Child and adolescent psychotherapy research has advanced remarkably. Well over 1,000 controlled outcome studies can be identified (e.g., Kazdin, 2000). Meta-analytic reviews have consistently concluded that treatments are effective and, in fact, produce rather strong effects (large effect sizes) (e.g., Weisz, Weiss, Han, Granger, & Morton, 1995). Outcome studies continue to emerge and their methodological quality continues to improve (Durlak, Wells, Cotten, & Johnson, 1995). Empirically supported treatments have been identified for several problem domains including anxiety, depression, and conduct disorder, to mention a few (see Kazdin & Weisz, 1998; Lonigan & Elbert, 1998). Guidelines for clinical practice have emerged to take into account mounting empirical evidence (e.g., American Academy of Child and Adolescent Psychiatry, 1998). Clearly, enormous strides have been made. Absent from the research is attention to theory, mechanisms, and processes that explain how therapy works. Hughes [this issue] nicely articulates the problem and conveys how research that neglects efforts to understand therapeutic change is shortsighted. The present comments build on key themes Hughes has raised. Specifically, these comments elaborate why there is need for theory in child and adolescent therapy research, the different foci of theory, and the progression of description to explanation in research.

THEORY AND CHILD AND ADOLESCENT PSYCHOTHERAPY

Why Theory is Needed

For present purposes, 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. The theory encompasses but goes beyond the specific empirical relations among the variables and phenomenon of interest. Theories can and do vary widely in comprehensiveness in terms of the range of influences they encompass and phenomena they explain.

In the area of psychotherapy, it is important to distinguish theory from approach. Much of so called “theory” in psychotherapy research reflects an approach or orientation toward therapy (e.g., psychoanalytic, cognitive-behavioral, familial, and others). An approach often begins with a broad view of processes (e.g., thwarted impulses, maladaptive family processes, distorted cognitions) considered to have wide applicability across many different disorders. The distinction, here, between theory and approach pertains to the level of abstraction and also the testability of key propositions. Typically, an approach is applied to many clinical problems and treatment techniques. In fact, an approach may include scores of constituent theories, not all of which are compatible. The pervasiveness of approaches is so evident that it is the air we breathe, and hence we do not think of them routinely, unless, of course, we are gasping for air after reading a journal, attending a convention, or chatting with someone from an approach quite different from our own. Theory, as referred to here, tends to be more focused on a particular problem and treatment.

There are several reasons why theory is needed in child therapy. First, over 550 treatments are currently in use for children and adolescents (Kazdin, 2000). This count omits various combinations of treatments, eclectic hybrids, and the uninterrupted, not to mention embarrassing, continued proliferation. The vast majority of treatments in use have never been studied empirically. Among those that have been studied, it is possible that their effects can be traced to some common set of mechanisms or processes that span several techniques. Theory can bring order and parsimony to the plethora of interventions.

Second, a large number of factors (moderators) can and do influence treatment outcome. Characteristics of the child (e.g., age of onset, severity of dysfunction), the parent (e.g., psychopathology, stress), the therapist (e.g., experience, personality style), and family (e.g., relationships, discord) are a small portion of factors that can moderate outcome. With an unlimited number of factors from which to draw, theory can focus empirical tests by positing those factors likely to make a difference. Also, theory can help to identify why a particular factor has impact. For example, high levels of parental stress can decrease the effectiveness of child therapy (Kazdin & Wassell, 1999). It is very helpful to posit and test the means through which this effect operates (e.g., poor parent adherence to treatment prescriptions, parent psychopathology, countertherapeutic influences at home). Once understood, one can make informed efforts to neutralize or attenuate those factors that thwart treatment progress.
Third, an obvious goal of treatment is to optimize therapeutic impact. Theory can help understand the processes that account for therapeutic change and hence, ought to be fostered and maximized. To improve therapy effects, contemporary research usually adds more treatment (e.g., more sessions during treatment or maintenance sessions after the core treatment has ended). This is based on the view that more is likely to be better. The importance of theory is evident from the query, “More what?” Serving time is not likely to be the critical factor accounting for therapeutic change. The “more” might entail more practice, rehearsal, exposure, role-playing, or problem solving. Theory is important to understand what is critical in treatment and how it can be deployed.

**Theory of What?**

There are all sorts of facets that may be the object of theory from which intervention research might draw including a conceptualization of how a particular clinical problem comes about, how it is maintained, how it ends, reappears, and so on. Two foci of theory are particularly pertinent. First, theory of dysfunction refers to the conceptual underpinnings and hypotheses about the likely factors leading to the clinical problem or pattern of functioning, the processes involved, and how these processes emerge or operate. Research on the nature of the clinical disorder is likely to focus on various risk and protective factors, paths and trajectories, and how early development results in subsequent dysfunction.

There are clear benefits of connecting treatment to what we know about the factors related to onset of a disorder. First, the factors implicated in the development of the problem may be directly relevant to treatment. This is nicely illustrated with parent management training, a behavioral treatment that is one of the better studied interventions for oppositional and aggressive children (Brestan & Eyberg, 1998; Kazdin, 1997). The treatment is based on the view and considerable evidence that inept discipline practices influence the development of aggressive behavior in children (Patterson, Reid, & Dishion, 1992). Parent training alters these practices by focusing directly on how the parents interact with their children. In this case, the theory of what factors contribute to the problem overlaps with the theory of how change can be achieved. The overlap stems in part from the fact that inept parenting practices can influence onset and maintenance of the problem and be used to develop nonaggressive and prosocial behavior.

Second, factors related to the onset of the problem may have relevance as moderators of therapeutic change. For example, parent stress, psychopathology, harsh child-rearing practices, and family adversity are risk factors for the onset and maintenance of conduct disorder in children (Kazdin, 1995; Stoff, Breiling, & Maser, 1997). These factors also are associated with
diminished responsiveness of these children to treatment. In this case, research on the factors related to the disorder are quite useful for identifying those cases likely to vary in their responsiveness to treatment.

Characteristics of the clinical problem also may serve as a moderator, as reflected by subtypes of a disorder. Subtypes can reflect differences in factors leading to onset, special characteristics of those with the disorder, and differences in prognoses. For example, many sexually abused children develop cognitions that the world is a dangerous place, that adults cannot be trusted, and that one’s own efforts to influence the world are not likely to be effective (Wolfe, 1999). Based on this understanding of the problem, one might predict that a subtype of sexually abused youths with these cognitions would respond less well to treatment, as, for example, measured by posttreatment prosocial functioning. If these cognitions are not altered in treatment, the children may be restricted in social activities compared to similar children without these cognitions. Perhaps another study using this information would evaluate if the effectiveness of treatment could be enhanced by including a component that focuses on these cognitions. In general, research on moderators and subtypes can suggest hypotheses for treatment studies. Interestingly, viable hypotheses can be formulated without understanding how the moderating influences work or how the subtypes emerge.

Knowledge about the onset of a problem may vary in the extent to which it is relevant as a guide to treatment. Indeed, in some cases, theory of onset and identification of factors implicated in its onset may not help to guide treatment. When antecedents and causal agents (e.g., child physical abuse, sexual abuse, parental marital conflict) have exerted their effect (e.g., development of psychopathology, odd attachment patterns), it is not axiomatic that they are the appropriate, optimal, or indeed relevant focus of treatment. This does not in any way mean that change is impossible. It only means that understanding etiology may not serve as a guide to treatment once the disorder or dysfunction is present.

Theory of therapeutic change refers to the conceptual underpinnings of the process(es) of change during treatment. The focus is on what therapy is designed to accomplish and through what means and processes. How will the procedures used in treatment influence the dysfunction, develop new repertoires, or simply overwrite, cancel out, or make the problem nugatory? The key question guiding the treatment is how does this treatment achieve change? The answer may involve an array of processes (e.g., memory, learning, neurotransmitters, family interaction) at many levels of analysis.

There is very little evidence available to explain how therapy works, even among well-studied treatments. For example, for adults, cognitive therapy for depression is one of the more well-investigated treatments (Hollon & Beck, 1994). The treatment is based on the view that cognitive schema contribute to and account for depression (theory of dysfunction) and that
changes in these schema and related processes will ameliorate depression (theory of change). Are the benefits of cognitive therapy due to changes in cognitive processes, that is, are these the processes that lead to and account for change? I do not believe this has been shown, although this does not detract from the accumulated outcome studies. There is no need to single out cognitive therapy for depression. Rather, the question is why does this or any other particular therapy effect change?

At least three steps are required to conduct the requisite research to isolate the answers, namely, specifying a conceptual view of the processes or factors responsible for change, developing measures of these processes, and showing that these process change during treatment. This latter requirement is needed to establish the time line; that is, processes are changing and are not merely concomitant effects of symptom improvement. The design of most studies does not provide for assessments during the treatment course to identify how the change process unfolds. Statistical techniques and tests (e.g., to evaluate mediators, paths) cannot provide the needed information if mediators and outcomes are assessed at the same point in time.

**MOVEMENT FROM DESCRIPTION TO EXPLANATION**

There is a need for much more research that attempts to explain how and why therapy achieves and induces change. Therapy research is largely restricted to identifying relations between various treatment and control conditions and outcome. I refer to this as descriptive research and distinguish this from explanatory research. For present purposes, descriptive research tests relations with little or no emphasis on explanation. Explanatory research includes an explicit component that focuses on mechanisms, understanding, processes, and why the effects are achieved. Consider descriptive and explanatory as end points of a continuum rather than distinct categories. Indeed, when descriptions are fine-grained, elegant, and process related they often are explanatory.

In virtually all therapy research, and for that matter risk factor research of the ilk cited by Hughes, investigators explain all sorts of things (in Introduction and Discussion sections). Although such material is often rich with

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1 It is often thought that statistical tests can make certain causal paths and mechanisms implausible, that is, at least rule out various mechanisms that are operative by showing that a proposed mediator (e.g., cognitions) cannot account for outcome. Yes and no. The hypothesis to be tested is whether change in a proposed mediator leads to change in the outcome. Evaluating the level or characteristic of the mediator at the time of outcome (rather than during treatment) does not necessarily reflect the role of the mediator during treatment. Evaluation of the proposed mediator at the end of treatment tests whether the mediator lingers (is still present). This is, as it were, evidence of a “smoking gun” and can be quite useful. Yet, absence of a smoking gun does not mean there has been no shooting; a mediator that served its role during treatment may not be evident or as evident once therapeutic change has been achieved.
Table 1

<table>
<thead>
<tr>
<th>Concept</th>
<th>Defined</th>
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<tbody>
<tr>
<td>Correlate</td>
<td>Characteristic associated with a problem or other characteristic of interest</td>
</tr>
<tr>
<td>Risk factor</td>
<td>Antecedent and predictor of the characteristic or problem of interest</td>
</tr>
<tr>
<td>Cause</td>
<td>A factor that, when altered, leads to a change in the problem or characteristic of interest</td>
</tr>
<tr>
<td>Mechanism</td>
<td>Ways in which the cause exerts influence; how the process unfolds, why it works</td>
</tr>
</tbody>
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Note. These terms do not exhaust the ways in which a particular characteristic can be related to an outcome, but are selected here to convey a progression and levels of understanding. These and additional terms, research strategies to move from one to the other, and examples where progress has been made are elaborated elsewhere (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer et al., 1997).

implicit hypotheses, this is not my intended meaning of explanatory research. Explanatory research empirically tests a mechanism or reason why some relation occurs. The move from descriptive to explanatory research is critical for the development of interventions.

Progression of Understanding

Four concepts convey the movement from description to explanation and how one can progress toward understanding how and why change occurs. The concepts include correlate, risk factor, cause (or causal risk factor), and mechanism, as defined in Table 1. These are familiar concepts in many ways but the progression is worth noting with an example. Early research on cigarette smoking included findings that people who had lung cancer were more likely to be cigarette smokers; that is, smoking and lung cancer were correlated. The time line might be obvious (i.e., smoking must have proceeded lung cancer). The time line was better demonstrated in longitudinal studies in which cigarette smokers and nonsmokers were followed over time. The greater incidence of cancer among smokers established that smoking was a risk factor. Evidence was brought to bear from case-control studies to suggest a causal relation between cigarette smoking and lung cancer (e.g., by showing a dose-response relation, ruling out a variety of competing explanations). The causal relation was firmly established from experimental intervention studies. Experimentally inducing smoking among animals in controlled experiments revealed the link between smoking and cancer (causal relation). This leaves unanswered the mechanism through which smoking operates to produce lung cancer. Only recently has that been identified. 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 provided a critical link between the
causal factor (cigarette smoking) and outcome (lung cancer) by conveying precisely how one leads to the other at the molecular level. Thus, beyond the demonstration of a causal relation, a fine-grained analysis of mechanisms is important as well.

There are not many examples of this type of movement and progression in research on theories of dysfunction or theories of therapeutic change. Indeed, much of the risk factor research in developmental psychopathology does not progress to the next step. To be sure, we have learned a great deal. For some disorders, we now have piles of risk factors but do not have a hint how these work to develop a particular outcome. Rather than moving forward to the next level of understanding, research often moves laterally by testing whether some finding is also true of this or that group. Thus, selected risk factors are identified for a particular disorder (e.g., conduct disorder) in x (where x equals boys, children, one ethnic group, or children from one country). Now we test whether the relations are also true of y (where y equals girls, adolescents, another ethnic group, or children from another country). The research is justified as being on a path toward understanding how processes operate, but these processes and differences in processes between groups are rarely examined. Yet, such research, to quote the summary statement of my dissertation committee, is “conceptually bereft and exceptionally uninspired.” The findings from risk factor research do not automatically serve as a reasonable basis for designing interventions unless more is understood about how the factors operate and whether they play a causal role (see Kraemer et al., 1997).

There are notable exceptions where palpable progress is evident. For example, in relation to aggressive behavior, much of the work of Patterson and his colleagues has demonstrated causal factors and models (e.g., Dishion & Patterson, 1999; Dishion, Patterson, & Kavanagh, 1992; Patterson et al., 1992) with parent management training, already mentioned, as one of the viable products. Another example is the research on ingestion of lead and its impact on learning and behavioral problems in children. A series of human and animal studies on lead ingestion convey the movement from correlate, risk factor, and cause; studies of the impact of lead on specific sites in the brain move closer to the mechanisms through which such effects occur (see Needleman, 1988; Needleman & Gatsonis, 1990). Occasionally, intervention research illustrates a progression by showing that a correlate (e.g., the extent to which a child reads to his or her parents) of a particular outcome (e.g., school achievement) plays a causal role (Hewison & Tizard, 1980; Tizard, Schofield, & Hewison, 1982). Direct intervention in a randomized controlled trial established that increasing reading, but not merely increasing exposure to reading materials, improves school achievement.
The goals of psychotherapy are to improve adjustment and adaptive functioning and to reduce maladaptive behaviors and various psychological and physical complaints. The methods to do this are based on interpersonal influences and psychological processes (e.g., learning, persuasion, social support). Although theory and research from developmental psychopathology may serve as rich resources for hypotheses about mechanisms of influence, conceptual ties to diverse areas of psychology ought to be sought. As developmental psychopathologists, we often raise the flag of how unique childhood and adolescence is and how failure to consider this uniqueness is perilous. Yet, the advisability of drawing on diverse areas of psychology and related disciplines is underscored by conveying the challenge Hughes raises in a broader context.

First, there are rather intriguing effects of psychotherapy that extend beyond the social, emotional, and behavioral problems to which therapy is often directed. Such effects beg for explanation. As quick and tantalizing examples, psychotherapy improves symptoms of physical health (Luborsky, Crits-Cristoph, Mintz, & Auerbach, 1988), increases survival rates among terminally ill cancer patients (Spiegel, Bloom, Kraemer, & Gottheil, 1989), and increases fertility among infertile couples (Domar, 1998). How can these effects occur? What hypotheses might be reasonable contenders to explain one or more of these effects?

Second, when we discuss therapy, the usual focus is on those interventions recognized as having a pedigree or heritage that we can trace within medicine, psychology, psychiatry, and allied disciplines. Stated simply and perhaps simplistically, most mainstream treatments begin with at least one of three premises: (a) psychoanalysis or one of its offshoots is a very reasonable treatment, (b) psychoanalysis or one of its offshoots is misguided, or (c) my treatment is terrific. There is large genre of other treatments less in the mainstream. For example, interventions for problem children and adolescents include sending youths to boot camps (e.g., with basic training and military models), ranches (e.g., where children take responsibility for the care of a horse), and the wilderness so that youths can breathe fresh air and bond with nature. We scoff at these because they are not theory based, have little or no evidence in their behalf, and have emerged quite out of the mainstream. However, we ought to take “scoff drops” to suppress these reactions. Most of mainstream treatment is not theory based and has no evidence on its behalf (Kazdin, 2000). I am not advocating marginal and seemingly wacky treatments (MSWTs) or interventions that ignore all that is known about a disorder. Rather, I merely wish to note that if “based on theory and research” were used as the criterion for grouping therapies, many mainstream treatments might well be classified as MSWTs.
Understanding mechanisms and processes is a way to identify how change is achieved, and from that can emerge different ways in which these mechanisms can be activated. Many existing and “legitimate” treatments would readily fall by the wayside and perhaps, many treatments struggling for legitimacy might look reasonable. The emphasis on different treatment techniques and approaches distracts us from the main unifying questions. How does (any) therapy achieve change? How might this mechanism or process be activated or invoked through different strategies?

Finally, psychotherapy, as a source of influence on human functioning or class of interventions has siblings that are often kept in the closet. As examples, controlled studies have shown that hypnosis, placebos, and exercise can all lead to change in adaptive functioning and improvements in mental and physical health. Similarly, participation in religion is rather consistently associated with reduced rates of suicide, depression, and death from heart disease, after ruling out all sorts of other influences (e.g., rates of smoking, obesity; Levin, 1994; McCullough, 1995). Moreover, there is a dose-response relation in which the mental and physical health benefits increase with orthodoxy or strictness of the sect to which one belongs within a religion. Clearly, as scientists interested in psychotherapy, we ought to understand change processes wherever they occur, but perhaps especially in related domains where similar outcomes (improved mental and physical health) are also achieved. The question of interest is how does one intervene to change social, emotional, and behavioral characteristics? Therapy is part of a broader class of influences on individual functioning. Theory that elaborates how therapy works might help us understand change processes in human functioning more generally. The other side is, of course, equally true. Theories that explain how other change methods work might well inform therapy.

At present, we know little about the process of therapeutic change. In science, one cannot criticize an area of work for not knowing. Certainly, that is not the point of the present comments. Rather, the present comments, in keeping with the cogent arguments of Hughes, are directed at a related problem, namely, there is little evidence that we are making concerted efforts to understand; in other words, we are not trying. We can reliably produce change with some treatments. How do these treatments work—that is, through what processes, in what ways, and for whom and why—for these individuals? It is likely that once this is answered for one or a few treatments, we will know a great deal about many treatments and possibly more, generally, about how humans develop and change.

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