for statistical mediation, to show evidence of a mechanism one must demonstrate that the independent variable (A) is associated with both the dependent variable (B) and the proposed mechanism (M), and that the proposed mechanism is associated with the dependent variable. Invoking the conditions for statistical mediation, it is also required that M continues to be associated with B, but that the relation between A and B is weakened when A and M are used to statistically predict B in the same equation. Unfortunately, recent evidence suggests that although many treatment studies include assessment of potential mechanisms of change, most fail to perform even these basic tests of statistical mediation (Weersing and Weisz, 2002).

### *Specificity*

The case for demonstrating a mechanism of change is strengthened if it can be shown that change in A is uniquely related to change in M, and also that change in M is uniquely related to change in B. As an example, it has long been known that administration of benzodiazepines causes a decrease in the experience of anxiety, as demonstrated in randomized controlled trials (Roy-Byrne and Cowley, 1998). However, only recently has the specific process through which these medications have their anxiolytic effect been elucidated. These compounds bind to  $\alpha 2$  GABA<sub>A</sub> (gamma-aminobutyric acid type A) receptors in the brain, thus amplifying the action of GABA in specific regions of the brain (Low et al., 2000). Specificity is demonstrated via the finding that benzodiazepines do *not* bind to  $\alpha 3$  GABA<sub>A</sub> receptors (i.e., specificity between A and M), and that they decrease anxiety but do not necessarily decrease other psychiatric symptoms (i.e., specificity between M and B).

### *Gradient*

Demonstrating a dose–response relationship between variables further strengthens the inference for causality. This goes beyond showing a simple association. For example, one may demonstrate that assignment to a given treatment condition is associated with change in the proposed mechanism, which is in turn associated with change in the outcome of interest. This is great, but does not provide evidence of a gradient. Showing that *more* of the treatment (i.e., a greater dose) is associated with more of the proposed mechanism, which in turn leads to greater increases in the outcome, would demonstrate a gradient. Studies of pharmacologic treatments often test for such gradients by examining different doses of the same agent (Cernea et al., 2005; Ferrante et al., 2005). Evaluation of stepped care models of psychological treatments has been advocated (Bower and Gilbody, 2005; Breslin et al., 1998; Sobell et al., 2002; Wilson et al., 2000), and those that examine different doses of the same active ingredients would provide valuable tests of gradients in this context.

### *Temporal Relation*

Demonstration of a mechanism of change requires evidence that change in the independent variable precedes change in the proposed mechanism, and that change in the mechanism precedes change in the outcome of interest. This requirement, although fairly straightforward, is among the most difficult to satisfy. The common procedure of measuring proposed mechanisms and outcomes of interest at pretreatment and post-treatment is insufficient. Showing a temporal relationship requires simultaneous and repeated assessment of the proposed mechanism and outcome over the course of treatment. Requiring repeated assessments greatly increases the difficulty of completing studies that can adequately satisfy this criterion; however, several research groups have done so with impressive results (Barber et al., 2000; Wilson et al., 2002).

It is worth noting that the use of some common statistical terms (e.g., “predictor” variables, “causal” modeling) can lead readers to assume that evidence for temporality or causality has been provided when in fact it has not. We have referred to such inferential errors as the Kazdin-Nock Illusion<sup>1</sup> and advocate the use of language that more precisely specifies the observed relationships (e.g., statistical predictor, structural equation modeling) to limit the likelihood of such errors.

### *Consistency*

As in all areas of science, replication of the observed result across studies, samples, and conditions strengthens the valid-

ity of the inferences drawn from such findings. Notably, inconsistency across studies does not always weaken confidence in the findings, but may instead signal the operation of a moderating variable (i.e., one that influences the strength or direction of the relationship between variables). For instance, a particular intervention may be found to be effective in decreasing alcohol use when tested among a sample of adolescents, but not among a sample of adults. Such a pattern of findings suggests age may moderate the effectiveness of the intervention, thus providing a more nuanced understanding of the observed treatment effect.

### *Experiment*

Use of an experimental design with random assignment to conditions and careful manipulation of the proposed causal agent is essential for demonstrating a causal relation. In testing mechanisms of change, the goal is to show that manipulation changes the proposed mechanism, which then changes the proposed outcome. This requires combining an experimental design and frequent assessment. Frequent assessment of key constructs can be used to establish the temporal relationship among variables, but the failure to use experimental manipulation can substantially weaken inferences about causal mechanisms of change. For instance, prior work on “sudden gains” within the context of psychological treatments has suggested that cognitive changes are significantly associated with sudden gains in symptom improvement (strong association), that the former occur immediately prior to the latter (temporal relation), and that changes in cognitions but not therapeutic alliance predicted symptom change (specificity) (Tang and DeRubeis, 1999). Moreover, the sudden gain phenomenon has been reported across several studies and types of treatment (consistency) (Tang et al., 2002, 2005). Unfortunately, the absence of an experimental design limits inferences about causality, as one cannot rule out alternative causal explanations for the observed effects. Many other excellent attempts to demonstrate the operation of mechanisms of change have been limited by the lack of an experimental manipulation of the proposed causal agent (Doss et al., 2005; Karno and Longabaugh, 2005; Moyers et al., 2005; Wilson et al., 2002).

### *Plausibility and Coherence*

The final criterion that should be satisfied to demonstrate the operation of a mechanism of change is that findings articulate a credible explanation of the actual process through which one construct influences another and that this explanation is consistent with the definition of a mechanism of change as well as with broader scientific knowledge. Of course, new scientific discoveries often challenge and disprove prior beliefs (e.g., it turns out the earth is neither not flat nor at the center of the universe) (see Copernicus, 1514). However, only those discoveries that provide a plausible explanation and coherence with existing or developing knowledge are accepted by

<sup>1</sup>Briefly, the Kazdin-Nock Illusion (KNI) is a variant of the well-known Figure-Ground Illusion (the one that looks like a vase/profile of 2 heads). The KNI is similar, but different. Here the viewer sees unidirectional arrows pointing from ‘predictor’ variables to ‘outcome’ variables and *figures* these are *grounds* for inferring a timeline or causal relations. This is only an illusion.

the scientific community. Adherents of a treatment technique called Thought Field Therapy propose that tapping a sequence of meridian points on one's own body while humming a tune results in the immediate cure of a wide range of ailments. The proposed mechanism of action is a change in one's heart rate variability (Callahan, 2001a,b). This approach has been viewed with some skepticism by clinical scientists given the implausibility of these findings and their complete lack of coherence with broader scientific knowledge (Kline, 2001; McNally, 2001).

### *Criteria in Concert*

The satisfaction of each of these criteria further increases the strength of the evidence for the operation of a causal mechanism of change. No one criterion is sufficient, but it is suggested that strong association, temporal relation, experiment, and specificity are the minimum necessary to qualify a construct as a mechanism of change. The satisfaction of each additional criterion both within and across studies furthers the case.

### **THIS SEEMS HARD: WHY STUDY MECHANISMS OF CHANGE?**

Designing and conducting traditional RCTs using only pre-treatment and posttreatment assessment of key constructs represents an enormous undertaking in any area of clinical science. Satisfaction of the criteria outlined above requires the next generation of RCTs to also include more conditions (thus boosting required sample sizes) and more frequent assessment of a wider range of constructs. This is admittedly an ambitious agenda and one that will require enormous time and resources, begging the question: why should we study mechanisms of behavior change? If we determine that a treatment is effective, does it really benefit us to know how it works? In short, yes. The benefits of studying and elucidating the mechanisms of behavior change in psychological treatments will far exceed potential costs for at least 3 reasons.

First, understanding mechanisms can help to bring order and parsimony to the current status of treatments. Many different psychological interventions exist; indeed, a recent examination of the literature on child and adolescent treatment revealed over 550 different treatments currently in use (Kazdin, 2000b)! However, many theoretically distinct treatment packages actually share common components or procedures (e.g., Ablon and Jones, 1999), and some have even argued that elements that are common among different approaches account for more change than do those that are unique to different approaches (Wampold, 2001). Identifying the mechanisms through which people change in treatment will provide the data necessary to resolve these debates and illuminate the common and specific factors responsible for clinical change.

A second, related benefit is that understanding mechanisms will increase the efficiency and effectiveness of current

treatments. Most psychological interventions have many, many components and if the active ingredients and mechanisms of change are identified, researchers and clinicians can include more of what is effective and discard the ineffective elements. Examples of such a progression are readily available in more mature areas of clinical science. For instance, malaria plagued Europe and the other parts of the world for centuries until the discovery in the 17th century that an effective treatment for this illness is consuming small quantities of bark from the South American cinchona tree. Scientists eventually isolated the mechanism through which this tree bark cures malaria in 1820 (specifically, the quinine found in the bark is the active ingredient and presumably kills the malaria parasite by hindering its ability to break down hemoglobin), which has led to the production of efficient and effective treatments for malaria that do not require eating bark (Werner, 2003). Identifying the precise mechanisms through which psychological treatments change behavior will similarly lead to more proficient and powerful clinical interventions.

Third, understanding mechanisms of change in psychological treatments will increase understanding of behavior change more generally. Psychological treatments are essentially highly structured interpersonal interactions that (ideally) include specified interventions and observation of resulting behavior change. Identifying the mechanisms through which behavior of the client is changed by the clinician (and the clinician by the client) can provide important information about how behavior change likely occurs in "natural" settings (i.e., outside the clinician's office). Conversely, understanding how people change in natural settings can greatly inform psychological treatments. For instance, most people experiencing behavioral problems (e.g., alcohol dependence, depressive disorders) do not seek treatment (Demyttenaere et al., 2004; Kessler et al., 2005) and many who do fail to respond to treatment (e.g., Keller, 2005). Yet, many individuals recover from these disorders. What are the mechanisms through which this change occurs? Clinicians often refer to recovery outside the context of formal treatment somewhat dismissively as "spontaneous remission"; however, understanding how and why such change occurs could lead to enormous advances in more formal treatment settings. Whether inside or outside treatment, illuminating how and why people change will undoubtedly lead to enormous scientific and clinical advances.

### **EVALUATING MECHANISMS OF CHANGE IN PSYCHOLOGICAL TREATMENTS**

Research from other areas of clinical science provides helpful models for studying mechanisms of change in psychological treatments. Although many examples from these other areas (e.g., benzodiazepine use, malaria) illustrate the operation of mechanisms of change at the level of basic biology or chemistry, this of course is not essential to evaluate mechanisms of behavior change. Although there are certainly biological and chemical mechanisms involved in behavior change (Guze, 1989), it would be a great oversimplification to take

such a biological reductionist perspective. Stated most succinctly, “psychology is not applied biology” (Anderson, 1972, p. 393). Indeed, the task for research on mechanisms of behavior change is to consider multiple levels of analysis in explanatory models of change (Kendler, 2005). Moreover, the models used to study change mechanisms need not be outstandingly complex or fully determined to inform scientific understanding and clinical practice. Many examples exist in which disease processes are known to be highly complex, yet relatively simple and effective treatments have been developed due to an understanding of key mechanisms involved (e.g., pernicious anemia is effectively treated by B<sub>12</sub> injection, phenylketonuria can be managed reasonably well by providing a diet low in phenylalanine) (see Rees, 2002 for review).

So on what should psychological scientists focus? In considering change within the context of psychological interventions (which is admittedly narrow) there are at least 3 classes of factors involved in the change process: (1) clinician factors, (2) client factors, and (3) actual mechanisms of change. In designing and conducting studies of change mechanisms, it is important to have clarity regarding which processes are being targeted and assessed (Doss, 2004; Kazdin, 2000b). Clinician factors refer to what the clinician does in treatment, including clinician behaviors, characteristics, and directives. Teaching skills, making supportive statements, and adhering to a standardized treatment are all clinician factors (Beutler et al., 2004; Schoenwald et al., 2000). Client factors refer to what the client does in treatment, including behaviors, characteristics, and verbalizations on their part. Client attendance and adherence to treatment, engagement in treatment procedures, expectancies and motivation for treatment, and statements to the clinician are all client factors (Clarkin and Levy, 2004; Nock and Ferriter, 2005; Nock and Kazdin, 2005). The actual mechanisms of change are the processes that emerge from or occur as a result of the clinician and client factors, and their interaction, that explain how those factors lead to change in the outcomes of interest. Improvement in impulse control, enhancements in problem-solving skills, and increased engagement in pleasurable activities are all potential mechanisms of change.

To be sure, clinician and client factors are important facets of the change process that can and often do influence treatment outcomes. Moreover, it is of great importance to isolate the active ingredients in current treatments that account for change (e.g., through dismantling studies/component analyses), because knowing which of the many components often included in treatments are active will aid significantly in the search for mechanisms of change. However, knowing which specific clinician or client factors (e.g., specific skills taught by the clinician, number or type of change statements made by the client during sessions) are most strongly predictive of change does not in itself provide an explanation of how change occurs, because they (i.e., the clinician and client factors) are the treatment. Of course, one could demonstrate that

clinician and client factors statistically mediate the relation between treatment and outcome; however, such analyses do not provide an explanation of the mechanism or process through which change occurred, only that variance in the outcome has been accounted for. The search for actual mechanisms must examine changes in the client that persist or generalize outside of the treatment setting to be able to explain the relation between treatment and lasting changes in clinical outcomes.

The important distinction here is between isolating the active ingredients of treatment (i.e., specific components that cause the observed change) and identifying the mechanisms or processes through which these ingredients actually cause change. Several examples may help clarify this important distinction. As mentioned above, consuming bark from the cinchona tree can effectively treat malaria. Quinine has been isolated as the active ingredient in the bark. More specifically, the mechanism through which quinine affects malaria is by killing malarial parasites by hindering their ability to decompose hemoglobin, thus leading to starvation or the accumulation of toxic levels of hemoglobin. So while quinine is the active ingredient, it does not itself explain the process through which it works. There are many other examples in medicine in which (1) organic compounds are found to affect behavior, (2) the active ingredient is then isolated, and (3) subsequent understanding of its mechanism of action leads to the development of additional therapeutic agents (Aguayo et al., 2006; Tagboto and Townson, 2001). Research on psychological interventions achieved step 1 via recent work on the development of evidence-based treatments; however, more attention is needed to steps 2 and 3.

Consider another example: drinking several gin and tonics can lead to alcohol intoxication. What is the actual mechanism through which these drinks lead to intoxication? The most important active ingredient is the alcohol contained in the gin (incidentally, it should be noted that while tonic water contains quinine, drinking lots of gin and tonics is not an effective treatment for malaria) (Meyer et al., 2004). Although alcohol is the active ingredient, we should not conclude that we have identified the actual mechanism or process through which the drink leads to intoxication, because this of course does not provide an explanation of how alcohol causes change. Instead, our explanation of the mechanism of action of alcohol would include a discussion of the cascade of chemical and biological changes that occur between the introduction of alcohol into the body and the clinical manifestation of intoxication. An analogous understanding of the mechanisms or processes through which specific, active ingredients in psychological treatments cause changes in human behavior is needed. Here too, there is undoubtedly a complex cascade of cognitive, affective, and behavioral changes that can explain how an intervention causes change in the clinical outcome of interest. The eventual understanding of such events will require the accumulation of converging evidence over many separate studies designed to test mechanisms of change.

## KEY QUESTIONS IN DESIGNING STUDIES OF MECHANISMS OF CHANGE

Designing studies that test proposed mechanisms of clinical change requires extensive planning and careful consideration of myriad factors. A discussion of the full range of methodological and design decisions is obviously beyond the scope of this paper (see Cook and Campbell, 1979; Kazdin, 2003; Shadish et al., 2001). However, I propose several key questions to consider in planning studies of mechanisms of change.

### *What Are the Putative Mechanisms of Change?*

The study of change mechanisms requires that researchers have a theory of how change occurs that includes clearly specified mechanisms. Such theories may include clinician and client factors, but ultimately must also account for variables that offer an explanation of how these factors lead to changes in key outcomes. Moreover, although initial studies, such as analyses of existing data, may be limited by the range of variables available to the researcher, future research in this area will be most fruitful if proposed mechanisms are specified in advance and tests of these constructs are incorporated into the study design.

### *When Are the Mechanisms Likely to Change?*

Researchers must give careful consideration to the timing at which change is most likely to begin and how quickly it is expected to occur. For instance, prior research has shown that change in many psychological treatments occurs early in the course of the intervention (Ilardi and Craighead, 1994; Wilson et al., 1999) and often quite suddenly (Tang and DeRubeis, 1999). Theories about when and how quickly change is likely to occur should guide the timing and frequency of assessment.

### *Where Are the Mechanisms Likely to Change?*

Similarly, careful consideration must be given to where change is likely to occur. Is the proposed mechanism expected to change within the treatment setting (e.g., increased self-efficacy) or outside (e.g., increased engagement in pleasurable activities), or both? Also, is it likely to occur within the person (e.g., changes in cognitive processing) or outside (e.g., increased interaction with others), or both?

### *What Is the Optimal Measurement Method?*

Efforts should be made to include reliable and valid measures of mechanisms and other study constructs, and special care should be taken to include measures that are objective and sensitive to change. Toward this end, researchers should ensure that measurement occurs at the appropriate time and place to best capture changes in key processes. For instance, if mechanisms are expected to change outside of treatment, is

it possible to conduct assessment outside of treatment (e.g., using electronic diaries, video surveillance)?

### *How Can the Putative Mechanisms Be Targeted Directly?*

Once change mechanisms are identified, can they be targeted directly, perhaps by modifying clinician and client factors? For instance, if there is evidence that changes in one's ability to tolerate distress may be a key mechanism of change in treatment, can the clinician and client work to make this a larger focus during treatment sessions?

## RECOMMENDATIONS FOR RESEARCH

Recommendations for future work in this area follow directly from the requirements and questions outlined above. Investigations of mechanisms are best grounded in theory and guided by broader scientific knowledge. In addition, inferences about causality require use of experimental designs, and satisfaction of most additional criteria requires careful consideration of the assessment procedures used in such studies. More specifically, examination of change mechanisms will be best accomplished with adherence to the following recommendation: Assess Early, Inclusively, Often, Ubiquitously, and sometimes with Your colleagues working in other research areas.<sup>2</sup>

### *Assess Early*

Given change often occurs before people even enter the treatment setting (Howard et al., 1986), and upon entering treatment most change occurs in the first several weeks (Ilardi and Craighead, 1994), there is a need to begin assessment early enough to capture this change and the factors likely to be influencing it. This can involve identifying people who have not yet decided to seek treatment (e.g., those at high-risk for a given outcome) and assessing them over time to examine the processes influencing pretreatment change.

### *Inclusively*

Studies should incorporate measures of multiple potential mechanisms of change. This is especially important given the time and resources required to conduct studies in this area, as well as the significance of demonstrating specificity of change mechanisms.

### *Often*

Demonstrating the temporal relation of change in proposed mechanisms and outcomes requires frequent assessment of all

<sup>2</sup>This is referred to as the "A-E-I-O-U and sometimes Y" approach to study design, which draws inspiration from the classic pedagogical tool (as well as the awesome 1984 hit single by *Ebn Ozni*).

of these constructs. As mentioned above, traditional pre- and posttreatment designs are insufficient to examine mechanisms of change.

### *Ubiquitously*

There is also a great need to progress beyond traditional treatment research designs that assess individuals in the treatment setting. If we believe that change is occurring outside of treatment, then we should assess change mechanisms outside of treatment. Doing so will provide more valid information about how, when, and why people change.

### *Your Colleagues*

Excellent methods of measuring potential mechanisms of change exist that are not used by treatment researchers. Over the past several decades, cognitive, social, and developmental psychologists, as well as neuroscientists and others, have developed wonderful methods for measuring many of the constructs likely to be leading candidates as potential mechanisms of behavior change. As an example, social psychologists have developed and tested reaction-time tests that measure implicit associations about a wide range of concepts (Fazio and Olson, 2003; Greenwald et al., 2003), and these have been shown to be related to psychopathology (e.g., Nock and Banaji, 2007, in press; Palfai and Wagner, 2004), and to change over the course of successful psychological treatment (Teachman and Woody, 2003; Wiers et al., 2006). This represents just one of an enormous range of reliable, valid, objective, and sensitive measures that can be used to begin to understand how change occurs.

## CONCLUSIONS

Impressive advances have been made in the development and evaluation of evidence-based treatments, and work aimed at broader dissemination of these treatments is currently underway. One central issue that has been neglected, however, is that we lack a firm understanding of *how* these treatments work. This article defined mechanisms of change and proposed a methodological framework for evaluating change mechanisms that synthesizes earlier work on statistical mediation and causal inference. Prior work using these separate approaches has led to important discoveries and amazing advances; however, neither is sufficient to adequately study mechanisms of change.

Although the work ahead is daunting, the fruits of this line of research will greatly improve scientific understanding as well as the efficiency and effectiveness of clinical interventions. Several research groups have laid an impressive foundation by demonstrating the feasibility of conducting the repeated assessments of potential mechanisms and outcomes over the course of treatment studies (Eddy and Chamberlain, 2000; Kolko et al., 2000; Wilson et al., 2002), a difficult but necessary requirement for studying

mechanisms of change. Additional research is sorely needed (Kazdin, 2006), but investing the time and resources needed to better understand the mechanisms through which people change is an essential step toward achieving the ultimate goals of clinical science.

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