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Leveraging the developmental science of psychosocial risk to strengthen youth psychotherapy

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Abstract

More than 50 years of randomized clinical trials for youth psychotherapies have resulted in moderate effect sizes for treatments targeting the most common mental health problems in children and adolescents (i.e., anxiety, depression, conduct problems, and attention disorders). Despite having psychotherapies that are effective for many children, there has been a dearth of progress in identifying the contextual factors that likely influence who will respond to a given psychotherapy, and under what conditions. The developmental psychopathology evidence base consistently demonstrates that psychosocial risk exposures (e.g., childhood adversities, interpersonal stressors, family dysfunction) significantly influence the onset and course of youth psychopathology. However, the developmental psychopathology framework remains to be well integrated into treatment development and psychotherapy research. We argue that advances in basic developmental psychopathology research carry promising implications for the design and content of youth psychotherapies. Research probing the effects of psychosocial risks on youth development can enrich efforts to identify contextual factors in psychotherapy effectiveness and to personalize treatment. In this article we review empirically supported and hypothesized influences of individual- and family-level risk factors on youth psychotherapy outcomes, and we propose a framework for leveraging developmental psychopathology to strengthen psychotherapies.

Keywords: child, childhood adversity, psychopathology, psychotherapy outcomes, risk factors

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The developmental psychopathology framework has inspired and informed more than three decades of scientific advances in the identification of psychosocial risk factors for mental health problems and disorders in children and adolescents (herein, youths). Pathways from psychosocial risk exposure to common youth psychopathologies are marked by disruptions in developmental processes (e.g., cognitive and emotion regulatory systems, stress response systems; Boyce & Ellis, 2005; Cicchetti & Rogosch, 2002; McLaughlin, Sheridan, & Lambert, 2014; Shonkoff et al., 2012) that shape current conceptualizations of etiology, course, and long-term functional and behavioral outcomes during youth development. Nearly two decades ago, Cicchetti and Hinshaw (2002) noted that the promise of developmental psychopathology models for translating advances in basic developmental science research to applied intervention science strategies had not been fully realized. To date, this remains true. Growth and innovations for developing and testing evidence-based psychotherapies (EBPs) have not yet included widespread adoption of developmental science evidence to identify target change mechanisms or evaluate intervention effectiveness.

A great deal of progress has been made, across the decades, in developing, documenting, and testing youth psychotherapies. That said, the tests have revealed a need to refine and improve these therapies. A recent meta-analysis showed that 50 years of randomized controlled trials (RCTs) have not resulted in significant improvement in the effectiveness of youth psychotherapies on average (as indicated by study effect sizes; see Weisz et al., 2019) and another recent analysis suggested that psychotherapies *as currently designed* face a rather modest upper limit to the effect sizes that can be achieved (Jones, Mair, Kuppens, & Weisz, 2019). Furthermore, effect sizes for psychotherapies – including EBPs – remain particularly modest for prevalent and costly disorders like depression (Eckshtain et al., 2020). The current state of psychotherapy research also leaves several crucial gaps in knowledge – including "for whom" a given treatment is most effective, "which treatment will work for which individual," and "under what circumstances" a specific treatment is likely to be optimal. To fill these and other scientific gaps, more work is needed to identify how the effectiveness of common EBPs such as cognitive behavioral therapy (CBT), interpersonal therapy (IPT), dialectical behavior therapy (DBT), and parent training programs is influenced by young people's exposure to various psychosocial risk factors and associated developmental disruptions underlying the emotional, behavioral, and cognitive self-regulatory processes that are central targets of these EBPs (Weisz, 2015).

One potential challenge to optimizing EBPs for internalizing (e.g., depression and anxiety) and externalizing (e.g., conduct problems and attention-deficit/hyperactivity disorder [ADHD]) psychopathologies is that prior RCTs have not routinely included or accounted for potential influences of developmental

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disruptions and psychosocial risk factors on treatment outcomes. Significant "third variable" problems may arise in the evaluation of EBP effectiveness in the event that core deficits targeted via an EBP (e.g., impaired executive function and attention processes underlying problem-solving deficits and negative cognitive biases in depression) are attributed solely to the psychopathology (Wagner, Müller, Helmreich, Huss, & Tadić, 2015). If an EBP is believed to target cognitive dysfunction attributed to a psychiatric disorder (e.g., biased attention to social threat) but the dysfunction is moderated by unmeasured psychosocial risk exposure, important explanations for disparate treatment outcomes across individuals may be missed. Identifying meaningful subgroups for whom certain treatments may be more or less effective is not only an important scientific objective, but a federal priority for research in the USA (National Institute of Mental Health, 2015).

A second challenge in the integration of developmental and intervention sciences is the complexity that arises from the blend of equifinality and multifinality in developmental pathways from psychosocial risk to youth-onset psychopathology (Cicchetti & Rogosch, 1996). Equifinality is the process by which multiple developmental pathways lead to a common outcome (e.g., multiple paths to adolescent depression; Hankin, 2015), whereas multifinality is the process by which an initial risk exposure may take multiple pathways leading to a variety of outcomes (e.g., any combination of mood, anxiety, or conduct behaviors, dysregulation in biological stress response systems, or disrupted cognitive development; Cicchetti & Toth, 2005; Doom & Cicchetti, 2020). Equifinality and multifinality complicate the search for clear connections between early risk exposure and later psychopathology, including efforts to identify and test predictors, moderators, and mediators of treatment outcomes. This is a significant challenge faced by any effort to build a developmentally-informed intervention science.

Despite these challenges, there have been notable efforts to integrate developmental and intervention sciences. Programs such as Head Start, a public preschool intervention for children from under-resourced settings, were designed to address several interconnected domains of risk (e.g., material resources, social competencies, parental factors) in order to enhance children's cognitive development and academic performance (Gilliam & Zigler, 2000). More recently, the Attachment and Biobehavioral Catchup (ABC) program (Dozier et al., 2006) for infants and toddlers at risk of maltreatment provides caregivers with skills to reinterpret and respond adaptively to children's regulatory signals, build secure attachment, and improve children's biobehavioral regulation. RCTs testing the ABC program have shown normalization of diurnal cortisol rhythms post-intervention (compared to a control intervention) that was maintained over time (e.g., Bernard, Hostinar, & Dozier, 2015). Perhaps the multi-risk approach used in these interventions to enhance outcomes can be adapted and applied to youth psychotherapies when psychopathology emerges in later childhood and adolescence. The full effects of disruptions to development may take years to manifest (Shonkoff et al., 2012).

Building on such prominent early intervention programs as Head Start and the ABC program, investigators and treatment developers may leverage the multi-risk framework to inform hypotheses regarding the levels of analysis at which to expect a change in developmental processes following treatment. For example, research on the development and plasticity of cognitive systems (e.g., attention, language, and working memory; Fortenbaugh et al., 2015; Peverill, McLaughlin, Finn, & Sheridan, 2016) can be leveraged to evaluate whether the use of CBT skills targeting cognitive self-regulation (e.g., problem solving, cognitive restructuring) could reasonably exert change in these underlying basic cognitive systems or whether effects will be limited to observed behaviors. This approach would draw upon the developmental psychopathology principal of developmental cascades, recognizing that a disruption in development may manifest across multiple levels of analysis (behavioral, cognitive, neural, etc.) in relation to a single form of psychopathology (Doom & Cicchetti, 2020). In other words, developmental science is key for identifying the levels of analysis at which risk factors may be most likely to impact treatment outcomes (e.g., behavior, cognition, physiology), and for appropriately operationalizing those levels of analysis in the evaluation of EBP effectiveness.

Forging a path toward an integrated intervention science and strengthened youth psychotherapies will require an understanding of the impact of psychosocial risk exposure and associated developmental disruptions on treatment effectiveness. In this article we describe a cluster of robust psychosocial risk factors for youth psychopathologies, grounded in a developmental psychopathology perspective, to (a) note empirically supported and hypothesized influences of these risk factors on youth psychotherapy outcomes and (b) propose conceptual and empirical approaches to strengthening EBPs for common youth psychopathologies. We have appreciated rich and thoughtful reviews on the implications of developmental psychopathology theories for preventive interventions by leaders in our field (e.g., Cicchetti & Gunnar, 2008; Cicchetti & Hinshaw, 2002; Masten, Long, Kuo, McCormick, & Desjardins, 2009). The present article builds on evidence that early developmental disruptions linked with psychosocial risk may not emerge as psychopathology, requiring treatment, until later in development. We focus on what can be done after psychopathology has developed and thus our review addresses treatment rather than prevention. The risk factors we address are both individual- and family-level constructs known to shape youths' environmental contexts in ways that magnify adversity and stress. We consider each risk factor at both psychosocial and biobehavioral levels of analysis, reflecting their association with common cognitive and behavioral treatment targets in EBPs.

Individual-Level Risk

Childhood adversities

Research on the etiology, course, and outcomes of youth psychopathology has revealed that childhood adversities show transdiagnostic associations with psychiatric disorders in youths; further, nearly 60% of youths in the USA are exposed to some form of adversity (McLaughlin et al., 2012). Childhood adversities are defined broadly as experiences "that are likely to require significant adaptation by an average child and that represent a deviation from the expectable environment" (McLaughlin, DeCross, Jovanovic, & Tottenham, 2019, p. 101). The aforementioned prevalence estimates suggest that exposures within this set of individual-level risk factors are quite common in the aggregate.

Childhood maltreatment

The experience of childhood maltreatment (defined as physical, sexual, or emotional abuse, or neglect) is a potent risk factor for youth-onset internalizing and externalizing psychopathology – and one that is not static across development. It has long been recognized that different forms of maltreatment may have

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varying impacts on youth outcomes at different points in development (Aber & Zigler, 1981). As an example, Aber and Zigler (1981) note that exposure to neglect may have a stronger impact on youths in early childhood, whereas sexual abuse may have a similar level of negative impact through adolescence. Aber and Zigler also note that developmental changes in the family environment contribute to "flux" in risk of exposure to maltreatment across youth development. Further, meta-analytic research suggests that specific forms of maltreatment (e.g., emotional abuse) are particularly salient risk factors for specific forms of psychopathology, such as depression (LeMoult et al., 2019). As a multifinal risk, it is not surprising that maltreatment has been shown to result in a host of varied outcomes across youth development (Cicchetti & Toth, 2005; Maughan & Cicchetti, 2002; Rodman, Jenness, Weissman, Pine, & McLaughlin, 2019). For example, maltreatment is associated with greater modulation in the prefrontal cortices and amygdala when engaging in cognitive reappraisal skills (Rodman et al., 2019), and maltreatment is also linked with disruptions in emotion and fear learning. Youths with a history of abuse show more difficulty discriminating between threat and safety cues and exhibit blunted sympathetic nervous system activity in the presence of a threat (McLaughlin, Rith-Narjarian, Dirks, & Sheridan, 2015).

As another example illustrating the range of sequelae that follow maltreatment, developmental disruptions are also evident in numerous associative learning processes (McLaughlin & Sheridan, 2016). Associative learning broadly guides youths' understanding and interpretation of input from their environments, shaping learned emotional, cognitive, and behavioral responses. Youths exposed to severe neglect in early childhood exhibit decreased reward responsiveness, which has been shown to mediate links between neglect and adolescent depression (Sheridan et al., 2018). History of child abuse has also been linked to deficits in reward sensitivity or detection of the changing value of rewards in the environment (Hanson et al., 2017). Maltreatment is further associated with difficulty reversing or updating learning when contingencies change (i.e., less flexibility in learning) (Harms, Shannon Bowen, Hanson, & Pollak, 2018). Disruptions in emotion learning and associative learning are relevant to youth psychotherapies because the skills taught in EBPs target some of these same learning processes.

Broad exposures to threat and deprivation

There are many other forms of adversity that pose a threat of harm, or deprivation of expected social and cognitive input from the environment, and thus also heighten risks for youth psychopathology (McLaughlin & Sheridan, 2016). A parsimonious model for organizing a broad range of childhood adversities is the Dimensional Model of Adversity and Psychopathology (McLaughlin et al., 2014), which delineates adverse experiences along axes of threat and deprivation. Exposures to threat (in addition to abuse) include experiences such as community violence and peer victimization, while exposure to deprivation (in addition to neglect) includes lack of provision of basic material needs or social and cognitive stimulation in home, community, or school environments (commonly associated with living in poverty). Both threat and deprivation are associated with reductions in cognitive flexibility, disruptions in executive function, altered reward processing, and disruptions in other higher order learning processes (e.g., Danese et al., 2017; Dennison et al., 2019). These adversity-related disruptions in cognition overlap notably with hallmark cognitive impairments in youth depression (Wagner et al., 2015) and attention and behavior problems (Arnsten & Rubia, 2012). The significance of adversity-related developmental disruptions in these cognitive domains lies in their central role in self-regulatory processes. In the context of treatment, these processes underlie skills to monitor cognitions, disrupt rumination, effectively problem solve, and select and use adaptive emotion regulation skills.

There is evidence that threat exposures such as community violence (e.g., witnessing the threat of or actual violent acts, or being personally victimized) during adolescence are indirectly associated with internalizing symptoms via a pathway of acute emotional reactivity to stress and emotion dysregulation (e.g., poorer problem solving, greater rumination) (Heleniak, King, Monahan, & McLaughlin, 2018). Further, correlations between more frequent threat exposure (community violence combined with peer victimization) and higher depression symptom severity are amplified by lower resting-state parasympathetic nervous system activity – indexed by respiratory sinus arrhythmia (RSA), an established proxy for emotion regulation capability (Beauchaine, 2015) – in younger and older adolescents (McLaughlin et al., 2015; Vaughn-Coaxum, Dhawan, Sheridan, Hart, & Weisz, 2020). These patterns are consistent with models that hold emotion dysregulation to be a core transdiagnostic feature underlying psychopathology (Lahey, Krueger, Rathouz, Waldman, & Zald, 2017)

Although threat and deprivation are both associated with similar cognitive disruptions, material deprivation is believed to exert a particularly robust effect on the *development* of cognitive processes beginning in early childhood (McLaughlin et al., 2014). There is evidence that the impact of deprivation, but not threat, on early childhood language development can have downstream effects on risk for psychopathology in late adolescence via a pathway of lower verbal abilities (Wade et al., 2017). Even recent deprivation (food insecurity in the last 12 months) is associated with disrupted cognition (e.g., reduced ability to attend to reward cues) (Dennison et al., 2019). Together, studies suggest the impact of deprivation on youths' cognitive functions across youth development may portend risk for psychopathology downstream.

Although the distinction between threat and deprivation is empirically supported and advantageous for identifying unique mechanisms that link different forms of adversity exposure with youth psychopathology, threat and deprivation co-occur at very high rates (McLaughlin et al., 2014). In some cases, multi-risk exposures reflect an alternative, cumulative risk model, where the accumulation of multiple adversity exposures – regardless of type – incrementally predicts greater risk of psychopathology (for a review, see Evans, Li, & Whipple, 2013). In a nationally representative sample of adolescents, higher numbers of adverse childhood events (but not different types) were associated incrementally with youths' reports of using less adaptive coping skills in response to stress (Vaughn-Coaxum, Wang, Kiely, Weisz, & Dunn, 2018). Furthermore, research from a 15-year prospective study indicates that low emotion awareness in middle childhood mediates associations between higher accumulated early life adversities and the severity of depression in adolescence (Luby, Barch, Whalen, Tillman, & Belden, 2017).

Implications for youth psychotherapy

Although childhood adversities show transdiagnostic associations with risk for psychopathology, meta-analyses specifically identify an association between material deprivation (lower socioeconomic status [SES]) and poorer outcomes of parent training interventions for externalizing behaviors (Leijten, Raaijmakers, de

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Castro, & Matthys, 2013; Reyno & McGrath, 2006). Lower SES is also correlated with disruptions in cognitive functioning. If cognitive deficits associated with depression, ADHD, and disruptive behavior pathologies are compounded by developmental disruptions following exposure to deprivation, it is important to consider how deprived environments may undermine efforts to improve self-regulatory function in areas that rely on those cognitive processes.

While SES moderates psychotherapy outcomes for externalizing problems, childhood maltreatment exposure is most robustly associated with psychotherapy outcomes in depression. Physical and sexual abuse have been linked with poorer response to CBT for depression in multiple randomized efficacy trials, with response rates ranging from 18% to 50% (Treatment of Adolescents with Depression Study, Lewis et al., 2010; Treatment of Resistant Depression in Adolescents, Shamseddeen et al., 2011). More broadly, childhood maltreatment has been associated with smaller effect sizes for psychotherapy, with poorer response rates persisting into adulthood (Nanni, Uher, & Danese, 2012).

The identification of a link between childhood adversities and associative learning processes (fear learning, emotion learning, reward learning, and reversal learning) is a development that has not yet been translated beyond basic science paradigms. However, these findings hold potentially great value for intervention research. The theoretical underpinnings of most skills-based psychotherapies (e.g., CBT, IPT, parent training) are based in learning theories (Harvey et al., 2014). Specifically, youths and/or parents are taught skills to target emotional, behavioral, and cognitive processes relevant to their target psychiatric problems. In the case of depression, this includes skills to manage hallmark symptoms like cognitive distortions, anhedonic/social withdrawal behaviors, anxious distress, and dysregulated emotional responses. Individuals are then expected to associate the use of the therapy skills with observable change (e.g., fewer depressive cognitions, increased motivation and reward seeking, more adaptive emotional responses, etc.) supported by self-monitoring and individual and parental reinforcement of skill use.

A hallmark of skills-based therapies is the expectation that individuals will practice the skills to gain competence and generalize the skills outside of the therapy context in response to novel problems as they arise in the real world. At a basic level, these processes require youths to build associations between skill use and outcomes, and to flexibly update or adapt learned associations as they apply skills in response to novel problems. Depressive pathology has been linked with disruptions in reward learning and reversal learning (the ability to flexibly update learned associations between a stimulus and response when the contingencies of the association change), particularly among adolescents (Dickstein et al., 2010; Morgan, Olino, McMakin, Ryan, & Forbes, 2013). These disruptions are also associated with adversities such as maltreatment (Goff & Tottenham, 2015; Harms et al., 2018). However, more research is needed to determine whether adversity amplifies or maintains learning disruptions in the context of depression, and if these disruptions inflate risk for poorer treatment outcomes.

Connections among associative learning, childhood adversity, and psychopathologies such as depression suggest several practical applications for intervention research. Slower learning trajectories and reduced flexibility in learning among youths with maltreatment history (Hanson et al., 2017; Harms et al., 2018) could translate to a need to modify the delivery of EBP skills. Adversity-exposed youths may benefit from more direct teaching and repetition from their therapist, or more intensive home practice of EBP skills to learn how to select and use different skills in real time and in novel situations. These youths may also benefit from more sessions and/or a more moderate pace at which skills are introduced in order to acquire the skills, build competency in them, and learn to detect associations between skill use and expected outcomes. A novel protocol developed to enhance learning in cognitive therapy for depressed adults is designed to equip therapists with skills to strengthen the reinforcement of skill acquisition and memory of therapy skills (Harvey et al., 2016). Adapting this adjunctive CBT component for youths could be a useful future direction in cases where learning disruptions are found to impede therapy gains.

Alternatively, the effects of maltreatment and other adversities on learning processes may facilitate identification of more precise treatment matching strategies. McLaughlin et al. (2019) propose that targeting learning processes with specific EBP skills matched to specific adversity-related learning disruptions may lead to transdiagnostic improvements in youth outcomes. For example, the authors propose that behavioral activation strategies may be robust in the context of adversities linked with disrupted reward processing (e.g., maltreatment and poverty). Behavioral activation – a common component of many CBT protocols and a standalone treatment for depression – focuses on enhancing experiences of reward and pleasure via reinforcement of engagement in meaningful activities. Importantly, little intervention research has focused on the acquisition or generalization of therapy skills in youth EBPs. This leaves both competing approaches described here as potentially useful future directions in the translation of developmental psychopathology research to intervention science.

For any future research aimed at pursuing the proposed or alternate hypotheses, careful consideration should be given to the framing and interpretation of adversity-related disruptions in learning and other developmental processes. Nearly two decades ago, a cautionary tale was published on what was, in the 1960s, a popular approach to early childhood intervention: the "deficits model." The deficits model aimed to close the achievement gap between lower and middle income youths, but ultimately signaled to the public that delays or disruptions in cognitive and social domains – associated with economic deprivation – were based on an "inferiority–superiority" ideology (Zigler & Berman, 1983). There was a subsequent shift to "differences" rather than "deficits" terminology, with the aim of building on the strengths that youths brought to early childhood interventions. However, Zigler and Berman (1983) argued that "difference" models still favored norms associated with higher economic classes. Similar concerns arise in relation to youth mental health treatment research.

The ways in which observed "deficits" in "normative" developmental trajectories are understood, framed, and communicated have implications for the work of clinicians and scientists, and shape perceptions among the general public and policy makers. The evolutionary–developmental theory of Boyce and Ellis (2005) provides important considerations for interpreting individual differences in developmental processes, highlighting numerous ways that deviations from *expected* norms may capture environmental *adaptations* that are actually advantageous in certain circumstances. As an example, one study of associative learning cited earlier suggested that observed disruptions in reward-based learning among maltreated youths may represent an adaptive pattern of information processing in certain high-adversity environments where rewards are not very predictable (Hanson et al., 2017). Great care should be taken in the study of

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adversity-related influences on treatment processes and outcomes, and in the design of personalizable interventions, to recognize cases in which apparent deficits represent appropriate adaptations.

Interpersonal stressors

Distinct from severe childhood adversities, interpersonal stressors (particularly among peers and families) are a known risk factor not only for first-onset psychopathologies, but also for maintenance or recurrent course of illness. Interpersonal stressors interact with a variety of biobehavioral stress response systems that hold important implications for youth psychotherapy outcomes. Persistent assaults on stress response systems may impede efforts to modify stress response (Boyce & Ellis, 2005) by building skills through psychotherapy.

Peer relationship stress

Peer-related stressors, including bullying victimization, have been linked to increased cognitive and emotional vulnerabilities associated with youth psychopathology. Meta-analytic findings suggest that peer victimization is associated with a nearly twofold increase in risk for anxiety disorders, greater than twofold risk for depression (increasing to a threefold risk in prospective studies), and a nearly twofold increase in risk for suicidal ideation and behaviors (Moore et al., 2017). Research also indicates that a combination of behavioral and biological indices of stress responsiveness and emotion regulation associated with peer stressors may partially account for heightened risk of psychopathology. Prospective research in a large community sample of young adolescents has identified higher levels of peer victimization as a predictor of greater emotion dysregulation (rumination, dysregulated emotional responses to sadness and anger, and low emotional awareness), which in turn mediated the effects of victimization on increases in anxiety and depression symptoms over time (McLaughlin, Hatzenbuehler, & Hilt, 2009). In another study, history of peer victimization predicted greater rumination during an in vivo peer stressor (challenging laboratory-based activity with a novel peer), which partially accounted for associations between victimization history and current depression symptoms (Monti, Rudolph, & Miernicki, 2017).

Furthermore, among adolescent girls, history of peer victimization and higher cognitive vulnerability (i.e., hopelessness) were associated with inflammatory response (greater baseline levels of proinflammatory cytokines); for victimized youths with high levels of hopelessness, inflammation increased acutely following a social stressor in the lab (Giletta et al., 2018). It has also been suggested that youths who have experienced victimization, either by peers or in the form of maltreatment, show blunted cortisol recovery following the Trier Social Stress Task – this blunted response is associated with greater social and behavioral problems (Ouellet-Morin et al., 2011). Turning to other indices of stress response, including parasympathetic nervous system reactivity during a stressor, lower vagal withdrawal (RSA reactivity) during an in vivo interpersonal stressor has been shown to amplify the strength of association between history of peer victimization and current depression symptoms (Lambe, Craig, & Hollenstein, 2019).

Another important factor (relevant to both childhood adversities and interpersonal stressors) is the effect of stress on the timing of pubertal onset. A recent review highlights findings that threat exposures in particular (e.g., violence exposure, child abuse) have been linked with earlier onset of pubertal maturation and accelerated biological aging (e.g., DNA methylation; Sumner, Colich, Uddin, Armstrong, & McLaughlin, 2019), while deprivation (e.g., poverty, neglect, food insecurity) has been linked with delayed pubertal onset (Belsky, 2019). Belsky (2019) suggests that the impact of adversity on aging may be an environmental adaptation, prioritizing reproductive strategy over emotional wellness, where it is adaptive to accelerate maturation under threat for increased reproductive chances and to maintain instead of mature in times of deprivation. Accelerated pubertal onset has further been identified as a transdiagnostic risk factor for youth psychopathology across both sexes (Hamlat, Snyder, Young, & Hankin, 2019) and there is evidence that this earlier onset is related to stronger associations between depression symptoms and the generation of subsequent interpersonal stressors (Rudolph, 2008). Given the cyclical nature of depression and the effects of interpersonal stress on behavioral and biological regulation, the chronicity of interpersonal stress may heighten risk for onset or recurrence of depression (Hankin, 2015).

Familial conflict and functioning

In addition to the effects of peer stressors, familial conflict and family dysfunction are well-documented risk factors for developmental disruptions and youth internalizing and externalizing psychopathology. Adolescents' perceptions of poorer family functioning are associated with less adaptive coping skills, which function as an indirect pathway from family functioning to greater internalizing and externalizing problems (Francisco, Loios, & Pedro, 2016). At the biological level of analysis, greater intrafamily conflict is associated with youths' hypothalamic–pituitary–adrenal (HPA) axis functioning across development. Of note, patterns of hyper- and hypocortisol reactivity have been found in response to different forms of stress and at differing points in development (Bosch et al., 2012; Ouellet-Morin et al., 2011; Spies, Margolin, Susman, & Gordis, 2011). In early childhood, hypocortisol reactivity, or blunted cortisol production, was observed in one study in response to parental conflict (simulated in the lab) and subsequently predicted greater externalizing behaviors over 2 years (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007). In later childhood, higher levels of family-reported marital conflict were associated with increasing trajectories of internalizing symptoms for youths' over time – specifically if they exhibited either blunted autonomic activity at rest or showed greater parasympathetic reactivity (higher RSA) in conjunction with lower sympathetic reactivity (skin conductance) in response to a stressor (El-Sheikh, Keiley, Erath, & Dyer, 2013). Research in adolescents also indicates that associations between observed familial conflict and blunted cortisol reactivity are evident for youths with greater internalizing symptoms, but not for youths with lower symptoms (Spies et al., 2011). In addition to the effects of familial functioning and conflict on youths' biological stress response systems, these stressors serve as a pathway from maternal depression to youth depression symptoms (Daches, Vine, Layendecker, George, & Kovacs, 2018).

Implications for youth psychotherapy

Despite the effect of interpersonal stressors on both behavioral and biological self-regulatory systems (e.g., autonomic nervous system activity, HPA axis function, less effective emotion regulation and coping behaviors), interpersonal stressors have not been systematically tested or identified as predictors or moderators of youth psychotherapy outcomes. In any such research evaluating the impact of psychosocial risk on psychotherapy effects, it

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would be important to consider whether measures of interpersonal stress are too distal from treatment outcomes and whether the *consequences* of interpersonal stress on dysregulated biobehavioral regulatory and stress response systems are more proximal to EBP outcomes. In other words, from a developmental cascade perspective, exposure to a risk factor may affect multiple domains of functioning that vary in their proximity to treatment outcomes (Masten & Cicchetti, 2010). The importance of disentangling proximal and distal effects of various risk factors aligns with existing theoretical recommendations for the use of multiple levels of analysis (e.g., neurobiological, behavioral, and social processes) in clinical trials. This approach could aid identification of the precise levels of analysis at which psychosocial risk may affect psychotherapy mechanisms or outcomes (Cicchetti & Gunnar, 2008).

One strength of a multilevel approach to evaluating psychosocial risk and EBP outcomes is the ability to investigate how targeted EBP skills impact hypothesized change processes in the context of multiple indices of risk. For example, some of the most common psychotherapy skills in EBPs for internalizing disorders involve identifying and changing biased cognitive appraisals (Chorpita & Daleiden, 2009), including attributions for others' behaviors and an individual's own ability to cope with stressful external circumstances. If these EBP skills change youths' behavioral responses but do not influence the biological processes underlying maladaptive cognitions, then youths with chronic interpersonal stress may have difficulty applying the skills when maladaptive stress appraisals are repeatedly triggered (Flynn & Rudolph, 2011). Gauging the reach of EBP skills across behavioral and biological levels of analysis offers the potential to better understand "where" and "how" psychosocial risk factors impact the treatment process.

On a practical level, intervention effectiveness may be strengthened through strategies that match treatments not only to youths' disorder-specific cognitive, emotional, and behavioral deficits, but also to developmental disruptions that may have been triggered by risks such as interpersonal stress. Two meta-analyses of EBPs for youth depression found that effect sizes for IPT were significantly larger than effect sizes for CBT – although there are significantly fewer IPT trials and it is quite possible that effect sizes will diminish as more trials are conducted (Eckshtain et al., 2020). However, the primary focus of IPT on relationships and interpersonal effectiveness could be well matched to the disruptions in behavioral and biological stress response systems associated with interpersonal stress for youths chronically exposed to this risk factor. Specifically, greater integration of developmental science on interpersonal stress may advance our efforts to identify "which treatment for which individual." Lastly, the developmental science of pubertal maturation and interpersonal stress has the potential to enhance our identification of high-risk youths. Research on adversity, stress, and the timing of pubertal onset suggests that youths presenting with early-onset depression may be a particularly vulnerable subgroup with more profound disruptions in development, thus warranting targeted efforts for personalizing EBPs.

Family-Level Risk

Familial circumstances and characteristics profoundly and persistently shape youths' immediate environments and represent the context in which psychotherapy gains are expected to take root for young people. The interconnected nature of family risk factors may also provoke a particularly robust set of additive risks for psychopathology and for poorer EBP outcomes.

Parental characteristics

Parental psychopathology

One of the most potent risk factors for youth-onset psychopathology is parental psychopathology, maternal depression in particular (Goodman et al., 2011). Long-standing developmental psychopathology models demonstrate that several factors likely heighten risk for youth-onset depression (e.g., Goodman & Gotlib, 1999), including the course of parental psychopathology and the developmental timing of youths' exposure. Meta-analytic research has shown maternal depression to be associated with both child and adolescent internalizing and externalizing behaviors (Goodman et al., 2011) and, even in the context of familial discord, longitudinal research suggests maternal depression may have the stronger effect on risk for youth anxiety and depression (Pilowsky, Wickramaratne, Nomura, & Weissman, 2006).

Some of the most robust associations are found between parental and offspring depression. There is some evidence that maternal depression is linked to earlier-onset youth depression (Hammen, Brennan, & Keenan-Miller, 2008) and that both lifetime history of parental depression and recent depressive episodes are additive risk factors for depression and other internalizing symptoms in offspring (Mars et al., 2012). Further, while there are no identified biomarkers for offspring risk of depression among depressed parents, maternal depression has been associated with depressogenic cognitive and emotion processing biases among never-depressed female offspring, but not female offspring of mothers with no history of depression (Joormann, Gilbert, & Gotlib, 2010; Joormann, Talbot, & Gotlib, 2007). Familial risk is also associated with differential patterns of neural activation in reward processing for high- compared with low-familial-risk girls, including lower striatal response to reward anticipation (Olino et al., 2014), which is a common finding in depressed individuals with a history of early life adversity (e.g., Goff & Tottenham, 2015).

The strength of associations between parental psychopathology and youth anxiety has been questioned in recent years. While there is some prospective research indicating a link between maternal depression (in combination with parenting behaviors such as overcontrol) and higher levels of anxiety over time in boys (Feng, Shaw, & Silk, 2008), meta-analytic research suggests that existing empirical support does not meet the quality benchmarks for drawing strong conclusions about links between parental psychopathology and anxiety (Yap & Jorm, 2015). However, parental psychopathology is more clearly associated with youths' externalizing behaviors (Goodman et al., 2011). Maternal depression in early childhood has been shown to mediate links between youths' early ADHD symptoms and the subsequent development of oppositional behaviors over time (Harvey, Metcalfe, Herbert, & Fanton, 2011). In addition to associations among parental depression and youth internalizing and externalizing behaviors, there is evidence that parental ADHD is associated with youths' oppositional, aggressive, and ADHD symptoms in middle childhood (Humphreys, Mehta, & Lee, 2012).

Parental psychopathology and youths' stress sensitivity and responsiveness

An important domain of youth functioning that may account for some portion of the association between parental psychopathology and youth psychopathology is stress exposure and responsiveness. Children of mothers with recurrent depression have been

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shown to report greater chronic and discrete stress in peer, parental, and other familial relationships over time as compared with children of mothers who were never depressed or had a single depressive episode (Feurer, Hammen, & Gibb, 2016). The longitudinal population based study Tracking Adolescents' Individual Lives Survey further demonstrated greater "sensitivity" to stress – meaning that stress exposure was more strongly associated with depressive symptoms – among offspring of parents with a lifetime history of depression compared with never-depressed parents (Bouma, Ormel, Verhulst, & Oldehinkel, 2008). Stress responsiveness in the form of poorer primary control coping (e.g., problem solving) and secondary control coping (e.g., cognitive restructuring, acceptance skills) has been shown to account for the association between higher parental and youth depressive symptoms in girls (Thompson, Mata, Gershon, & Gotlib, 2017).

At the cognitive and biological levels of analysis, low effortful control (a central cognitive self-regulatory process involving attention and inhibition systems that facilitates emotional and behavioral coping responses) has been shown to amplify the association between maternal depression and youth internalizing and externalizing symptoms (Lengua, Bush, Long, Kovacs, & Trancik, 2008). Autonomic nervous system reactivity also moderates relations among parent and offspring psychopathologies. Greater sympathetic reactivity (skin conductance response) to stress in the lab was found to amplify associations between parent and child psychopathology (Cummings, El-Sheikh, Kouros, & Keller, 2007). Lower resting-state parasympathetic activity (RSA) in young girls amplified relations between parental depression and higher offspring internalizing symptoms over 3 years (Wetter & El-Sheikh, 2012).

Parenting behaviors

Parental psychopathology and its association with youths' stress sensitivity and stress response may heighten risk for youth-onset psychopathology via another familial risk mechanism: parenting behaviors. Parental psychopathology has been shown to predict less optimal parenting behaviors, which in turn predict greater youth internalizing and externalizing problems. There is also some evidence of specificity in the association between different parenting behaviors and varied youth outcomes. Specifically, poor parental monitoring and higher parental rejection have been shown to mediate links between parental depression and offspring internalizing problems, while low nurturance and higher parental rejection mediate links between parental depression and offspring externalizing problems (Elgar, Mills, McGrath, Waschbusch, & Brownridge, 2007). Similar to rejection, parental withdrawal and intrusiveness nonspecifically mediate associations between parental depression and youths' internalizing and externalizing problems in childhood and adolescence (Reising et al., 2013). These effects are not specific to parental depression – negative parenting behaviors (inconsistent discipline, corporal punishment, poor monitoring) also mediate links between parental ADHD symptoms and offspring's ADHD and oppositional symptoms (Moroney, Tung, Brammer, Peris, & Lee, 2017).

In addition to linking parental and offspring psychopathology, parenting behaviors are independent and additive predictors. Parental behaviors that directly predict disruptive behavior problems and emotion dysregulation (a transdiagnostic risk for psychopathology) include inconsistent discipline and negative emotional expressiveness (Duncombe, Havighurst, Holland, & Frankling, 2012). Harsh verbal discipline predicts increases in depressive and conduct symptoms among adolescents, while low parental warmth is associated with increases in youths' depression symptoms over time (Hipwell et al., 2008; Wang & Kenny, 2014). In addition to the effect of parenting behaviors on youths' self-reported behavioral problems, parenting behaviors such as hostility may amplify the association between parents' depression and synchrony of parent–youth biological stress responses in early childhood. The association between lower parent/youth diurnal cortisol production (typically considered a blunted response) and parental depression was amplified by greater parental hostility, and hypocortisol production was then further associated with higher youth externalizing problems in one study (Merwin, Leppert, Smith, & Dougherty, 2017).

Household "chaos"

Finally, a potentially key risk factor that ties together parental psychopathology, parenting, and individual-level risks such as deprivation-related adversities is greater instability and unpredictability in youths' environments – referred to as household chaos (Dumas et al., 2005). Chaos includes less predictable discipline from parents, fluctuations in residence and the number of people in the house, less structured daily routines for children, greater background noise in the home, and mealtime insecurity. Greater chaos has been shown to amplify associations between poor paternal–youth relationship quality and higher youth internalizing and externalizing behaviors (Coldwell, Pike, & Dunn, 2006). In early childhood, associations between chaos and youth externalizing behaviors were accounted for by poorer youth cognitive inhibition (Hardaway, Wilson, Shaw, & Dishion, 2012).

More chaotic environments seem to disproportionately influence youth outcomes among families with greater deprivation. Prospectively, greater household chaos has been shown to mediate links between familial poverty and youths' cognitive control and total internalizing and externalizing problems in middle childhood (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005). Under conditions of low economic resources, greater household chaos is also associated with greater biological production of proinflammatory cytokines and interleukin-6 (Schreier, Roy, Frimer, & Chen, 2014). Finally, given that disruptions in executive function underly psychopathology, it is notable that greater household chaos is associated with poorer maternal executive functioning, specifically in the context of greater economic deprivation (Deater-Deckard, Chen, Wang, & Bell, 2012). Since maternal psychopathology is a potent risk factor for youths, the interaction among chaos and cognitive functions underlying maternal health could help to explain links between parental and offspring psychopathology.

Implications for youth psychotherapy

Exposure to each of the above familial risk factors can be linked in some way to youth psychotherapy outcomes. There is evidence that parental depression is associated with poorer CBT outcomes (slower trajectories of change and less reduction in symptoms) for depressed youths (Eckshtain, Marchette, Schleider, & Weisz, 2018). In the treatment of co-occurring conduct problems and depression, clinic-referred youths with a depressed mother were more likely than youths without a depressed mother to maintain clinical-level severity of depressive symptoms at the end of a combination treatment that included parent management training and CBT for youths (Van Loon, Granic, & Engels, 2011). The evidence for effects of parental psychopathology on the treatment

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of youth anxiety disorders is less clear. Parental anxiety and depression have been associated with poorer CBT response among anxious pre-adolescents (Liber et al., 2008). In other cases, parental depression has predicted improvements in youths' psychotherapy outcomes via improvements in both family functioning and parental stress (Schleider et al., 2015). Other studies have found no association between parental psychopathology and youth psychotherapy outcomes for anxiety (Knight, McLellan, Jones, & Hudson, 2014). There is stronger evidence that parental psychopathology has deleterious effects on psychotherapy outcomes for externalizing behaviors, even among some of the most widely tested parent training programs, including Incredible Years, the Triple-P Parenting Program, and parent management training, which have augmented versions for families at higher risk of nonresponse (Maliken & Katz, 2013; Reyno & McGrath, 2006).

Given the associations of parental psychopathology with youths' cognitive biases and altered biobehavioral functioning, the role of caregivers in youth psychotherapies warrants further evaluation. Although parents are the primary participant in EBPs for disruptive behaviors (parent training protocols), preliminary evidence suggests that similar approaches may strengthen EBPs for youth depression. One study found that traditional parent training was as effective as individual CBT in reducing depression among treatment-seeking youths with a depressed caregiver (Eckshtain, Kuppens, & Weisz, 2017). Another study, comparing family-focused treatment for child depression with individual psychotherapy, found evidence that family-focused treatment was associated with parents' improved understanding of how to manage their children's depression and how to help their children at home (Tompson, Sugar, Langer, & Asarnow, 2017), even though the treatments were similarly effective for youth depression at 1-year follow-up (Asarnow et al., 2020). Many CBT protocols encourage transmission of treatment information to parents via parent handouts, check-ins at the end of therapy sessions, or designated therapy sessions for the parents. Systematic integration of parents into treatments may hold promise for improved outcomes, given some evidence of improved parental psychiatric symptoms and reductions in negative parenting behaviors following parent training (Shaffer, Lindhiem, & Kolko, 2013; Van Loon et al., 2011). As a cautionary note, though, we stress that the effects of parent training on parent outcomes are mixed (Maliken & Katz, 2013).

Traditional parent training programs are not the only caregiver-focused approaches previously tested for depression treatment. For example, an open trial of an augmented CBT protocol for youth depression with ~50% of the sessions designed for caregiver–youth dyads has shown promise in meeting or exceeding benchmarks for CBT effectiveness for youth depression (Eckshtain & Gaynor, 2012). The first author, Dikla Eckshtain (Massachusetts General Hospital/Harvard Medical School), recently completed a Sequential, Multiple Assignment, Randomized Trial, funded by the National Institute of Mental Health (NIMH), comparing the dosing and sequence of the parent-augmented CBT protocol to traditional youth-focused CBT for depressed children and adolescents. It is not surprising that parental involvement in depression psychotherapies may strengthen treatment outcomes given that parent training programs for youth externalizing problems have long been considered gold standard and inclusion of parents in CBT for youth anxiety (e.g., teaching parent training skills for reinforcement and modeling, parental psychoeducation) is considered a front-line treatment approach (Higa-McMillan, Francis, Rith-Najarian, & Chorpita, 2016).

Although greater caregiver inclusion in psychotherapies may be a practical approach for targeting the effects of parental psychopathology on youth outcomes, it is not clear whether this approach will strengthen the impact of psychotherapies on behavioral and biological processes that *link* parental factors with youth dysfunction. As reviewed above, parental psychopathology and parenting behaviors impact youth outcomes across development and predict disruptions in underlying stress response systems as well as cognitive, emotional, and behavioral self-regulatory systems (e.g., cognitive and emotion processing biases, lower reward responsiveness, stress physiology). These systems are believed to be important in the successful uptake of EBP skills. One approach for operationalizing and testing the effects of parent-focused strategies on improvement in youths' biobehavioral self-regulation is to explore reinforcement behaviors as a possible mechanism of change (Segers et al., 2018). At the core of parent training programs, caregivers learn strategies to intentionally and consistently engage in positive parenting practices by reinforcing youths' self-regulatory behaviors through praise for positive behaviors, attention withdrawal and consistent consequences for negative behaviors, extrinsic rewards to increase youths' motivation to use skills, and strategies for improving the quality of youth–parent relationships.

Given the deleterious effects of environmental inconsistency (household chaos), assessing and operationalizing consistency of parental reinforcement in youth psychotherapy could shed light on whether changes in environmental predictability affect biobehavioral regulation. Basic developmental science theories indicate that dysregulation in emotion and stress response systems are influenced by chronic environmental insults that shape individuals' openness to environmental inputs (Ellis & Del Giudice, 2014). Elucidating whether parental reinforcement and environmental consistency lead to adaptive shifts in youths' biobehavioral regulatory responses (Boyce & Ellis, 2005) would, importantly, inform our understanding of the malleability of biobehavioral systems via psychotherapy. This approach could be applied to existing EBPs for internalizing problems by incorporating parental reinforcement to promote youths' use of skills for problem solving, cognitive restructuring, emotion regulation, and other strategies for promoting self-regulation. Across both internalizing and externalizing problems, predictability in the environment may be key for the uptake and generalization of psychotherapy skills. Assessing household chaos at the start of treatment and promoting strategies to enhance predictability (e.g., establishing daily routines, using consistency-based strategies from parent training programs) could be a promising approach to strengthening psychotherapies and mitigating the effects of psychosocial risk on outcomes.

Emphasizing the role of families and parents in youth interventions is not a novel concept. In the 1980s, the Family Support Movement arose in the USA, highlighting the support that families needed for youth interventions to be effective (Zigler & Black, 1989). However, Zigler and Black (1989) reported that this movement was plagued by a lack of well-defined outcomes that ruled out rigorous evaluation. To ensure that mechanism-focused research on youth mental health treatments does not suffer the same fate, it will be important to identify well-operationalized and psychometrically sound measures of stress response and biobehavioral regulation, relevant parenting behaviors, and psychotherapy skills acquisition.

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A Framework for Developmentally-Informed Intervention Science

We have suggested throughout this article that developmental psychopathology has much to offer scientifically to intervention research and the enterprise of treatment development. In this perspective, we join with prominent leaders in the field whose research and conceptual advances have pointed the way toward such a synthesis (e.g., Cicchetti & Gunnar, 2008; Doom & Cicchetti, 2020; McLaughlin et al., 2019) As new findings emerge, it will be possible to build increasingly elaborated frameworks for integrating these bodies of work. Figure 1 shows our particular take on what such a framework might be, based on the evidence reviewed here. This framework builds on the developmental science of psychosocial risk and resulting developmental disruptions associated with psychopathology, linking that body of work with both hypothesized and empirically supported mechanisms that might be targeted in intervention, and extending to hypotheses about psychotherapy methods to address those targets.

Future directions and considerations for strengthening youth psychotherapy

Developmental psychopathology research has generated a rich evidence base documenting the associations between psychosocial risk and the onset and course of youth psychopathology. This framework, rich in potential to inform intervention science (Cicchetti & Hinshaw, 2002), continues to hold significant promise for the integration of contextual risk factors into psychotherapy research. Common forms of psychosocial risk at both the individual level (e.g., childhood adversities, interpersonal stress) and family level (e.g., parental psychopathology, maladaptive parenting behaviors) affect youth functioning in many of the domains targeted by EBPs (e.g., cognitive, behavioral, and emotional self-regulatory processes) for transdiagnostic problems. Investigating these risk exposures in the development and evaluation of psychotherapies could shed critical light on individual differences in treatment outcomes, inform approaches to treatment design and personalization, and increase precision in identifying change mechanisms and moderators of treatment response in EBPs. Effectively treating psychopathology in youths has significant long-term implications, considering that the risk-related disruptions in development reviewed throughout this article have been shown to predict poorer prognosis for psychopathology well into adulthood (Zigler & Glick, 2001).

The psychotherapy mechanisms highlighted throughout this article are specifically relevant to psychosocial risk exposure and disruptions in developmental processes. Although there are many other psychotherapy mechanisms that extend beyond the scope of this review, addressing target participants (e.g., inclusion of caregivers in youth treatments), associative learning and reinforcement processes in skills-based EBPs, and treatment matching innovations build feasibly upon advances in developmental and intervention sciences (McLaughlin et al., 2019; Ng & Weisz, 2016). These specific intervention processes are also consistent with the NIMH's current research priorities of identifying risk factors that predict response to intervention or that may serve as target change mechanisms in treatments (National Institute of Mental Health, 2015).

The framework presented in this article and the NIMH strategic plan may be seen as consistent, in some respects, with a medical model in which specific treatment mechanisms produce change in specific behavioral, biological, and cognitive processes. However, there are certainly psychotherapy mechanisms that we did not review and it is important to acknowledge common factors in psychotherapies that contrast with the medical model approach. Cuijpers, Reijnders, and Huibers (2019) review decades of research suggesting that most psychotherapies work through a set of common factors, including (a) the positive effects of a relationship with a trusted clinician (who may provide reinforcement and social stability), (b) the promotion of patients' expectations for improvement, and (c) specific ingredients in therapies that promote adaptive actions (e.g., engaging in meaningful activities) but do not necessarily target a deficit. That said, this meta-analysis comparing different psychotherapies shows that existing studies lack both the statistical power and the design rigor that would be needed to determine whether common or specific factors are more responsible for outcomes across EBPs (Cuijpers et al., 2019). Thus, it is important to remember that the landscape of intervention development, implementation, and dissemination is complex, and any research agenda focused on understanding the impact of psychosocial risk exposure on psychotherapy outcomes must consider the influence of a host of third variables that have not yet been clearly identified or adequately studied.

Another important consideration for any research agenda leveraging developmental science to strengthen youth psychotherapies is access to mental health care. Fewer than half of youths with psychiatric disorders receive treatment (Merikangas et al., 2011; Whitney & Peterson, 2019) and rates of psychotherapy dropout range from nearly 30% to 75% in clinical trials alone (de Haan, Boon, de Jong, Hoeve, & Vermeiren, 2013). Some clinical trials data suggest that early dropout from depression psychotherapies did not affect outcomes and there is evidence that single-session interventions can be effective for a variety of internalizing/externalizing problems – together indicating that some treatment is often better than none (O'Keeffe et al., 2019; Schleider & Weisz, 2017). Yet, any innovations in treatment design or identification of treatment mechanisms that may increase the robustness of psychotherapies against the deleterious effects of psychosocial risk should include strategies for enhancing access to these interventions. A key example of the effective dissemination of services comes from the field of early childhood intervention, including the work of Ed Zigler on the Head Start program. These services have been strategically embedded in settings where youths and families can most easily access them, and Head Start has shown positive effects on both youth and family outcomes (Zigler & Berman, 1983; Zigler & Styfco, 1994). While Head Start is focused on academic and social outcomes, some of the strategies employed in the coordinated engagement of federal and local stakeholders in Head Start's dissemination and implementation might warrant attention in efforts to increase accessibility to psychotherapies.

In addition to considering access to mental health care, efforts to mitigate the research–practice gap will be important for strengthening psychotherapies against the deleterious effects of psychosocial risk exposure. The translation of basic research findings into everyday clinical practice is a laborious process and only a fraction of treatments developed by researchers make it through the pipeline to widespread adoption. One model that may support more rapid integration of developmental and intervention sciences is the deployment-focused model of psychotherapy research (Weisz, 2004). This framework makes a case for developing and refining psychotherapies in the settings in which they are ultimately to be implemented (e.g., community-based clinics and

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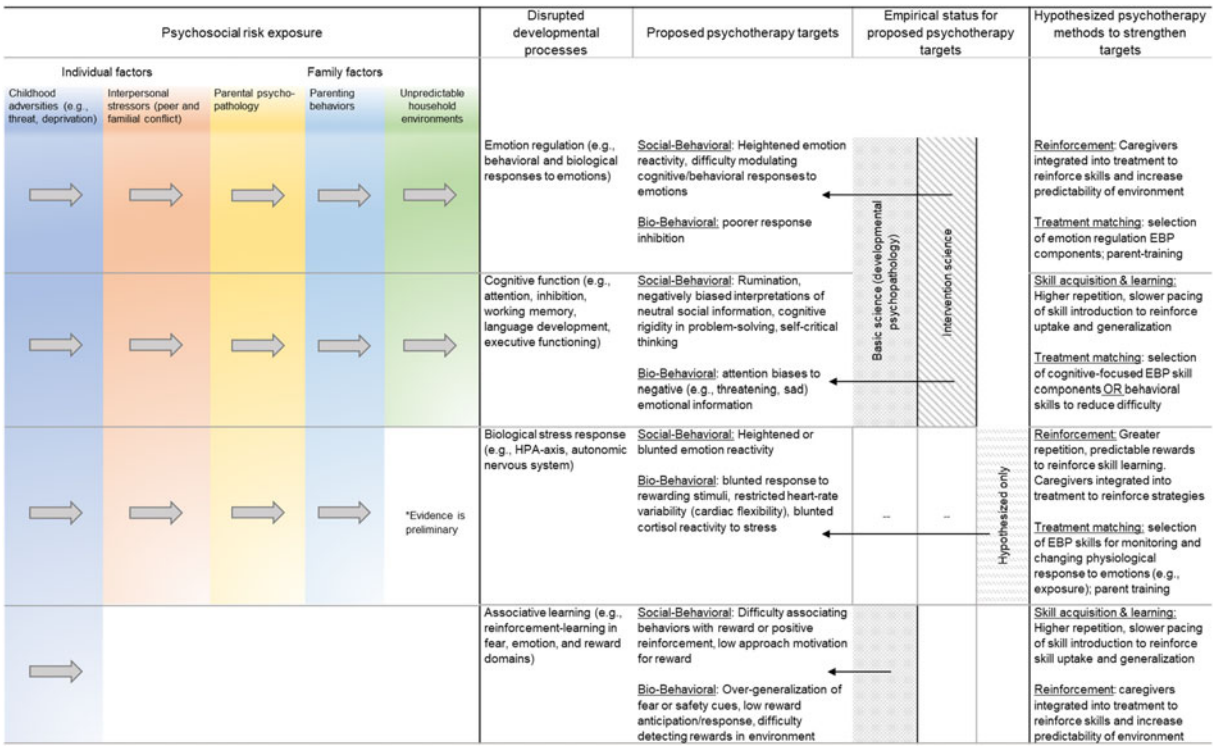

Figure 1. Psychosocial risk exposure, developmental processes, and psychotherapy targets and methods.
Risk factors associated with each area of disrupted development are indicated with an arrow in the corresponding row (e.g., all risk factors are associated with cognitive function, and all risk factors except for unpredictable household environments [evidence is preliminary] are associated with biological stress response). Social–behavioral psychotherapy targets represent observed behaviors and social processes typically assessed in the course of evidence-based psychotherapies (explicit targets of psychotherapy skills). Biobehavioral psychotherapy targets represent underlying processes typically assessed with basic behavioral or neuropsychological tasks, psychophysiological methodology, and biological sampling (e.g., salvia collection to measure cortisol production). Hypothesized psychotherapy methods for addressing treatment targets all remain to be empirically tested.

centers), with the providers and patient populations that will, respectively, ultimately deliver and receive the psychotherapies (Weisz, 2004). Lack of psychotherapy research in community contexts where treatments will be deployed may contribute to reduced likelihood that the treatments will attain widespread adoption. A strength of effective, developmentally-informed programs such as the aforementioned Head Start and ABC programs is that they were designed specifically for implementation and evaluation within their target populations in youths' and families' everyday contexts, thus reaching youths who might well have been out of reach to researchers operating solely within academic or laboratory settings.

It is feasible to examine the effects of psychosocial risk on both developmental processes and treatment mechanisms within a deployment-focused model of intervention science. Innovations in developmental psychopathology research have produced many mobile and wearable technologies to assess cognitive and biobehavioral functioning outside of the lab. The administration of computerized behavioral tasks designed to assess complex cognitive processes on laptops and mobile devices allows for the practical transport of these measures to clinics. The National Institutes of Health has provided a comprehensive cognitive battery, available at no cost within the "NIH Toolbox" (National Institutes of Health, 2017). Biological self-regulatory processes, including autonomic nervous system activity, HPA axis regulation of cortisol, and even circadian rhythms, can be assessed via noninvasive wearable devices that may permit testing of change mechanisms in psychotherapies in the very settings (e.g., community mental health clinics) and under the typical conditions in which these treatments would ultimately be implemented. Innovations supporting the integration of developmental science with deployment-focused intervention models are also valuable for reaching youths disproportionately affected by psychosocial risk that may not otherwise participate in research (e.g., Weisz et al., 2020).

Conclusion

To date, there has been relatively little systematic integration and testing of the association between psychosocial risk factors and youth psychotherapy outcomes, despite evidence that exposure to psychosocial risk is associated with the onset of transdiagnostic forms psychopathology and adversely affects the course of dysfunction. Individual and familial risk factors have been shown to predict disruptions in developmental processes that are often the proposed targets of EBPs (e.g., biological and behavioral emotion regulation capabilities) across therapy modalities that include CBT, DBT, IPT, and parent training programs. To more successfully design and personalize interventions to meet youths' and families' needs for effective mental health care, it is essential to understand the role of contextual factors in psychotherapy outcomes. Leveraging developmental psychopathology frameworks in intervention science is a necessary step for building therapies that can mitigate the effects of risk exposure on youth mental health.

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Conflicts of Interest. None

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