

Delineating mechanisms of change in child and adolescent therapy: methodological issues and research recommendations

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Background: Mechanisms of therapeutic change are rarely studied in child and adolescent therapy. Our central thesis is that the study of mechanisms of treatment is an excellent investment for improving clinical practice and patient care. Indeed, extending treatment trials to clinical settings, without complementary research that studies why and how treatment works, could have great limitations. **Method:** In this article, we discuss the importance of studying mechanisms, the logical and methodological requirements, and why almost no studies to date provide evidence for why or how treatment works. Standard statistical practices (tests of mediation) and designs (randomized controlled clinical trials) contribute greatly to outcome research but have little to say about mechanisms given the way they are commonly used. **Conclusions:** The article ends with recommendations to guide research on mechanisms of therapeutic change. **Keywords:** Child and adolescent therapy, mechanisms of therapeutic change, child treatment.

Child and adolescent therapy research has advanced enormously in the past two decades. Hundreds of randomized controlled trials and scores of reviews have attested to the effects of treatment (Kazdin, 2000a; Weisz, Weiss, Han, Granger, & Morton, 1995). Children who receive therapy are much better off than those who do not. For several clinical problems, including anxiety, depression, oppositional and conduct disorder, and attention-deficit/hyperactivity disorder, evidence-based treatments have been identified (Christophersen & Mortweet, 2001; Kazdin & Weisz, in press; Lonigan & Elbert, 1998). These treatments have been delineated based on multiple criteria, including tests in randomized controlled trials with clinic samples and replication of the effects of well-defined and manualized treatments. The advances in child and adolescent therapy are likely to continue in light of the continued stream of treatment outcome studies.

Notwithstanding major progress, fundamental issues about treatment and its effects are not resolved. Whether the effects of treatment demonstrated in research can be obtained in clinical practice (efficacy versus effectiveness studies) has not been well studied. In addition, the impact of treatment on children and families in either research or clinical settings is not clear. Statistically significant changes on treatment outcome measures and changes designated on a priori grounds as 'clinically significant' do not convey how a treated child is actually functioning in everyday life. Finally, there are over 550 treatments in use for children and adolescents (Kazdin, 2000b). The vast majority of these have not been subjected to controlled or, indeed, to uncontrolled investigation. Consequently, most treatments in use clinically have no clear empirical base.

As a research priority, strong emphasis has been placed on the importance of testing whether treatment effects can be extended to clinic service settings (e.g., National Advisory Mental Health Council [NAMHC], 1999; NAMHC Workgroup on Child and Adolescent Mental Health Intervention Development and Deployment [NAMHC-W], 2001). This is a logical and needed emphasis to ensure that the benefits of research can be applied to clinical practice. There is long-standing recognition in intervention research involving both treatment and prevention that demonstration projects show what *can happen* under often ideal or quite special conditions but this may be different from what *does happen* when the intervention is extended to situations and settings of everyday life. Extension of findings to clinical practices is critically important. The issue of translation from basic research to practice is not unique to psychotherapy research and indeed is also a current issue and concern in biology and medicine (Rees, 2002).

Progress in child therapy and in the clinical applications of effective treatment is needed on multiple fronts (see Kazdin, 2000b; Nock, in press). This article focuses on one area in need of particular attention, the study of mechanisms of therapeutic change, i.e., why therapy works. This line of work is likely to be left by the wayside with the emphasis and accelerated attention to research in clinical services and on the dissemination and transportability of current treatments. We reiterate that the comments that follow do not detract from the important work on treatment services. The study of mechanisms of treatment will be an essential partner. We underscore the need for the study of mechanisms for three reasons: (1) the topic is neglected in contemporary

research, (2) the designs used in current research are not likely to be able to evaluate mechanisms, and (3) there are occasional claims that in fact mechanisms are currently being studied. The combination of empirical neglect of the topic and views that mechanisms are being studied, we argue, is hazardous to progress.

The central thesis of this article is that study of mechanisms of treatment is probably the best short-term and long-term investment for improving clinical practice and patient care. There are many questions and foci of therapy research (e.g., why, how, for whom treatment works, what components and combinations contribute to outcome). We single out the importance of understanding why and how treatment works. Understanding why treatment works can serve as a basis for maximizing treatment effects and ensuring that critical features are generalized to clinical practice. The role of understanding (theory) and application (real-world tests) are complementary and essential. Real-world tests are absolutely vital and great progress can be made with only such analyses. For example, the first airplanes, which only flew seconds and minutes, were cleverly devised with little understanding of the aerodynamics of flight such as fluid flow, skin-friction drag, and aspect wing ratios and the physics and mathematics that govern the design of current airplanes. Many prior attempts of flight, often replayed as comical and tragic film clips of humans sporting feigned wings and jumping off cliffs and rocks, show a worst case of what empirical tests can yield where there is minimal to no understanding. Needless to say, application (i.e., flight in everyday life) has been enhanced enormously by advances in understanding the underpinnings of flight well outside of real-world application.

In the context of treatment, there have been few efforts to understand how and why therapy works. As critical perhaps, research designs (randomized controlled trials, pre-test/post-test designs), at least as currently executed, cannot adequately evaluate mechanisms of action, a point we shall elaborate. This article outlines the study of mechanisms, the importance of such a focus, and why research, as usually designed, may not be able to address mechanisms very well. We also delineate steps for demonstrating mechanisms of change in treatment research.

Study of mechanisms

Mechanisms defined

By *mechanisms*, we refer to those processes or events that lead to and cause therapeutic change. There are many different meanings of cause in science that vary as a function of the temporal relation (e.g., proximal, distal) and mode of operation (e.g., direct, indirect) of an event or characteristic to the outcome

(Haynes, 1992). As a rule, several conditions usually need to be satisfied to infer cause. These conditions have been outlined elsewhere (see Hill, 1965; Kenny, 1979; Schlesselman, 1982), and are described later in this article as they relate to psychotherapy research.

In psychotherapy research, and psychological research more generally, *mediator* is often used as the term intended to signify a cause or mechanism of change and distinguished from *moderator*, as evident in the now classic paper by Baron and Kenny (1986). Mediator refers to the process(es) through which change occurs; moderator refers to those characteristics that influence the extent to which or indeed whether change occurs. Mediator and moderator are related. For example, if one finds sex differences in response to an intervention (e.g., treatment, harsh child-rearing practices), one can say that child sex serves as a moderator for the intervention. The finding raises the prospect that different mediators may be at work. That is, change occurs for different reasons or the impact of different causes varies as a function of sex of the individual. There are many such findings in the social and biological sciences in which moderators clearly suggest the likelihood that different mediators are at work. For example, early aggressive behavior (elementary school) predicts later delinquency in adolescence for boys but not for girls (Tremblay et al., 1992). The finding raises the prospect that different mechanisms might be involved in the processes leading to delinquency for males and females. In a quite different context, breast cancer is moderated by sex. Although breast cancer is evident in both males and females, the vastly different rates suggest that different mediators may be involved (Ravandi-Kashani & Hayes, 1998). In short, moderators have implications for mediators, a topic of keen interest but beyond our present scope.

In the context of this discussion, we refer to mechanisms of change to reflect the processes through which therapeutic change occurs. This requires showing a causal relation between an intervention and outcome of interest. But a causal relation alone is not sufficient. For example, a randomized controlled clinical trial of treatment (e.g., comparing treatment vs. no treatment) can establish a causal relation between an intervention and therapeutic change. Demonstrating a causal relation does not necessarily provide the construct to explain *why* the relation was obtained. In the case of our hypothetical treatment trial, the treatment may have caused the change but was this due to specific or conceptually hypothesized components of treatment (e.g., eye movements, cognitive restructuring, habituation) or some other construct(s) (e.g., attention/placebo influences, stress reduction, mobilization of hope)?

Although our focus is on child therapy, these concerns can be more dramatically conveyed by

illustrating a treatment from the adult therapy literature. There are very few psychotherapies as well established for depression as cognitive behavior therapy (CBT; American Psychiatric Association, 2000). By all counts, this treatment is evidence-based and then some in light of the range of trials. But why does CBT work, i.e., through what mechanisms? We have read, taught, and used the rationale so often that it is heresy to raise the question in an empirical arena. In fact, little can be stated as to why treatment works, i.e., that changes in cognitions are the mechanisms operative to decrease depression. It is not so much that evidence refutes the original conceptual view, although the scant evidence has not been kind (Burns & Spangler, 2001; Whisman, 1993, 1999). Rather, the suitable studies are rarely done. We have a firm basis for stating that CBT can change depression but no empirical basis for stating why. In the context of research design, Cook and Campbell (1979) refer to the notion of *construct validity* to elaborate the difference between the cause and the basis for the cause. Thus, we may know *that* the intervention (e.g., CBT) caused the change but not understand *why* (the basis for the cause or the mechanism) the intervention led to change.

The distinction between the cause and the basis of the cause can be readily conveyed with an example from the extensive literature on cigarette smoking and lung cancer. Cross-sectional and longitudinal studies and research with humans and animals have established a causal role between cigarette smoking and lung cancer. Establishing a causal relation does not automatically explain the mechanisms, i.e., the process(es) through which change has come about. The mechanism has only been demonstrated relatively recently. Specifically, a chemical (benzo[*a*]pyrene) found in cigarette smoke induces genetic mutation (at specific regions of the gene's DNA) that is identical to the damage evident in lung cancer cells (Denissenko, Pao, Tang, & Pfeifer, 1996). This finding is considered to convey precisely how cigarette smoking leads to cancer at the molecular level. Thus, beyond the demonstration of a causal relation, a fine-grained analysis of mechanisms or mediators is important as well. Knowing the mediator of a relation between variables obviously does not require only knowing the biological substrates. The mechanism or process through which two variables are related may involve all sorts of psychological constructs. In the context of treatment, research that focuses on mechanisms examines why change has occurred and hence explains the effects of treatment.

Why study mechanisms?

Evaluating mechanisms of therapeutic change is important for several reasons. First, we mentioned previously that there are 550+ psychotherapies in

use for children and adolescents (Kazdin, 2000b). This count is conservative because it omits various combinations of treatments and eclectic hybrids and only included interventions that could be documented in published sources within the English language. Even so, consider the number as an approximation. It is not likely that all of the different treatments produce change for different reasons. Understanding the mechanisms of change can bring order and parsimony to the current status of multiple interventions.

Second, an obvious goal of treatment is to optimize therapeutic change. By understanding the processes that account for therapeutic change one ought to be better able to foster and maximize improvements in the clients. Consider the development and use of treatment manuals to convey the point. The advance that treatment manuals represent is not at all in question here. However, without knowing how therapy works and what the necessary, sufficient, and facilitative ingredients are and within what 'dose' range, it is difficult to develop optimal treatment manuals. Much of what is contained in treatment manuals may be low doses of effective practices, ancillary but important facets that make delivery more palatable, superstitious behavior on the part of those of us who develop manuals, and factors that impede or merely fail to optimize therapeutic change. The difficulty is that without understanding how treatment works, which element in a manual falls into which of these categories is a matter of surmise.

Third, understanding how therapy works can help identify moderators of treatment, i.e., variables on which the effectiveness of a given treatment may depend. There are an unlimited number of moderators of child treatment that can plausibly influence outcome. Examples include characteristics of the child (e.g., age of onset, severity of dysfunction, comorbidity), the parents (e.g., psychopathology, stress), the therapist (e.g., experience, personality style), family (e.g., constellation, relationships, discord), and social context (e.g., school, neighborhood, culture). Understanding through what processes treatment operates can help sort through those facets that might be particularly influential in treatment outcome. For example, if changes in cognitive processes account for therapeutic change, this might draw attention to characteristics of these processes or their underpinnings at pretreatment. Pretreatment status of cognitive processes (abstract reasoning, problem-solving, attributions), stages of cognitive development, neurological or neuropsychological characteristics on which these cognitions might depend are just some of the moderators that might be especially worth studying, depending on the specific processes shown to mediate treatment effects. Other promising moderators that influence treatment might be proposed on theoretical grounds once the mechanisms of therapy are known.

Identifying moderators and the mechanisms through which they may operate can improve treatment outcomes by providing better triage of patients to treatments from which they are likely to benefit. For example, recent research on heart disease among women has found that estrogen replacement therapy varies in effectiveness according to (i.e., is moderated by) gene receptors that approximately 19% of women have. Women with the gene encoding receptor are much more responsive to treatment (Herrington et al., 2002). The processes involved appear to relate to increases in high-density lipoprotein (HDL or the 'good' cholesterol) for women who receive treatment and who have the gene receptor. The clinical utility of the finding stems in part from better triage of patients to effective treatment. Based on a genetic moderator, one can identify in advance who is likely to be responsive to treatment and who is likely to need additional intervention.

Fourth, understanding the mechanisms through which change takes place is important beyond the context of psychotherapy because of its broader relation to psychological science. There are many therapeutic processes in everyday life. By therapeutic, we refer to interventions or experiences that improve adjustment and adaptive functioning, ameliorate problems of mental and physical health, help people manage and cope with stress and crises, and more generally navigate the shoals of life. As examples, participating in religion, chatting with friends, exercising, undergoing hypnosis, and writing about sources of stress all have evidence on their behalf. The benefits and outcome effects include reducing psychopathology (e.g., suicidal ideation, depression) and physical ailments (e.g., pain, blood pressure) and improving recovery from surgery or illness (see Kazdin, 2000a). Therapy research is not merely about techniques but rather about the broader question, namely, how does one intervene to change social, emotional, and behavioral characteristics? Therapy is part of a broader process of functioning. Mechanisms that elaborate how therapy works might have generality for understanding human functioning more generally. The other side is of course equally true. Mechanisms that explain how other change methods work might well inform therapy. Basic psychological processes (e.g., learning, memory, perception, persuasion, social interaction) and their biological pathways (e.g., changes in neurotransmitters, responsiveness of receptors to transmitters) may be common to many types of interventions, including psychotherapy.

Research on mechanisms of change in child therapy

It is not difficult to make the case that understanding how therapy works is useful and important. A key question, however, is whether much attention has been devoted to the topic. A survey of

treatment research, now over a decade old, suggested that approximately 3% of treatment outcomes studies were focused on processes during therapy (Kazdin, Bass, Ayers, & Rodgers, 1990). This particular finding is not very informative in relation to understanding mechanisms of treatment. Process research was defined by measuring facets during therapy that might contribute to outcome. Yet, no formal evaluation was provided in this report about whether mechanisms were studied so this evaluation does not address the precise question of interest. On the other hand, if processes during treatment were rarely studied, then the number or proportion of studies that could look at mechanisms might well be limited.

More recently, Weersing and Weisz (2002) reviewed the child and adolescent literature on empirically supported treatments for evidence of mechanisms of therapeutic change. From their review, 67 studies on the treatment of depression, anxiety, and disruptive behavior problems were identified. Contrary to previous suggestions, they concluded that processes of change *are* studied in child psychotherapy. The authors reported that 63% of the studies evaluated a potential mediator of therapeutic change. However, only six of the studies (9%) in their review included a formal test of statistical mediation (i.e., used methods outlined by Baron & Kenny, 1986; Holmbeck, 1997). That is, the majority of studies had *assessed* at least one potential mediator but few had *actually tested* statistical mediation. Even so, these authors concluded that regarding the identification of mechanisms of change: 'Considerable evidence exists, but has not been fully exploited' (p. 22). As a result, Weersing and Weisz called for increased testing of statistical mediation in future studies as a means of evaluating mechanisms of change, and suggested that research shift to increasingly include formal mediation tests in more naturalistic, clinical settings.

The review by Weersing and Weisz (2002) suggests that the field has progressed considerably in studying mechanisms and understanding why therapeutic change occurs. However, we have a less favorable interpretation of the extant child psychotherapy literature and the evaluation of mechanisms of change. The inclusion of tests of statistical mediation in the six studies they describe certainly represents an improvement over past research. Yet multiple criteria must be met in order to demonstrate the role of a given mechanism of therapeutic change. Once these criteria are considered, none of the six studies meets the threshold necessary to demonstrate the operation of a causal mechanism of change. This is not an academic or esoteric point about research. As we noted previously, an understanding of mechanisms of change is critical for both scientific and clinical applications. Our interest in fostering research on mechanisms in both adult and child therapy and the absence of guide-

lines on how one might proceed serve as the impetus for this article.

Requirements for demonstrating mechanisms of change in psychotherapy research

Conditions for inferring a causal relation in scientific research have been outlined by previous authors (see Hill, 1965; Kenny, 1979; Schlesselman, 1982). Yet, acknowledgement or discussion of these criteria has been absent from the psychotherapy literature and from virtually all studies of therapeutic change. We outline these criteria and discuss how they are relevant to studies of change in psychotherapy research (for more extensive discussions of the criteria, please see for citations noted previously). A brief review of these criteria is important as a basis for considering how mechanisms are studied in current research and whether, or the extent to which, statistical approaches and research designs in use address these criteria. Each criterion is important in its own right. However, inferences are based on their convergence. It is useful to present them separately but we emphasize the fact that they are essential to consider together.

Strong association. The first and perhaps most basic criterion for demonstrating the operation of a mechanism of change is the demonstration of a *strong association* between the psychotherapeutic intervention (A) and the hypothesized mechanism of change (i.e., the mediator) (B). Then of course, there ought to be an association between the proposed mechanism (B) and therapeutic change (C). Indeed, if these three variables are not related, the case for the operation of a causal mechanism is greatly weakened, if not eliminated.

Specificity. The second criterion refers to the demonstration of the *specificity* of the associations between the intervention, proposed mechanism, and outcome. More directly, demonstration that many seemingly plausible constructs do not account for or contribute to change in the outcome, with the exception of the one proposed by the investigator, strengthens the argument that the proposed construct is responsible for change. The scientific method does not allow one to *prove* a connection. However, the connection or explanation gains plausibility by surviving multiple empirical tests while other explanations become less plausible.

As science has progressed, we have learned that simple causal paths and one-event-leading-to-one-outcome does not represent many phenomena we wish to understand (e.g., earthquakes, weather, adolescent substance use, reviewer manuscript rejection). In many disciplines and domains within a given discipline, multiple pathways (i.e., multi-causality, reciprocal causality, bi-directional changes) tend to be the rule rather than the exception.

However, given the current state of psychotherapy research, the demonstration of even a single causal relation would represent major progress.

Gradient. The third criterion is showing a gradient in which more of the allegedly critical process, mechanism, or causal agent is associated with greater change in the outcome of interest. A common analysis in medicine, epidemiology, and public health is showing a dose–response relation. For example, recent research has demonstrated the presence of a dose–response relation between passive cigarette smoking (i.e., exposure to ‘second-hand’ smoke) and coronary heart disease. The risk of heart disease increases significantly (and linearly) as one’s exposure to environmental cigarette smoke increases (He et al., 1999). A positive correlation underlies the notion of a dose–response relation but the relation is dissected beyond one summary statistic (e.g., Pearson product-moment r). Different levels of the mechanism or causal agent are shown to relate to the outcome of interest. Demonstrating a dose–response relation increases the plausibility of an agent being causally involved. Of course, it is possible that there is no dose–response relation (e.g., a qualitative or on–off gradient) or that the relation is not linear. Such relations do not mean a particular construct is not causally related, but may make inferences more difficult or require supplementary information. Again, although we delineate gradient as a separate criterion, it acts in concert with the other criteria to help infer cause or mechanism of action.

Experiment. The fourth criterion is the use of an *experiment*, through which one can demonstrate that manipulation of the proposed causal agent is associated with a change in the outcome of interest. Experimental techniques can be used to aid in the identification of precise mechanisms of change by holding specific variables constant across individuals in different experimental conditions while varying only the proposed mechanism of change. True experiments can rule out (make highly implausible) alternative explanations for the observed effects except for the condition(s) manipulated by the investigator. Indeed, the availability of experimental evidence strengthens the case that a change in the proposed mechanism is associated with a change in the outcome of interest.

In relation to therapy, mechanisms or mediators refer to the quite specific causes that may account for change. Thus, an experiment may establish that the treatment caused the change (e.g., treatment vs. no treatment) but may not be able to move to the more specific construct or mediator that accounts for the change (mediator). This has important implications for future research because the appropriately praised randomized control trial alone does not uncover mechanisms.

Temporal relation. The fifth criterion necessary to infer causality is demonstration that the occurrence of or change in the proposed mechanism preceded change in the outcome of interest. Obtaining evidence to satisfy this criterion is absolutely vital in order to infer the operation of a mechanism of change. This requirement is the Achilles' heel of treatment studies that might be considered to demonstrate or evaluate mechanisms of change, as we elaborate later.

Consistency. The argument for the demonstration of a mechanism of change is also strengthened with the satisfaction of the sixth criterion, *consistency*, which refers to the replication of an observed result across studies, samples, and conditions. Of course, the importance of replicating research findings in order to strengthen the validity of the inferences that are drawn is not unique to the demonstration of psychotherapeutic mechanisms of change. This criterion is particularly important, however, in the delineation of mechanisms of change given the great implications that such findings hold for subsequent clinical and research initiatives.

In passing, it is important to acknowledge that inconsistency across two or more demonstrations does not necessarily mean that the proposed mechanism is not involved. It is possible that the relation between a proposed mediator and outcome would be very consistent but is moderated by a variable we have not yet understood. For instance, the relation between two variables could be consistent for prepubertal children but not for adolescents. In this case, the results from several studies that include children, adolescents, or both may be inconsistent given that the moderator has not been identified. This more complex case need not detain us. When consistency across studies is obtained, this greatly facilitates drawing inferences about whether a particular mechanism may be involved.

Plausibility and coherence. To fulfill the seventh and final criterion for demonstrating the operation of a mechanism of change, the proposed mechanism must be characterized by *plausibility* and *coherence*. This refers to the articulation of a credible explanation of how such a mechanism operates and an integration of such a finding with the broader scientific knowledge base. In biology and medicine, pathophysiology is a concept that may be invoked to meet this criterion. That is, in light of the findings, is there likely to be a plausible, coherent, and reasonable process through which the disorder might be caused by the supposed mechanism? For example, cholesterol traversed the criteria for serving as a mechanism leading to heart disease. Among the criteria is the pathophysiology, in this case, of how lipids operate, how cholesterol combines with other influences to accumulate in the arteries, and so on.

Plausibility and coherence do not require a biological explanation and we use pathophysiology only because there are many well-worked-out exemplars. In relation to psychotherapy, plausibility and coherence convey the importance of theoretically based investigation of mechanisms of change. The findings ought to be plausible in light of a theory that has been proposed or in light of other findings about what is known about either the proposed mediator or the outcome.

Criteria in concert

The satisfaction of each criterion increases the strength of the argument for the operation of a mechanism of change. Perhaps not all criteria might be equally weighted or important. For example, the first four criteria: strong association, specificity, experiment, and temporality, represent the minimal criteria necessary to consider a construct a mechanism of change in psychotherapy. These criteria are consistent with the definition of mechanisms of change offered earlier, and used in most areas of scientific study. The remaining criteria: gradient, consistency, and plausibility/coherence, can be used to increase the strength of the argument for the presence of a mechanism of change.

Rather than lobbying for any subset or for the importance of any specific criterion, we underscore how these criteria are brought to bear on a hypothesis. Drawing inferences requires convergence of multiple criteria because they act in concert. For example, showing there is a gradient (dose-response relation) is hardly enough to infer that the mechanism may be involved. One also looks for this relation as being strong and associated with a plausible or coherent explanation. Yet, even if the gradient is demonstrated, it is still prudent to say, 'correlation is not causation.' However, there is no equally memorable or cryptic statement to handle the embellishments. Given our own non-memorable writing, we offer the following run-on friendly amendment: 'Correlation is not causation but oh boy does the case get stronger when the time line is established, when the association is strong, when there is some specificity so that the correlate (possible mechanism) is not associated with many outcomes, when manipulation of that correlate leads to change and there is a dose-response relation, when this relation has been replicated, and when there is a plausible and coherent explanation for the relation.'

Interpretation of what accounts for or explains a particular relation (mediator, mechanism) is not likely to come from a single investigation. By the very nature of one of the criteria (consistency), replication is required. Yet, apart from that criterion, the case for causation is built by a sequence of studies that may vary in the set of criteria they address and the clarity of the demonstration. After several studies, all or most of the criteria are met and one can state that

the mechanism of change is very likely to be x or y . There still is no certainty but as the studies accumulate, parsimony enters. That is, in light of the accumulated evidence and fulfillment of the criteria noted previously, the proposed mediator provides the simplest and best available account of the outcome.

Current methods for studying mechanisms of change in psychotherapy

The task for studying why and how therapeutic change occurs is conveyed nicely in the criteria for demonstrating a causal relation. Each criterion raises nuances that may account in part for why mechanisms have not been studied very much and why we do not really know why treatment techniques achieve change. Below we outline the statistical and methodological techniques typically used in attempts at the evaluation of mechanisms of change in psychotherapy.

The statistical mediation approach

Overview. Statistical evaluation can play a central role in addressing whether a particular construct accounts for change. Multiple regression techniques, path analysis, and structural equation modeling are prominent among the options or ways of discerning whether a given construct is likely to account for an outcome (Baron & Kenny, 1986; Holmbeck, 1997, 2002; Hoyle & Smith, 1994). For the statistically unsophisticated (a group in which we are Charter Members), it is useful to note that almost any statistical analysis that can partial out the effects of one influence versus another in relation to a particular outcome or whose results can show arrows pointing in all sorts of directions can be quite useful in

studying mediators, and can help demonstrate a strong association, specificity, and a dose–response relation.

Multiple regression analyses have been the most commonly used techniques and an overview of the logic involved conveys the benefits as well as the problems. These techniques have been nicely detailed elsewhere (Baron & Kenny, 1986; Holmbeck, 1997). We highlight the conditions for demonstrating statistical mediation by referring to Figure 1. The figure conveys four conditions describing the relations among three constructs or events for studying and demonstrating statistical mediation:

- 1 Efficacy test: The treatment or intervention (A) must be related to therapeutic change or treatment outcome (C).
- 2 Intervention test: The treatment has the specific effect intended; it must be related to the proposed mediator (B).
- 3 Mediator and change test: As a test that the proposed mediator is related to change in symptoms (or outcome domains), the mediator (B) must be related to therapeutic change (C).
- 4 Mediation, intervention, and change test: The relation between the intervention (A) and therapeutic change (C) must be reduced after statistically controlling for the proposed mediator (B).

Our purpose here is not to detail these techniques or their variants, but rather to illustrate the methods that are currently used in attempts to evaluate mechanisms of change in child and adolescent therapy. Statistical approaches can play a central role in establishing mechanisms of treatment by contributing directly to the many conditions required for demonstrating a causal relation, as discussed previously. Thus, statistical tests can demonstrate a strong association between the psychotherapeutic intervention and the hypothesized causal agent

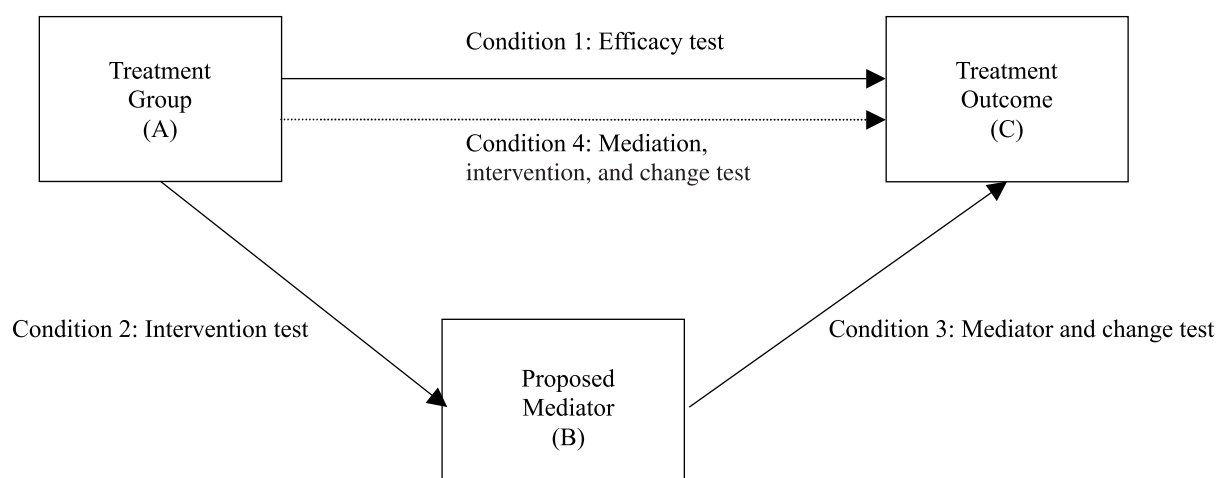


Figure 1 Four conditions to test statistical mediation among three therapy variables: the Intervention (A), the Proposed Mediator (B), and Treatment Outcome (C). *Note:* Solid lines refer to tests of univariate relations between variables. Dashed line refers to test of relation between (A) and (C) while statistically controlling for (B), as outlined in the text

(i.e., the mediator) and between that agent and the specified outcome of interest. Also, the statistical mediation approach allows for a test of the specificity of the associations between the treatment, proposed mechanism, and outcome. More directly, the demonstration that many seemingly plausible constructs do not account for or contribute to change in the outcome, with the exception of the one proposed by the investigator, strengthens the argument that this construct is a causal mechanism of change.

Limitations. As is invariably the case with statistical analyses, the various techniques are not free from controversy, and whether mediation is shown even if central conditions are met has been challenged (e.g., Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). A detailed discussion of these matters is beyond our present scope; however, we outline limitations that are relevant in the consideration of therapeutic mechanisms of change. Statistical analyses are often subject to misinterpretation in relation to mediation and causal analyses. The language of many data-analytic strategies lends itself to misconception in relation to the time line. For instance, regression analyses identify variables as *predictors* or independent variables and others as *outcomes* or dependent variables. Also, computer printouts may have lines and arrows to imply that one construct *leads to* another, something we refer to as the Kazdin–Nock Illusion.¹ The statistical analyses make no assumption of a time line for the variables that are entered; the distinction between antecedent (independent) and outcome (dependent) variables, from the standpoint of the steps of the statistical analyses, is arbitrary.

Clearly, the statistics are not at fault, but it is easy to misinterpret the results. The language used in reporting results often exacerbates the misunderstanding. In our prior CBT example, a typical conclusion might be worded that ‘changes in cognitions *predicted* recovery in depression’ (e.g., discriminant function). This same issue is common in other investigations, such as risk research where one variable (e.g., family stress) is said to *increase the risk* of a particular outcome (e.g., child deviance). Such communications could be mistaken to suggest that cognitive change or family stress came first in the

sequence of recovery and child deviance, respectively. The terminology suggests a time line but the design may be cross-sectional, a point to which we return.²

Design methods for studying mechanisms

Overview. Design refers to the plan or arrangement of the study and the strategies that will be used to rule out the various sorts of threats to validity and artifacts and biases. Randomized controlled clinical trials remain the primary method of demonstrating a causal relation between treatment and therapeutic change, although there are other rigorous experimental methods (Kazdin, 2003). In the context of psychotherapy research, assessment is typically conducted at pre- and posttreatment. Most psychotherapy research that is considered to reflect the study of mechanisms of change is of this type. A causal relation between the intervention (as a whole) and therapeutic change is nicely addressed in a randomized trial.

Limitations. Identifying specific mechanisms of change, or what active ingredient is responsible for the observed effects, is not usually permitted by such designs. The difficulty is not in the design per se, but the assessments commonly used in such designs. The problem is that the proposed mechanism and outcome are usually assessed at the same time, namely, at pretreatment and posttreatment. For example, showing that depressed patients improved and that their cognitions changed, as assessed at posttreatment, does not provide evidence showing that a change in cognitions was the causal mechanism of therapeutic change. It may even be generous to say that this pattern (change in symptoms and cognitions) is ‘compatible with’ cognitions being a mediator. This latter statement is not very helpful because the findings are also compatible with cognitions having no mediational role at all. A more subtle point might also be that even if cognitions did not change by the end of treatment, they still could have served as a mechanism. Something that caused a change during treatment might not be still evident at end of treatment. The study of mechanisms of change in therapy, or the reasons why therapy produces effects, requires assessment *during* the course of treatment to establish the time line. Showing that the proposed process changed and that therapeutic change followed (i.e., temporality) moves us closer to

¹ The Kazdin–Nock Illusion is a variant of the more familiar Figure/Ground Illusion (as depicted in the vase/profile figure most readers will know). In relation to statistical analyses and interpretation, the K–N Illusion works like this. An investigator sees arrows plotted from a data analysis or chart. These arrows point in a particular direction between one or more ‘predictors’ and an outcome. The data analyses and the arrows suggest a direction whether or not a time line permits one to infer that the predictor came before the ‘outcome’, i.e., whether a predictor actually was assessed and came before an outcome. The investigator *figures* that these arrows are good *grounds* for concluding a causal relation, ergo the resemblance to the Figure/Ground Illusion. This is an illusion.

² From the standpoint of language and statistical reporting of results, it probably would be advisable to use a term such as ‘statistical predictor’ to delineate a case when one variable statistically predicts an outcome but where the time line has not been established. Ordinarily, the term ‘correlate’ captures the same meaning, but this term does not handle the misinterpretations that the predictor may foster in evaluating various analyses where the investigator has a strong interest in directionality.

establishing that the process may be the mechanism involved.

The most common limitation of current techniques used to study mediation and mechanisms of change pertains to the absence of a time line. Consider an example in which CBT (A) might be administered to depressed patients and we propose that changes in cognitive processes are the mechanisms (B) leading to changes in symptoms (C). We assess depression and cognitions at pre- and posttreatment and conduct various statistical analyses to evaluate the relation of cognitive processes to treatment outcome. An obvious difficulty is that cognitive processes and symptom change were assessed at the same time (pre, post) and one cannot show whether one preceded the other. Symptom change could lead to changes in cognitions $C \rightarrow B$, vice versa ($B \rightarrow C$), or of course the 'third variable' problem (some $D \rightarrow B, C$). We may know that A (treatment) was responsible for change but, strictly speaking, we cannot tell why the change occurred, i.e., what the mediator is. Without a time line between B and C, the mechanism(s) of change cannot be known.

Assessing proposed mechanisms during treatment is necessary but not sufficient to show the time line between mechanism and outcome. The assessment of symptom change is required during treatment as well. An example from a well-studied area of therapy conveys these requirements. A great deal of research has focused on the therapeutic alliance, i.e., the extent to which the client and the therapist bond, work collaboratively, and have a positive relationship. There are many measures and views of alliance and their differences need not detain us. One view has been that alliance leads to therapeutic change. That is, during the course of treatment, an alliance develops and if this is a good alliance, this predicts and accounts for improvement in symptoms. To study this requires that one looks at alliance during treatment. Assessment is usually based on videotaping one or a few sessions early and mid-treatment, coding alliance from the tapes, and showing that alliance predicts later therapeutic change. To conduct this research requires that there are assessments during the course of treatment rather than just pre- and posttreatment assessment. There are scores of excellent studies showing that therapeutic alliance statistically predicts therapeutic change (e.g., Henry, Strupp, Schacht, & Gaston, 1994).

To demonstrate that alliance is a critical mechanism requires that the putative cause (alliance) come before the outcome (therapeutic change). It is possible that alliance and symptoms both changed in the middle of treatment and showing that alliance predicts later symptom change by itself does not show that alliance plays a causal role. Ideally, to show the temporal relation one must show that alliance has changed and symptoms have not but eventually change later. Just because symptoms are

not assessed in the middle of treatment does not mean they have not already changed. Indeed, it is conceivable that very early in treatment, clients get a little better (some symptom improvement) and that they then form a stronger or better alliance with the therapist as a result. That is, symptom change may mediate or lead to a strong alliance. Any demonstration that alliance predicts therapeutic change does not necessarily show that alliance is the mechanism – symptom change may have already occurred before the putative mediator operated.

Actually, some research to help sort this out has been completed. A study of psychodynamically oriented supportive therapy showed that changes in alliance early in treatment predict symptom change at the end of treatment (Barber, Connolly, Crits-Christoph, Gladis, & Siqueland, 2000). This is in keeping with a larger literature on this point. However, a critical addition was included. Both symptom change and alliance were assessed at multiple points. The evidence indicated that symptom changes early in treatment predicted alliance and that alliance also predicted further symptom change. This suggests that symptom change and alliance mutually influence each other. Assessments of both symptom change and alliance were completed at multiple points during the course of treatment to identify these interesting relations.

As a more general point, establishing the time line requires that assessment of the putative cause occurs before the putative outcome. To accomplish this may require that both the 'outcome' (e.g., symptoms) and proposed mechanisms are assessed during treatment. Either assessing proposed mechanisms during treatment or the more stringent requirement of assessing both processes and outcomes repeatedly over the course of treatment is rare indeed. It is not necessarily that we need many such studies to adopt these more onerous assessment and design requirements. However, we do need some.

Recommendations for research

The impetus for this article is to make the case for more research on mechanisms of therapeutic change. This is not a new plea. However, there are at least two reasons to make the plea anew. First, there is now a keen interest in transporting gains of therapy research to clinical practice. The appeal for more work on mechanisms is in response to this interest. It is still the case that the very best practice will come from the best science, i.e., theory and research that focus on understanding. The difficulty with efficacy research is not the lack of studying 'real' patients, with 'real' clinicians, in 'real' clinics. Rather, the controls of the laboratory have not been used to do what such research does best, namely, understand how something works. Second, there is a belief that mechanisms are being studied and

that methods currently in place in most studies can isolate mechanisms. Articles on mediators, moderators, and statistical analyses might unwittingly imply that various analyses can identify mechanisms when some of the logical conditions have not been met. We count among those key conditions showing a time line of the proposed mediators and outcome. We very much value research that may not demonstrate this time line (e.g., as generating hypotheses, as describing other relations among outcome and other domains). We are only concerned here with demonstrating mechanisms of change. In any study proposed to study mechanisms, the reader must ask: could the outcome have occurred before the proposed cause? In most therapy research considered to support the study of mechanisms, it is quite possible that the 'outcome' really changed the 'mediator.' This will not do on logical grounds. Also, was the mediator shown to lead to some change in the outcome? Statistical analyses can contribute enormously to answer the question; however, assessment and design rather than statistics determine whether the matter of the time line can be addressed.

We propose several recommendations to enhance our understanding of the mechanisms of therapeutic change. These recommendations correspond to several of the criteria for demonstrating the presence of a causal mechanism outlined previously, as presented in Table 1. In keeping with science more generally, it is notable that a broad portfolio of research is needed and no one recommendation is sufficient.

Include measures of potential mediators in treatment studies

The study of mechanisms requires the investigator to propose the possible bases for change. This is an obvious recommendation because a precondition for studying mechanisms is considering this a worthy topic and one to influence measurement selection. Many therapy studies discuss mechanisms in the Introduction and Discussion sections, but there is nothing in the Method or Results sections to provide

evidence. Methods for assessing constructs over the course of treatment have been nicely described elsewhere (Eddy, Dishion, & Stoolmiller, 1998). Studies occasionally include such measures and examples have been reviewed elsewhere (Weersing & Weisz, 2002). We hasten to add that inclusion of such measures is a requirement to evaluate causal relations, but without appropriate design features (e.g., multiple assessments of supposed mediators and outcomes during the course of treatment) such measurement is insufficient.

Assess more than one mediator

The accumulation of evidence would profit from the assessment of more than one mediator in a given study. It is rare that one mediator is studied and hence there may be little value in raising the bar even higher by recommending the assessment of two or more mediators. Clearly, this is one of the more demanding recommendations we provide. Assessing one mediator requires that prepost treatment designs are modified to include at least one additional assessment occasion during treatment (premidpost treatment designs). During treatment the supposed mediator and outcome (e.g., symptoms) must be assessed to help establish the time line. Recommending the assessment of more than one mediator during treatment means that the assessment battery (e.g., how many measures) may be increasing with each mediator that is to be studied. In laboratory (so-called efficacy) studies, this may not be particularly onerous. Increasing the assessment battery in clinical research invariably has a cost in patient and therapist reaction.

Nevertheless, as the occasion allows, the assessment of multiple mediators in a given study has enormous benefits. If two or more mediators are studied, one can identify if one is more plausible or makes a greater contribution to the outcome. In addition, the assessment of multiple potential mediators within individual studies makes sense economically, given the tremendous amount of time and resources needed for each investigation. Across

Table 1 Research recommendations for studying mechanisms of change in psychotherapy studies

| Criteria | Recommendations for research |
|------------------------------|---|
| 1 Strong association | Include measures of potential mechanisms of change in psychotherapy studies. |
| 2 Specificity | Assess more than one potential mechanism, as well as possible confounding variables. |
| 3 Gradient | Assess whether greater changes in proposed mechanisms are related to subsequent, greater changes in outcomes. |
| 4 Experiment | Intervene to change the proposed mechanism of change. |
| 5 Temporal relation | Establish a time line by using continuous measurement of proposed mechanisms and outcomes of interest. |
| 6 Consistency | Replicate observed effects in different studies, samples, and conditions (e.g., naturalistic and laboratory settings). |
| 7 Plausibility and coherence | Use theory as a guide to select potential mechanisms for focus of study, as well as to explain observed effects and integrate them into broader knowledge base. |

many studies, some mediators may repeatedly emerge as possible contenders while others fall by the wayside. This would be a very important advance.

Assess whether there is a gradient (dose-response relation)

Examine whether the proposed mechanism is related to the outcome in a way that suggests a dose-response relation. The absence of such a relation does not preclude a causal relation in part because not all relations are linear. Also, variations of responses among different subgroups (moderator effects) may mask such a relation. Even so, a dose-response relation between the proposed mechanism and outcome can add considerably to the overall argument that a particular mechanism may play a central and causal role.

Intervene to change the proposed mediator

When possible, it is useful to conduct an experiment in which the proposed mediator is in fact altered or varied across groups. For example, groups randomly composed might be assigned to low, high, and medium levels of a proposed mediator. Results showing that outcome varies directly as a function of levels of the mediators, assessed during treatment, would be quite powerful support. There are nuances that require accumulation of studies. It is possible that changing some mediator actually works for a different reason from the one proposed by the investigator. For example, changing level of expectations (a proposed mediator) or alliance (bonding statements of the therapist) might work because of other constructs (e.g., how much hope is developed in the patients). Intervening to change a mediator is an excellent strategy. Here too, assessing more than one mediator would be helpful in understanding why change occurred. That is, one can rule out as well as provide evidence for the operation of various mediators.

Address the time line

It is important whenever possible to establish that the proposed mediator is changing before or in advance of the outcome. For example, changes in cognitions and depression at the end of treatment cannot support that changes in one were responsible or led to changes in the other. In addition, if the mediator is studied in mid-treatment, it is useful to assess the proposed outcome as well. It is possible that the outcome (symptom change) actually preceded change in the mediator. We have indicated the need for assessment somewhere between pre- and posttreatment assessment. Actually, assessment on more than one occasion during treatment can provide better information

on the time line of mechanisms and outcomes and the possibility of bi-directional changes and causality.

Naturalistic studies

Understanding mechanisms through which therapeutic change occurs may well profit from non-therapy research and naturalistic studies. If one is proposing a mediator of change, is there a sample, population, or setting in which this mediator may be expected to vary on naturalistic grounds? For example, if changing parenting style is proposed to explain why a parent- or family-based treatment is effective, naturalistic studies examining families with and without these practices and the short- and long-term child behaviors with which these are associated are relevant.

Naturalistic studies by themselves may not be sufficient for all the reasons that quasi-experimental designs are not true-experimental designs. Yet, such evidence can be enormously helpful. Many advances in understanding cancer, heart disease, and stroke began by looking for variation in putative mechanisms among individuals with varied or different outcomes (e.g., occurrence, nonoccurrence; severity of the outcome). Similarly, in children, advances in understanding low levels of lead as a predictor of hyperactivity and diminished IQ came from naturalistic studies (Needleman, 1988; Needleman, Schell, Bellinger, Leviton, & Alldred, 1990). Observing processes that may be operative in the natural environment and their short- and long-term correlates can be very useful.

Laboratory studies of therapeutic processes

Laboratory studies of therapy encompass many types of research and we mention two here. Efficacy studies refer to outcome studies under well-controlled situations. We advocate such studies in the context of this article with a slight twist. Controlled studies of therapy outside of the context of clinics are in the best position to study mechanisms. The careful control afforded such research is precisely what is needed. The problem with efficacy studies, in our view, has been their almost exclusive focus on *outcome* questions under highly controlled situations. Naturally, one wants to then ask, 'But will these outcomes be obtained on the battlefield?' However, more studies are needed with the high levels of control that investigate potential mechanisms. The study of mechanisms is precisely why we want laboratory research. Studying mechanisms in real-world settings is possible but much less advisable than in the laboratory.

Another illustration of laboratory research is research on animal models. Granted, many of the mediators of therapy may not be amenable to mouse models. Also, some of the interventions that

might be of interest to a therapy researcher cannot easily be studied. For example, our own model of therapy (Ear Movement Desensitization) is based on people building trust by falling backward and catching each other. We have not been able to get mice to do this to test our view that trust-building decreases stress and cortisol. Even so, some of the mechanisms of therapy might be studied in the lab and we ought not to be shy about them or shy away from them.

We mention some of the ways of studying mechanisms with no intent to exhaust all of the options. The issue in any given study is whether some of the conditions required to provide evidence for a mechanism of action are met. The most critical point is the need for multiple studies and strategies, among those recommended and indeed others, that can converge on meeting the criteria we have outlined.

Use theory as a guide to study mechanisms

Whenever possible, theory is useful to guide research. Theory refers to an explanatory statement that is intended to account for, explain, and understand relations among variables, how they operate, and the processes involved. Theory can focus on etiology, change, and maintenance of a problem. In the context of the present article, we focus on theories of change, i.e., how changes come about. Investigators ought to propose such theories and test them. The advantage of a theory is that it may suggest many contexts and experimental arrangements (e.g., laboratory and naturalistic studies) in which critical hypotheses can be tested, beyond treatment studies.

Conclusions

Understanding how psychotherapy works can serve as a basis for maximizing treatment effects and ensuring that critical features are generalized to clinical practice. Research examining psychotherapy for children and adolescents has increased in frequency and sophistication over the past several decades, and the gains have been impressive. Despite this progress, research advances are sorely needed in studying the mechanisms of therapeutic change. It is truly remarkable that after decades of psychotherapy research, we as clinical researchers cannot confidently provide an evidence-based explanation for how or why even our most effective interventions produce change. More surprising is the infrequency with which this issue, or research addressing this issue, appears in the research literature. There are few studies and of these almost none meet the criteria of establishing the time line between the mechanism and behavior change (see Weersing & Weisz, 2002 for review), let alone one or two other requirements for establishing the relation.

Although prior research has not succeeded in demonstrating the operation of potential mechanisms of change, it has provided important groundwork on which future studies should build. For instance, several research groups have included assessments during the course of treatment, rather than only using pre- and posttreatment assessment, in order to investigate potential mechanisms of change (e.g., Eddy & Chamberlain, 2000; Kolko, Brent, Baugher, Bridge, & Birmaher, 2000). Although neither of these studies was able to demonstrate that the proposed mechanism changed before the outcomes of interest, the designs used in these investigations represent a great improvement over prior studies, and signal progress in research on mechanisms of change. Existing studies have attempted to evaluate only a handful of potential mechanisms of change. Many others have been advanced (Brent & Kolko, 1998) but have not received empirical attention. Examples of such mechanisms include parent and child expectancies for treatment, therapeutic alliance, and other 'common factors.' No doubt others could be readily proposed. Most clinical researchers and practitioners have a conceptual view about why their treatment 'works'; we are only underscoring how critical it is to put these explanations to the test and that these tests can greatly enhance clinical care. Indeed, the study of mechanisms of change is relevant to all psychotherapy researchers, regardless of theoretical orientation. All researchers and clinicians have beliefs about how treatment works, and research on mechanisms may help bring order to the hundreds of treatments currently in use by delineating a much smaller set of mechanisms through which these treatments operate.

The scientific study of mechanisms of change is certainly not an easy path on which to embark. Advances in science in general add to the excitement and complexity of the tasks. That is, there is not likely to be a single mechanism for a technique, just as there is no simple and single path to many diseases, disorders, or social, emotional, and behavioral problems (e.g., lung cancer, attention-deficit/hyperactivity disorder). Also, two children in the same treatment conceivably could respond for different reasons. These complexities ought not to serve as deterrents to progress. Indeed, they are evident in areas we are already pursuing. For example, evidence-based treatments are not effective for all children with a particular problem. We would argue that studying mechanisms has both benefits in understanding the complexities of therapy and in providing better (more effective) treatments. There is much work to be done in this very important area, and the potential benefits are tremendous for researchers, and most importantly for children and families seeking mental health services. We hope the methods and recommendations advanced in this article will provide a useful starting point and a stimulus for

forthcoming work on mechanisms of change in child and adolescent psychotherapy.

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