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Causal Reasoning Skills Training for Mental Health Practitioners: Promoting Sound Clinical Judgment in Evidence-Based Practice

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Recent calls to action for competency-based training, evaluation, and credentialing of mental health professionals focus heavily on instilling the knowledge and skills needed for performing evidence-based assessment and treatment. We propose the content of a companion training curriculum in clinical decision-making that reflects the pervasive and indispensible role of causal reasoning in clinical practice. Contents of the proposed curriculum include review and discussion of the following areas: (a) domains in which practitioners are routinely required to make and evaluate causal inferences; (b) definitions of key concepts related to causality and errors in causal reasoning; (c) guidelines for evaluating the internal and external validity of findings in empirical studies; (d) guidelines for formulating and evaluating working theories of the origin and/or maintenance of client presenting problems; and (e) methods for planning, targeting, and evaluating interventions. This curriculum is designed to help mental health practitioners use causal modeling to enhance case conceptualization, develop intervention objectives, and prioritize and target foci of interventions that use evidence-based treatments or practice elements. Practitioners who use causal modeling to guide clinical practice are, in effect, deliberately generating causal hypotheses, implementing causal experiments, and evaluating outcomes as they monitor client response to intervention. Causal reasoning competencies may be enhanced through application of causal modeling diagrams to clinical case examples. Development and implementation of a causal reasoning curriculum will provide a basis for "value-added" research regarding its benefit to practitioners in terms of enhanced clinical competencies, decisions, and improved client outcomes.

Keywords: causal reasoning, clinical judgment, skills training, evidence-based practice, mental health

Recent calls to action from around the world have been issued to transform mental health service delivery through workforce education and training (Hogan, 2003; Huang et al., 2005; Power, 2005). Primary among these recommendations are calls for nationwide training in evidence-based practice (EBP)—a call that has received considerable impetus through policies that link remuneration for services to the provision of specific evidence-based assessments and treatments. As a result, large-scale initiatives are currently underway to provide competency-based training and credentialing in EBP (Fouad et al., 2009; Kaslow, Celano, & Stanton, 2005; Kaslow et al., 2009).

EBP is commonly defined as an approach to clinical practice in which the practitioner, in consultation with the client, uses the best available evidence gleaned from systematic research and clinical experience to choose intervention options that are best suited to the client's needs, circumstances, strengths, and informed wishes (Wilton & Slim, 2012). Evidence-based interventions are approaches to treatment that are based in theory and repeatedly subjected to rigorous scientific evaluation that demonstrates significant benefit with one or more populations (American Psychological Association, 2002, 2005).

Major progress has been made in advancing evidence-based assessment and treatment for various disorders in the child, adolescent, and adult psychotherapy fields (e.g., Chalmers, 1993; Haynes, Smith, & Hunsley, 2011; Hunsley & Mash,

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2008; Kazdin & Weisz, 2003). However, commensurate progress has not been made in promoting skills in causal reasoning that complement the implementation of evidence-based interventions (Kazdin, 2008; McLennan, Wathen, MacMillan, & Lavis, 2006). Indeed, causal reasoning skills have been identified as a core competency for the scientific practice of psychology (Bieschke, Fouad, Collins, & Halonen, 2004; Nezu & Nezu, 1995; Stricker & Trierweiler, 1995). A rationale for the role of causal reasoning skills training is found in its promise for improving clinical judgment and decision-making in a wide variety of domains involving evidence-based assessment and treatment. These domains include assembling causally relevant information into a working theory of the origin, maintenance, consequences, and potential remediation of client problems; identifying and prioritizing intervention foci and objectives; aligning selected intervention foci with appropriate practice elements theorized to achieve those objectives; implementing a treatment plan that uses empirically based strategies; and monitoring response to treatment to determine treatment effectiveness or the need for timely "course correction." Causal reasoning skills training extends well beyond training students to implement empirically supported treatment protocols with fidelity. Indeed, the aim of such skills training is to develop well-rounded evidence-based practitioners equipped with a broad range of knowledge and skills required for gathering and integrating clinical information into a clinically actionable working theory; tailoring interventions to clients' specific needs, strengths, circumstances, and available resources; and evaluating the effectiveness thereof (Collins, Leffingwell, & Belar, 2007).

In light of its pervasive and indispensable role in EBP, we propose that foundational concepts, strategies, and skills of causal reasoning be formally incorporated in the training of mental health practitioners. Indeed, much of the literature on prepracticum training of mental health professionals advocates for the inculcation of such skills as critical scientific thinking, problem-solving, organized reasoning, application of scientific methods to clinical practice, and evaluation of intervention effectiveness (e.g., see Hatcher & Lassiter, 2007; Hatcher, Wise, Grus, Mangione, & Emmons, 2012). The curriculum presented below "unpacks" these broad domains into constituent clinical skill elements that are amenable to competency-focused training, evaluation, and remediation.

As described in detail below, causal reasoning involves embedding proposed explanations of phenomena within a conceptual matrix of theorized causal precursors, mechanisms of action, causal consequences, mediators, moderators, and their associated pathways of influence. Given that a broad range of mental health disciplines have embraced EBP as a professional standard (e.g., Fouad et al., 2009; Nelson et al., 2007), the proposed curriculum in causal reasoning will have wide applicability across clinical and counseling psychology, social work, psychiatry, marital and family therapy, and psychiatric nursing.

The causal reasoning curriculum should cover the following topics: (a) domains in which practitioners routinely make and evaluate causal inferences; (b) definitions of key concepts related to causality and errors in causal reasoning; (c) guidelines for evaluating the generalizability and applicability of causal findings in empirical studies to specific clients; (d) guidelines for formulating and evaluating working theories concerning the origin or maintenance of client presenting problems, especially as these pertain to the need for further assessment, case conceptualization, and treatment planning; and (e) methods for targeting and evaluating interventions. Recommendations for the content of training are presented below.

Module 1: Domains in Which Practitioners Routinely Make and Evaluate Causal Inferences

Module 1 should facilitate a thorough review and discussion of the important domains of clinical practice in which mental health practitioners are routinely required to make causal inferences. Discussion of these topics can be enhanced through the use of causal modeling diagrams (see below) and illustrative clinical case examples (Galanter & Jensen, 2009). Domains of clinical practice that require causal inference include (a) formulating a working theory of the (often) complex etiology and maintenance of a clients presenting problems, including causal agents and pathways of influence, and identifying factors that mediate or moderate the relation(s) between theorized causal agents and their effects; (b) formulating an assessment plan to further identify potential causal factors, as well as mediating and moderating factors, that are theorized to contribute to the clinical course; (c) making decisions about whether symptoms of a disorder cause clinically significant distress and/or impairment in behavior and functioning (a "causal coda" found near the end of many diagnostic criteria sets in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition); (d) formulating an intervention plan that specifies intervention objectives, intervention foci, and practice elements that, when implemented, are theorized to produce clinical improvement; (e) evaluating the effects of theorized mechanisms of therapeutic change on treatment response; (f) making decisions to modify treatment objectives and practice elements and to refer, alter, or terminate treatment; (g) evaluating the validity of causal inferences in empirical studies; (h) evaluating the generalizability of causal findings in empirical studies to specific clients; and (i) drawing and evaluating causal inferences that are based on one's own clinical experiences and the experiences of other professionals.

Module 2: Definitions of Key Concepts Related to Causality and Errors in Causal Reasoning

Module 2 should provide basic definitions of causal concepts. A useful way of framing the discussion is to present a fundamental causal model consisting of causal agents, pathways of influence, mechanisms of action, mediators, moderators, and causal consequences (Steinberg, Layne, & Steinberg, 2012). The concept of causality may be defined as a relation between two conditions or events in which one condition or event (the antecedent) determines (either wholly or in part) the occurrence or nonoccurrence of the other (the consequent). Given the pragmatic focus of the curriculum, we advocate the adoption of a simple approach to causality in contrast to discussions of the philosophical foundations of causality found in the works of Aristotle, Hume, Descartes, Kant, Mill, and contemporary philosophers of science (for reviews see Antonakis, Bendahan, Jacquart, & Lalive, 2010; Mellor, 1995; Pearl, 2010; Tooley, 1987). Such a pragmatic approach to causality centers on evaluating whether three classic conditions for the attribution that *X* causes *Y* are met:

1. *X* must temporally precede *Y* (or be simultaneous with *Y*).
2. *X* must be reliably correlated with *Y* (i.e., co-occur in a reliable manner beyond chance).
3. The relation between *X* and *Y* must not be explained by other causes.

Module 2 should also explicate key causal concepts that constitute the basic "building block" vocabulary that underlies a causal modeling approach. Table 1 presents definitions of key concepts (adapted and expanded from Jonassen & Ionas, 2008).

Thus, causal risk factors are of greater value than simple risk markers for planning and implementing preventive and clinical interventions because they serve as "red flag" markers of risk as well as candidate intervention targets and mechanisms of change.

Module 2 should also explicate common errors in causal reasoning and their possible consequences in clinical practice. Evidence for causal claims accrues by observing a positive or inverse correlation between two events. Although such findings often have heuristic value for generating causal hypotheses, they must be approached with the strong caveat that correlations can be explained in multiple ways. Figure 1 (adapted from Mulaik, 2009) is useful for depicting the "third variable problem": For any postulated causal relationship (*X* causes *Y*), two additional explanations should be considered; that is, *Y* causes *X* and *Z* causes *X* and *Y*.

Module 2 can also explicate common errors in causal reasoning (for a review, see Evans, 2008), including (a) a post hoc ergo propter hoc fallacy occurs when the fact that one event occurred before another is taken as sufficient evidence that the earlier event caused the subsequent event; (b) oversimplification or reductionism occurs when a complex situation is reduced to a single, simple cause; (c) asserting a wrong common cause occurs when two correlated, but noncausally related, events are erroneously attributed to the same cause; (d) appealing to anecdotal evidence constitutes insufficient grounds to infer that a causal relation exists; (e) appeal to authority occurs when a causal attribution is made simply based on a statement by an individual or entity with a reputation; (f) circularity in claiming a causal relation occurs when the statement of the cause simply restates the effect, typically as a defini-

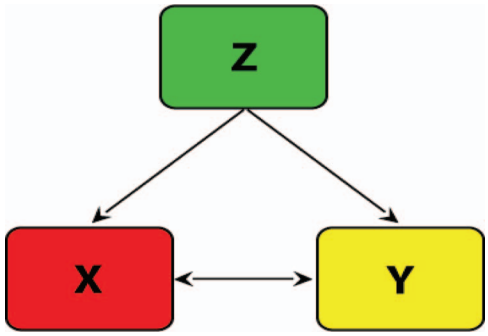

Figure 1. Depiction of the "third variable" problem.

Table 1
Definitions of Key "Building Block" Causal Concepts

- *A partial or contributory cause* plays a limited role in the cause/effect relation.
- *A necessary cause* is a causal agent without which the effect cannot occur.
- *A sufficient cause* is a causal agent the presence/occurrence of which ensures the effect.
- *A sole cause* is a causal agent that is necessary and sufficient for the effect to occur.
- *A conjunctive cause* is a causal agent that must be jointly present with one or more other causal agents for the effect to be produced.
- *A disjunctive cause* is a causal agent among several other causal agents, any of which may produce the same effect.
- *A reciprocal causal relation* occurs when two entities are causes and effects of one another.
- *A causal chain or multistep causation* occurs when there are several successive causal agents that collectively produce an effect.
- *A moderator* interacts with a causal agent such that the effect of the causal agent on a given outcome varies as a function of the moderator. Moderators can be classified as *not therapeutically modifiable* (e.g., age, sex) vs. *therapeutically modifiable* (e.g., social support, help-seeking attitudes).
- *A vulnerability factor* is a type of moderator the presence or level of which is associated with an exacerbation of the adverse effect(s) of a causal risk factor on an outcome.
- *A protective factor* is a type of moderator variable the presence or level of which is associated with attenuation of the adverse effect(s) of a causal risk factor on an outcome.
- *A facilitative factor* is a type of moderator the presence or (increasing) level of which is associated with an intensification of the beneficial effect(s) of a promotive factor on an outcome.
- *An inhibitory factor* is a type of moderator the presence or level which is associated with an attenuation of the beneficial effect(s) of a promotive factor on an outcome.
- *A mediator*: is a link (B) in a causal chain that transmits the effects of a prior cause (A) to a subsequent cause (C) such that A→B→C, in which B is an effect of A and a cause of C.
- *A fully mediated relationship* occurs when the effect of a causal agent is fully transmitted via the mediator.
- *A partially mediated relationship* occurs when the effect of a causal agent is transmitted by a direct pathway and via the mediator.
- A *risk marker* is a factor (a) the presence or level of which is associated with a substantial increase in the statistical likelihood of a given outcome or effect but (b) for which direct alteration of the risk marker does not necessarily alter the risk of the outcome and thus (c) is not an appropriate target for prevention or intervention. For example, elevations in blood plasma PSA levels are a marker of risk for prostate disease given that it is a byproduct of the body's immune response; however, developing a drug that causes blood plasma PSA levels to decrease will not also cause risk for prostate disease to decrease.
- A *causal risk factor* is a factor (a) that is causally related to a given outcome, (b) the presence or level of which is associated with an increased likelihood of a given outcome (thereby serving as a risk marker), and (c) for which direct alteration of the causal risk factor does alter the risk of the outcome (either increasing or decreasing risk, depending on the direction of alteration) and thus (d) may be an appropriate target for prevention or intervention. For example, elevation in blood plasma LDL levels is a causal risk factor for coronary artery disease; moreover, treatments (e.g., statin drugs) that cause blood plasma LDL levels to decrease also reduce risk for coronary artery disease.

Note. PSA = prostate-specific antigen; LDL, low-density lipoprotein.

tion or description, (e.g., the claim that only sufficient willpower can bring about behavior change); (g) lack of testability occurs when the postulated causal relation is impossible to verify or falsify, such as when postulating a causal agent for which the existence cannot be operationalized or established or when the statement of the cause is vague or uses undefined terms; and (h) a causal claim is compromised when it involves unnecessary assumptions and thus violates Occam's Razor or the Principle of Parsimony.

Module 3: Evaluating the Internal and External Validity of Findings in Empirical Studies

Many textbooks and undergraduate and graduate courses are available to assist in training mental health professionals to evaluate the internal validity (whether causal inferences in experimental designs are justified) and the general rigor of research designs, methods, and analytic strategies. Although they are essential to training evidence-based practitioners, we omit detailed discussion of these issues given space considerations. Greenhalgh (2001) presents a series of useful questions that can be used to build an attitude of healthy skepticism and teach trainees to systematically evaluate the legitimacy of causal inferences made in the literature.

Many training materials pertaining to internal validity unfortunately lack sufficient focus on external validity—a circumstance that impedes effective knowledge transfer from research to practice (Green & Glasgow, 2006). This deficiency in training can deprive practitioners of the full spectrum of reasoning skills needed for competent EBP given that interpreting, evaluating, and applying empirical findings derived from the literature to individual clients and to populations other than the one under study are essential and defining features of EBP. To facilitate the acquisition of skills needed to systematically evaluate the applicability of research evidence to circumstances and populations other than those in which the evidence was produced, we recommend that Module 4 review the RE-AIM framework (Glasgow, 2002; Glasgow, Vogt, & Boles, 1999; see also Cook & Campbell, 1979) that also includes questions that mental health practitioners can use to evaluate the generalizability of findings to specific clients.

Practitioners must also learn to evaluate potential cultural differences between their clients and research subjects. For example, interventions must be sensitive to ethnocultural issues as manifest in such forms as variability between cultures, family practices, conceptions of mental illness, stigma attributed to mental illness, and prior history with and expectations of the provider system (Barrio, 2000; Nezu, 2010). Issues for lesbian, gay, and bisexual clients (American Psychological Association, 2000) and older adults (American Psychological Association, 2004) are other client characteristics that should receive attention.

Module 4: Formulating and Evaluating Working Theories of the Origin and/or Maintenance of Client Presenting Problems

A first step in establishing and addressing the underlying causes of a client's presenting problem(s) is to clearly characterize the nature of the presenting problem(s) in ways that frame the problem(s) as a causal consequent. Imprecision in identifying and separating distinct causal consequences may undermine efforts to clarify causal agents, pathways of influence, mediators, and moderators. Such conceptual blurring can lead to serious "downstream" consequences, including impeding efforts to develop effective interventions for which the components precisely target key causal mechanisms that cause distress and dysfunction and to provide efficient "as-needed" services to specific subgroups at high risk because of their levels of exposures to specific causal risk and vulnerability factors and inadequate protective and promotive factors.

Figure 2 presents a causal model diagram that can be used to clarify and facilitate discussion regarding these complex issues. The diagram provides a framework to guide clinical assessment, case conceptualization, treatment planning, and the development of a working theory of the origin, maintenance, and remediation of the client's presenting problem.

The diagram focuses on a negative outcome that may be the target of intervention (e.g., posttraumatic stress disorder [PTSD], depression, or anxiety). It includes consideration of a focal causal risk factor (e.g., a traumatic experience, recent bereavement, harsh economic adversities). The plus sign (+) on the path leading from the causal risk factor to the negative outcome indicates that as the causal risk factor is present or increases in intensity, its effect on the negative outcome also increases (e.g., the more severe the trauma, the greater the severity of posttraumatic stress symptoms). The positive sign (+) on the path leading from the vulnerability

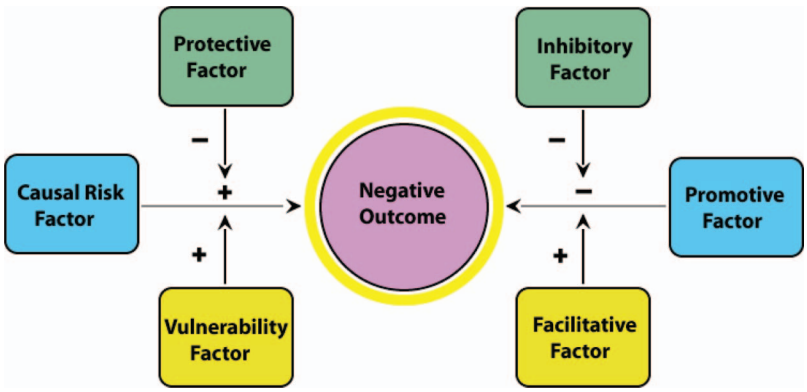

Figure 2. Causal model diagram of the presenting problem and its pathways of influence.

factor (e.g., age, gender, prior history of childhood trauma) to the path leading from the causal risk factor to the negative outcome reflects an interaction such that the presence or level of the vulnerability factor is associated with an intensification or exacerbation of the adverse effects of the causal risk factor on the negative outcome. The minus sign (–) on the path from the protective factor (e.g., self-esteem, mastery) to the path leading from the causal risk factor to the negative outcome reflects an interaction, such that the presence or increasing level of the protective factor is associated with mitigation or buffering of the adverse effects of the causal risk factor on the negative outcome.

In contrast, a promotive factor (e.g., good parenting, strong interpersonal attachments, and effective coping skills) exerts a direct beneficial effect (reflected by a minus sign [–]) on the negative outcome, such that it causally diminishes the negative outcome independent of the causal risk factor's effects. The plus sign (+) on the path from the facilitative factor (e.g., sex, age, prior history of quality parenting) to the path leading from the promotive factor to the negative outcome reflects an interaction such that the beneficial effects of the promotive factor on the negative outcome are further intensified when the facilitative factor is present or increases. Finally, the minus sign (–) on the path from the inhibitory factor (e.g., negative attitudes toward help-seeking, negative expectations) to the pathway leading from the promotive factor to the negative outcome also reflects an interaction such that the beneficial effect of the promotive factor in reducing the negative outcome is reduced when the inhibitory factor is present or increases. It should be noted that this diagram depicts linear causal relations that serve as fundamental building blocks for case conceptualization and treatment planning. These basic elements can be expanded to form more complex constellations of interacting, bidirectional, and interdependent effects that involve reciprocal causation. For example, in a transactional model, a child's development is the product of the continuous dynamic interactions of the child with the surrounding social ecology (Sameroff, 2009). It should also be noted that there may be considerable variability among elements of a causal model in the relative magnitude of causal influences as well as the specific pathways through which causal influences are exerted (e.g., direct, indirect, and total effect) as may be revealed by effect decomposition in path analysis or structural equation modeling. Evidence from the empirical literature may assist practitioners in weighing the relative contribution of multiple causes, mediators, and moderators and in formulating hypotheses concerning the most probable sources of continuing distress. These hypotheses can then be tested clinically at the individual case level.

Many wellness-oriented approaches, including positive psychology and positive youth development (e.g., Seligman, 1990), emphasize the promotion of positive outcomes as a primary goal of intervention. Figure 3 can promote this area of emphasis by serving as a tool to facilitate clinical consideration of causal mechanisms that lead to positive outcomes and how best to achieve them. Note that a shift in the valence of the primary target of intervention (i.e., shifting the focus of intervention from a negative to a positive outcome) results in the reversal of the valence of each path in the model (from + to – and – to +) and a similar reversal in the interpretation of relations among elements in the model (e.g., promotive factors exert direct beneficial effects on positive outcomes as indicated by + signs).

Given that Figures 2 and 3 focus on direct and moderated effects, an alternative model (see Figure 4) is useful for elaborating the causally intermediary role of a mediator. A mediator is an inherently causal concept given that a mediator acts as an intermediary link in a causal chain that connects a causal precursor to a causal consequence. In the simple example in Figure 4, the causal risk factor (e.g., trauma exposure) generates two causal consequences—secondary adversities and trauma reminders—each of which acts as a mediator that contributes to the negative outcome (e.g., PTSD). In this model, secondary adversities and trauma reminders mediate the link between trauma exposure and PTSD; further, the negative outcome (PTSD) mediates the link among the causal risk factor, secondary adversities, trauma reminders, the negative outcome, and functional impairment (e.g., poor school performance). Case conceptualization that "unpacks" complex ecologies into causal pathways hypothesized to lead to specific positive and negative outcomes can promote the identification of key causal factors, mediators, and moderators as well as negative and positive outcomes. Such efforts can significantly expand the range of "evidence-based" intervention targets. For example, assessment-driven modularized interventions (e.g., Macy et al., 2004) are tailored to target the reduction of causal risk, vulnerability, and/or inhibitory factors; to strengthen protective, promotive, and facilitative factors; to interrupt mediated pathways

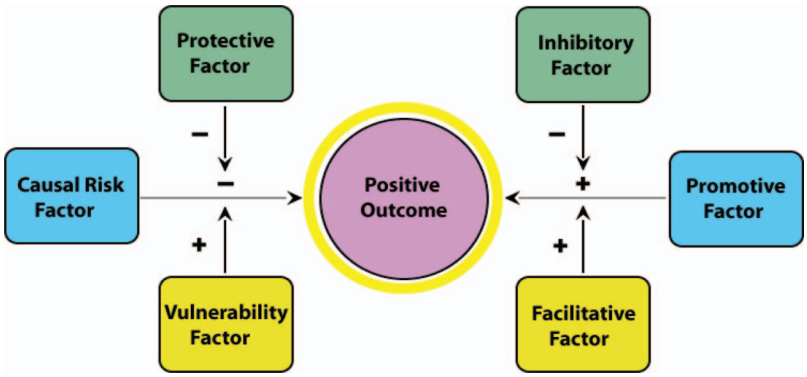

Figure 3. Causal model diagram of a positive outcome and its pathways of influence.

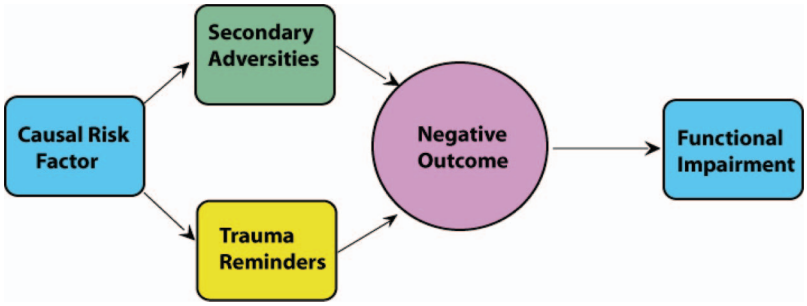

Figure 4. Causal model diagram of presenting problem and mediators.

leading to negative outcomes; and to facilitate mediated pathways leading to positive outcomes.

More broadly, such training tools hold the potential of helping to shift the mental health field from a psychopathology-centric paradigm, which emphasizes symptom-focused, diagnosis-centered clinical assessment, case conceptualization, and treatment planning (with negative outcomes serving as the primary focus of assessment and intervention), toward an ecopathology- and ecoresilience-focused paradigm. The latter perspective adopts a more balanced approach by integrating wellness- and strength-based approaches into these activities, with positive outcomes, beneficial resources, and resource gain cycles serving as equally important foci of assessment and intervention.

Module 4 should include experiential exercises in which learners use the conceptual models as tools to organize and convert "raw data" scenarios concerning clients' problems, strengths, and circumstances into a therapeutically actionable working theory within a broader ecological context. For example, clinical casebooks (e.g., Galanter & Jensen, 2009) can be used to develop case conceptualization skills by training learners to adopt the perspective of each component (e.g., causal risk, promotive, and vulnerability factors as well as mediators/moderators) as a method for sorting through, organizing, and integrating different types of information.

Module 5: Planning, Targeting, and Evaluating Interventions

The focus of Module 5 is to help trainees learn to apply the causal model diagrams from Module 4 to select, implement, monitor, and evaluate the implementation of evidence-based interventions with specific clients. Module 5 should begin with a discussion of the importance of delineating and sequencing clear intervention objectives), identifying associated intervention foci, and selecting specific practice elements by which to achieve those objectives. Training exercises should elucidate the logical connections that interlink various levels of intervention, including the empirical evidence base, theoretical and conceptual foundations, intervention objectives, intervention foci, practice elements, and appropriate assessment instruments to monitor the course of therapeutic progress. (Layne et al., 2011). In learning to judge which specific intervention is most likely to be effective in producing a desired outcome for a specific client, trainees can evaluate not only the empirical evidence that justifies the selection of specific elements of the ecology as targets for intervention but (also) the evidence that specific practice elements cause changes in the targeted foci in accordance with intervention objectives. It is also helpful to emphasize that a manualized treatment protocol is a complex amalgam of an undergirding conceptual framework (which typically contains many causal assumptions) and many intervention objectives and practice elements that have been carefully selected, prioritized, and sequenced to therapeutically address the needs of specific populations (Chorpita, Bernstein, & Daleiden, 2011; Weisz et al., 2012).

Figure 5 presents a causal model diagram that depicts potential targets of intervention, including causal precursors, moderators, mediators, consequences, and pathways of influence that interconnect them. Each element constitutes candidate foci for intervention.

This diagram uses information gathered in completing the diagrams from Module 4 to invite systematic consideration of the broad range of potential intervention foci available for treatment and the armamentarium of specific evidence-based interventions (or practice elements thereof) that may be strategically mobilized. For example, in performing the tasks of case formulation, treatment planning, and treatment monitoring, a therapist may (a) identify a focal problem, its consequence, or one or more causal risk factors to be therapeutically addressed; (b) identify mediators and moderators that, if targeted, are hypothesized to produce improvements in the focal problem, its consequence(s), or its causal risk factor(s); and (c) prioritize, target, implement, and monitor the outcomes of specific therapeutic strategies. This model promotes efforts to match specific intervention foci with specific intervention objectives and interventions. Interventions can consist of evidence-based treatment protocols and specific practice elements that include psychoeducation, cognitive restructuring, relaxation training, guided imagery, modeling, problem-solving and coping skills, positive activity scheduling, assertiveness training, relationship building, and relapse prevention (Chorpita, Becker, & Daleiden, 2007; Layne et al., in press).

Trainees should understand that in designing and populating causal model diagrams, they are generating causal hypotheses (e.g., concerning the origin and maintenance of problems on one hand and the potential effectiveness of practice elements to address them on the other), designing causal experiments (i.e., implementing interventions and practice elements during treatment), and evaluating outcomes by monitoring client response to treatment. In performing the tasks of case formulation, treatment planning, and treatment monitoring, a therapist may (a) identify a focal problem,

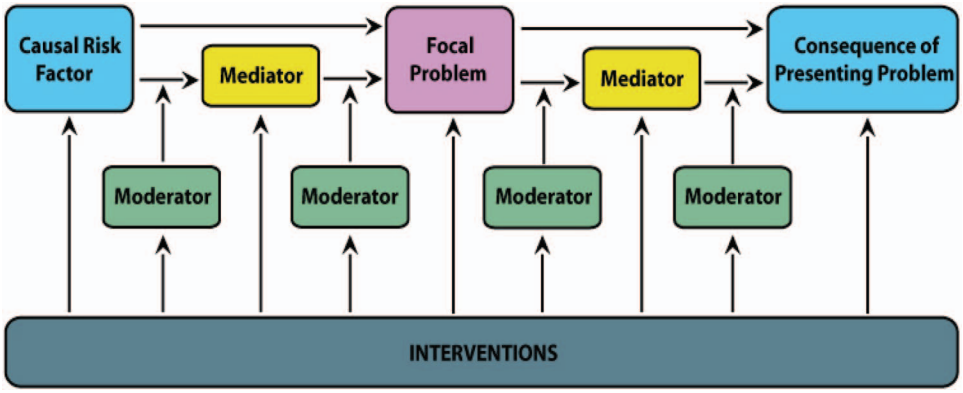

Figure 5. Causal model diagram of targets of intervention and pathways of influence.

its consequence, or one or more causal risk factors that are to be therapeutically addressed; (b) identify mediators and "modifiable" moderators that are hypothesized to produce improvements in the focal problem(s), its consequence(s), or its causal risk factor(s); and (c) prioritize, target, implement, and monitor the outcomes of specific therapeutic strategies.

As an illustrative example, trainees can participate in a "double-check" exercise in which they apply a heuristic for systematically considering a broad range of negative and positive potential outcomes as well as socioenvironmental factors theorized to play a role in their origin, maintenance, remission, exacerbation, or consequences (see Figure 6, a and b).

The objective of this exercise is to teach trainees how to use available information to begin building a clinically useful theory that can, in turn, guide further clinical assessment, case conceptualization, treatment planning, and treatment monitoring activities; in turn, these activities will produce additional information that is iteratively used to test, refine, and further elaborate on the theory throughout treatment. More broadly, this exercise can help trainees to consider a range of negative and positive potential outcomes within the context of a surrounding matrix of factors theorized to serve as causal precursors, mediators, and moderators. The exercise can commence at different entry points, such as by "checking for negative factors" by identifying possible negative outcomes (e.g., depression, PTSD) and inserting this outcome in the center circle of Figure 6a. Trainees can then conceptually "populate" remaining domains of the model by systematically adopting the vantage point of each domain and identifying specific factors hypothesized to operate in that capacity. These include protective factors (e.g., supportive extended family), vulnerability factors (e.g., prior trauma), promotive factors (e.g., quality parenting), inhibitory factors (e.g., negative attitudes toward help-seeking), and facilitative factors (e.g., living near extended family). A second "causal precursor-centric" strategy involves identifying causal risk factors (e.g., traumatic bereavement, poverty), inserting this theorized etiologic agent into the left-hand box of Figure 6a, and then forming hypotheses concerning their potential sequelae (e.g., grief, PTSD, depression, disruptive behavior). Trainees can apply a similar "outcome"- or "causal precursor"-centric approach to positive factors in 6b.

This double-check heuristic has six advantages. First, adopting an outcome-centric strategy invites trainees to apply the developmental psychopathology concept of equifinality by developing hypotheses regarding how multiple factors may contribute, either alone or in combination, to a given outcome. Second, adopting a "causal precursor-centric" strategy helps trainees apply the concept of multifinality by developing hypotheses regarding how a focal causal risk or promotive factor may lead to a range of different outcomes (often through different pathways, as reflected by different configurations of moderators and mediators). Third, trainees can apply the concept of differential validity by observing that different elements of the model are meaningfully distinct because they relate to other variables in dissimilar ways and carry different implications for assessment and intervention. For example, different causal risk factors (e.g., traumatic bereavement vs. poverty) may work through different causal pathways and produce different outcomes; conversely, different outcomes (e.g., PTSD vs. grief) are meaningfully distinct because they are produced through different pathways and call for different interventions. Fourth, helping trainees identify underpopulated domains in the model helps them to specify "next steps" by identifying areas in need of further assessment. Fifth, this heuristic encourages trainees to adopt a balanced approach by systematically considering a broad range of positive and negative influences and outcomes. Last, this method illustrates the dynamic connections among assessment, treatment planning, and treatment implementation and how each activity informs the others and helps practitioners evaluate progress throughout treatment.

This exercise can segue into active discussion and exercises that focus on how to strategically consider the various diagrammatic elements to develop a treatment plan. This approach to mapping the broader physical and social ecologies that encompass and differentially contribute to the presenting problem(s) widens the window of potential intervention foci well beyond those typically considered using a traditional symptom- or problem-focused approach. It also lays the groundwork for ecologically focused interventions, including those that target multiple systems (e.g., Kazak et al., 2010; Macy et al., 2004; Swenson, Schaeffer, Henggeller, Faldowski, & Mayhew, 2010).

In addition to considering which intervention objectives, intervention foci, and practice elements are most likely to be effective in producing desired outcomes, consideration should be given to maximizing intervention efficiency. For example,

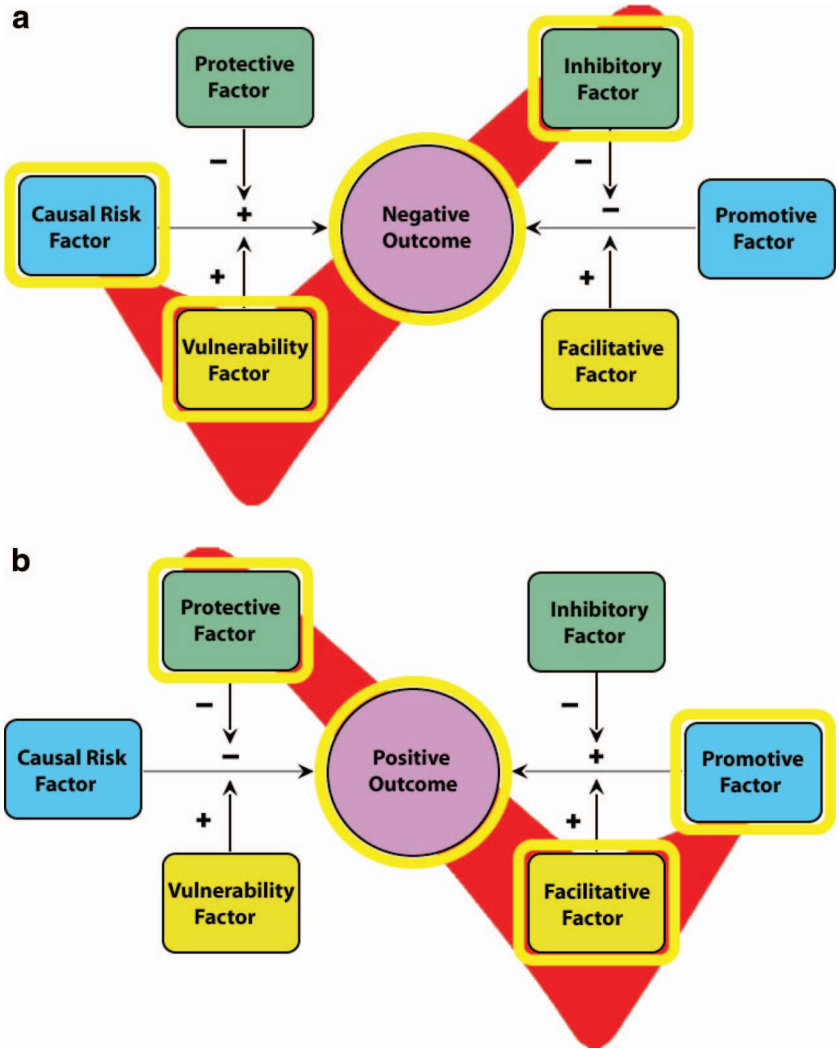

Figure 6. Double-check heuristic for evaluating beneficial and adverse factors.

enhancing promotive factors is often more desirable than buttressing protective factors because promotive factors produce beneficial effects regardless of whether a focal causal risk factor is present. Further, synergistic effects (e.g., therapeutically targeting promotive factors, enhancing facilitative factors, and reducing inhibitory factors) are often more desirable than simple additive effects given that their outcomes are greater than the sum of the individual effects.

Because randomized controlled trials of treatment protocols, which are complex amalgams of many different causal assumptions, intervention objectives, and practice elements, now constitute the "gold standard" making up the current evidence base, comparatively little evidence is now available to address the question of which specific practice element within specific treatment protocols is effective in achieving a specific objective within a given context (Kazdin, 2006). This constitutes a call for randomized controlled trials that "unpack" or "dismantle" treatments into practice elements to determine specific factors that contribute to therapeutic change. It also points to a limitation in treatment decision-making in regard to which set of practice elements is likely to produce a given set of outcomes.

Given these limitations in the empirical evidence base, the following rationally derived guidelines (adapted from Berkowitz et al., 2010) can, in conjunction with the causal model diagram above, guide practitioner decision-making in selecting and prioritizing intervention objectives, foci, and practice elements:

1. The client identifies the problem as a source of significant distress or impairment.
2. The problem needs to be addressed sooner than other problems.
3. The problem is worsening considerably over time.
4. Addressing the problem will reduce other problems.
5. The client prefers help with this problem first.
6. The client feels that he or she is motivated to work on this problem.

In keeping with the tenets of EBP, practitioners may share their professional observations and recommendations with clients to jointly decide on intervention objectives, foci, and practice ele-

ments. Mental health practitioners should be aware that intervention practice elements can exert a range of potential effects, including effective, efficient, robust, circumscribed, prophylactic, weak, inert, and iatrogenic effects (Dimidjian & Hollon, 2010; Layne et al., 2009).

Conclusion

We have provided recommendations for the modules of a curriculum for training in causal reasoning skills as a complement to training in EBP. Similar to a case-based curriculum to strengthen clinical knowledge and clinical judgment concerning childhood trauma (Layne et al., in press), this causal reasoning skills training curriculum has wide application, ranging from beginning doctoral trainees in professional psychology to in-service training of practicing clinicians. In particular, this curriculum can be used as a foundation for developing prepracticum competencies in beginning trainees, but it can also be flexibly tailored for advanced students or for groups in which the members vary in their knowledge and skills. Potential settings include didactic classroom presentations as well as more interactive small-group, externship, internship, and postdoctoral training programs as delivered in individual or group supervision.

Of particular relevance to the theme of the special section, descriptions of core competencies necessary for prepracticum training in professional psychology have included such skills as critical scientific thinking, problem-solving, organized reasoning, application of scientific method to practice, case conceptualization, intervention planning, and evaluating the efficacy of interventions (Fouad et al., 2009; Hatcher & Lassiter, 2007; Hatcher et al., 2012; Kaslow et al., 2009; Pidano & Whitcomb, 2012). As noted above, the proposed curriculum in causal reasoning operationalizes these skill sets in ways that are amenable to formulation of specific learning objectives and evaluation of specific clinical competencies.

Causal reasoning skills training is one of various possible complements to training in EBP among such alternatives as training in core affective, cognitive, and behavioral skills needed to enhance empathic therapeutic interactions, self-correct, surmount obstacles, leverage opportunities, and address therapeutic biases (Kahneman, 2003). Supports for causal learning incorporated into this curriculum include the use of causal model diagrams to promote understanding of complex causal relations (Glickauf-Huges, 1991; Jonassen & Ionas, 2008) and the use of problem-based learning methods (Layne et al., in press).

Notwithstanding the rationale we have provided for its adoption, little evidence is currently available concerning ways in which causal reasoning skills training may enhance clinical practice. As noted by Jonassen and Ionas (2008), causal reasoning skills training is in its formative state and will require the development and use of innovative research methods to build a substantial evidence base. As a first step, research on the potential benefit of training in causal reasoning should include competency- and skill-based evaluation of trainees' deep conceptual learning, knowledge retention, and the application of causal reasoning skills to novel cases and circumstances. Value-added studies could then compare case conceptualization, use of evidence-based assessment tools, judicious identification of intervention foci, treatment planning and implementation monitoring treatment effects, and client outcomes among evidence-based practitioners trained in causal reasoning skills compared practitioners without such training.

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