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# Future Directions in Childhood Adversity and Youth Psychopathology

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## Abstract

Despite long-standing interest in the influence of adverse early experiences on mental health, systematic scientific inquiry into childhood adversity and developmental outcomes has emerged only recently. Existing research has amply demonstrated that exposure to childhood adversity is associated with elevated risk for multiple forms of youth psychopathology. In contrast, knowledge of developmental mechanisms linking childhood adversity to the onset of psychopathology—and whether those mechanisms are general or specific to particular kinds of adversity—remains cursory. Greater understanding of these pathways and identification of protective factors that buffer children from developmental disruptions following exposure to adversity is essential to guide the development of interventions to prevent the onset of psychopathology following adverse childhood experiences. This article provides recommendations for future research in this area. In particular, use of a consistent definition of childhood adversity, integration of studies of typical development with those focused on childhood adversity, and identification of distinct dimensions of environmental experience that differentially influence development are required to uncover mechanisms that explain how childhood adversity is associated with numerous psychopathology outcomes (i.e., multifinality) and identify moderators that shape divergent trajectories following adverse childhood experiences. A transdiagnostic model that highlights disruptions in emotional processing and poor executive functioning as key mechanisms linking childhood adversity with multiple forms of psychopathology is presented as a starting point in this endeavour. Distinguishing between general and specific mechanisms linking childhood adversity with psychopathology is needed to generate empirically informed interventions to prevent the long-term consequences of adverse early environments on children's development.

The lasting influence of early experience on mental health across the lifespan has been emphasized in theories of the etiology of psychopathology since the earliest formulations of mental illness. In particular, the roots of mental disorder have often been argued to be a consequence of adverse environmental experiences occurring in childhood. Despite this long-standing interest, systematic scientific inquiry into the effects of childhood adversity on health and development has emerged only recently. Prior work on childhood adversity focused largely on individual types of adverse experiences—such as death of a parent, divorce, sexual abuse, or poverty—and research on these topics evolved as relatively independent lines of inquiry. The transition to considering these types of adversities as

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indicators of the same underlying construct was prompted, in part, by the findings of a seminal study examining childhood adversity as a determinant of adult physical and mental health and advances in theoretical conceptualizations of stress. Specifically, findings from the Adverse Childhood Experiences (ACE) Study documented high levels of co-occurrence of multiple forms of childhood adversity and strong associations of exposure to adverse childhood experiences with a wide range of adult health outcomes (Dong et al., 2004; Edwards, Holden, Felitti, & Anda, 2003; Felitti et al., 1998). Around the same time, the concept of allostatic load was introduced as a comprehensive neurobiological model of the effects of stress (McEwen, 1998, 2000). Allostatic load provided a framework for explaining the neurobiological mechanisms linking a variety of adverse social experiences to health. Together, these discoveries sparked renewed interest in the childhood determinants of physical and mental health. Since that time there has been a veritable explosion of research into the impact of childhood adversity on developmental outcomes, including psychopathology.

## CHILDHOOD ADVERSITY AND PSYCHOPATHOLOGY

Over the past two decades, hundreds of studies have examined the associations between exposure to childhood adversity and risk for psychopathology (Evans, Li, & Whipple, 2013). Here, I briefly review this evidence, focusing specifically on findings from epidemiological studies designed to allow inferences to be drawn at the population level. These studies have documented five general patterns with regard to childhood adversity and the distribution of mental disorders in the population. First, despite differences across studies in the prevalence of specific types of adversity, all population-based studies indicate that exposure to childhood adversity is common. The prevalence of exposure to childhood adversity is estimated at about 50% in the U.S. population across numerous epidemiological surveys (Green et al., 2010; Kessler, Davis, & Kendler, 1997; McLaughlin, Conron, Koenen, & Gilman, 2010; McLaughlin, Green et al., 2012). Remarkably similar prevalence estimates have been documented in other high-income countries, as well as in low- and middle-income countries worldwide (Kessler et al., 2010). Second, individuals who have experienced childhood adversity are at elevated risk for developing a lifetime mental disorder compared to individuals without such exposure, and the odds of developing a lifetime mental disorder increase as exposure to adversity increases (Edwards et al., 2003; Green et al., 2010; Kessler et al., 1997; Kessler et al., 2010; McLaughlin, Conron, et al., 2010; McLaughlin, Green, et al., 2012). Third, exposure to childhood adversity confers vulnerability to psychopathology that persists across the life-course. Childhood adversity exposure is associated not only with risk of mental disorder onset in childhood and adolescence (McLaughlin, Green, et al., 2012) but also with elevated odds of developing a first onset mental disorder in adulthood, which persists after adjustment for mental disorders beginning at earlier stages of development (Green et al., 2010; Kessler et al., 1997; Kessler et al., 2010). Fourth, the associations of childhood adversity with different types of commonly occurring mental disorders are largely nonspecific. Individuals who have experienced childhood adversity experience greater odds of developing mood, anxiety, substance use, and disruptive behavior disorders, with little meaningful variation in the strength of associations across disorder classes (Green et al., 2010; Kessler et al., 1997; Kessler et al., 2010; McLaughlin, Green, et

al., 2012). Recent epidemiological findings suggest that the associations of child maltreatment —a commonly measured form of adversity—with lifetime mental disorders operate entirely through a latent liability to experience internalizing and externalizing psychopathology with no direct effects on specific mental disorders that are not explained by this latent vulnerability (Caspi et al., 2014; Keyes et al., 2012). Finally, exposure to childhood adversity explains a substantial proportion of mental disorder onsets in the population, both in the United States and cross-nationally (Afifi et al., 2008; Green et al., 2010; Kessler et al., 2010; McLaughlin, Green, et al., 2012). This reflects both the high prevalence of exposure to childhood adversity and the strong association of childhood adversity with the onset of psychopathology.

Together, findings from epidemiological studies indicate clearly that exposure to childhood adversity powerfully shapes risk for psychopathology in the population. As such, it is time for the field to move beyond these types of basic descriptive studies to research designs aimed at identifying the underlying developmental mechanisms linking childhood adversity to psychopathology. Although ample research has been conducted examining mechanisms linking individual types of adversity to psychopathology (e.g., sexual abuse; Trickett, Noll, & Putnam, 2011), far less is known about which of these mechanisms are common across different types of adversity versus specific to particular types of experiences. Greater understanding of these pathways, as well as the identification of protective factors that buffer children from disruptions in emotional, cognitive, social, and neurobiological development following exposure to adversity, is essential to guide the development of interventions to prevent the onset of psychopathology in children exposed to adversity, a critical next step for the field. However, persistent issues regarding the definition and measurement of childhood adversity must be addressed before meaningful progress on mechanisms, protective factors, and prevention of psychopathology following childhood adversity will be possible.

## FUTURE DIRECTIONS IN CHILDHOOD ADVERSITY AND YOUTH PSYCHOPATHOLOGY

This article has two primary goals. The first is to provide recommendations for future research on childhood adversity and youth psychopathology. These recommendations relate to the definition and measurement of childhood adversity, the integration of studies of typical development with those on childhood adversity, and the importance of distinguishing between general and specific mechanisms linking childhood adversity to psychopathology. The second goal is to provide a transdiagnostic model of mechanisms linking childhood adversity and youth psychopathology that incorporates each of these recommendations.

### Defining Childhood Adversity

Childhood adversity is a construct in search of a definition. Despite the burgeoning interest and research attention devoted to childhood adversity, there is a surprising lack of consistency with regard to the definition and measurement of the construct. Key issues remain unaddressed in the literature regarding the definition of childhood adversity and the boundary conditions of the construct. To what does the construct of childhood adversity refer? What types of experiences qualify as childhood adversity and what types do not?

Where do we draw the line between normative experiences of stress and those that qualify as an adverse childhood experience? How does the construct of childhood adversity differ from other constructs that have been linked to psychopathology risk, including stress, toxic stress, and trauma? It will be critical to gain clarity on these definitional issues before more complex questions regarding mechanisms and protective factors can be systematically examined. Even in the seminal ACE Study that spurred much of the recent research into childhood adversity, a concrete definition of adverse childhood experience is not provided. The original article from the study argues for the importance of understanding the lasting health effects of child abuse and "household dysfunction," the latter of which is never defined specifically (Felitti et al., 1998). The CDC website for the ACE Study (http://www.cdc.gov/violenceprevention/acestudy/findings.html) indicates that the ACE score, a count of the total number of adversities experienced, is designed to assess "the total amount of stress experienced during childhood."

Why has a concrete definition of childhood adversity remained elusive? As I see it, there is a relatively simple explanation for this notable gap in the literature. Childhood adversity is difficult to define but fairly obvious to most observers, making the construct an example of the classic standard of *you know it when you see it*. Although this has allowed a significant scientific knowledge base on childhood adversity to emerge within a relatively short period, the lack of an agreed-upon definition of the construct represents a significant impediment to future progress in the field.

How can we begin to build scientific consensus on the definition of childhood adversity? Critically, we must come to an agreement about what childhood adversity is and what it is not. Adversity is defined as "a state or instance of serious or continued difficulty or misfortune; a difficult situation or condition; misfortune or tragedy" ("Adversity," 2015). This provides a reasonable starting point. Adversity is an environmental event that must be *serious* (i.e., severe) or a series of events that *continues over time* (i.e., chronic). Building on Scott Monroe's (2008) definition of life stress and models of experience-expectant brain development (Baumrind, 1993; Fox, Levitt, & Nelson, 2010), I propose that childhood adversity should be defined as *experiences that are likely to require significant adaptation by an average child and that represent a deviation from the expectable environment*. The expectable environment refers to a wide range of species-typical environmental inputs that the human brain requires to develop normally. These include sensory inputs (e.g., variation in patterned light information that is required for normal development of the visual system), exposure to language, and the presence of a sensitive and responsive caregiver (Fox et al., 2010). As I have argued elsewhere (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014), deviations from the expectable environment often take two primary forms: an absence of expected inputs (e. g., limited exposure to language or the absence of a primary caregiver), or the presence of unexpected inputs that represent significant threats to the physical integrity or well-being of the child (e.g., exposure to violence). A similar approach to classifying key forms of child adversity has been articulated by others as well (Farah et al., 2008; Humphreys & Zeanah, 2015). These experiences can either be chronic (e.g., prolonged neglect) or involve single events that are severe enough to represent a deviation from the expectable environment (e.g., sexual abuse). Together, this provides a working definition of childhood adversity: exposure during childhood or adolescence to

environmental circumstances that are likely to require significant psychological, social, or neurobiological adaptation by an average child and that represent a deviation from the expectable environment.

This definition provides some clarity about what childhood adversity is *not*. The clearest boundary condition involves the developmental timing of exposure; experiences classified as childhood adversity must occur prior to adulthood, either during childhood or adolescence. Most research on childhood adversity has taken a broad definition of childhood, including events occurring during either childhood or adolescence. Although the demarcation between adolescence and adulthood is itself a point of debate, relative consensus exists regarding the onset of adult roles as the end of adolescence (Steinberg, 2014). Second, childhood adversity refers to an event or ongoing events in the environment. Childhood adversity thus refers only to specific environmental circumstances or events and not to an individual child's response to those circumstances. Third, childhood adversity refers to environmental conditions that are likely to require significant psychological, social, or neurobiological adaptation by an average child; therefore, events that represent transient or minor hassles should not qualify. What types of events should be considered severe enough to warrant classification as adversity? Although there is no absolute rule or formula that can be used to distinguish circumstances or events requiring significant adaptation from those that are less severe or impactful, childhood adversity should include conditions or events that are likely to have a meaningful and lasting impact on developmental processes for most children who experience them. In other words, experiences that could alter fundamental aspects of development in emotional, cognitive, social, or neurobiological domains are the types of experiences that should qualify as adversity. Studies of childhood adversity should clearly define the study-specific decision rules used to distinguish between adversity and more normative stressors. Finally, environmental circumstances or stressors that do not represent deviations from the expectable environment should not be classified as childhood adversity. In other words, childhood adversity should not include any and all stressors that occur during childhood or adolescence. Two examples of childhood stressors that would likely *not* qualify as childhood adversity based on this definition, because they do not meet the condition of representing a deviation from the expectable environment, are moving to a new school and death of an elderly grandparent. Each of these childhood stressors should require adaptation by an average child, and could influence mental health and development. However, neither represents a deviation from the expectable childhood environment and therefore does not meet the proposed definition of childhood adversity.

A key question for the field is whether the definition of childhood adversity should be narrow or broad. This question will determine whether other common forms of adversity or stress should be considered as indicators of childhood adversity. For example, many population-based studies have included parental psychopathology and divorce as forms of adversity (Felitti et al., 1998; Green et al., 2010). Given the high prevalence of psychopathology and divorce in the population, consideration of any form of parental psychopathology or any type of divorce as a form of adversity results in a fairly broad definition of adversity; certainly, not all cases of parental psychopathology or all divorces result in significant adversity for children. A more useful approach might be to consider only those cases of parental psychopathology or divorce that result in parenting behavior that

deviates from the expectable environment (i. e., consistent unavailability, unresponsiveness, or insensitive care) or that generate other types of significant adversity for children (e.g., economic adversity, emotional abuse, etc.) as meeting the threshold for childhood adversity. Providing these types of boundary conditions is important to prevent the construct of childhood adversity from meaning everything and nothing at the same time.

Finally, how does childhood adversity differ from related constructs, including stress, toxic stress, and trauma that can also occur during childhood? What is unique about the construct of childhood adversity that is not captured in definitions of these similar constructs? First, how is childhood adversity different from stress? The prevailing conceptualization of life stress defines the construct as the adaptation of an organism to specific circumstances that change over time (Monroe, 2008). This definition includes three primary components that interact with one another: environment (the circumstance or event that requires adaptation by the organism), organism (the response to the environmental stimulus), and time (the interactions between the organism and the environment over time; Monroe, 2008). In contrast, childhood adversity refers only to the first of these three components—the environmental aspect of stress. Second, how is adversity different from toxic stress, a construct recently developed by Jack Shonkoff and colleagues (Shonkoff & Garner, 2012)? Toxic stress refers to the second component of stress just described—the response of the organism. Specifically, toxic stress refers to exaggerated, frequent, or prolonged activation of physiological stress response systems in response to an accumulation of multiple adversities over time in the absence of protection from a supportive caregiver (Shonkoff & Garner, 2012). The concept of toxic stress is conceptually similar to the construct of allostatic load as defined by McEwen (2000) and focuses on a different aspect of stress than childhood adversity. Finally, how is childhood adversity distinct from trauma? Trauma is defined as exposure to actual or threatened death, serious injury, or sexual violence, either by directly experiencing or witnessing such events or by learning of such events occurring to a close relative or friend (American Psychiatric Association, 2013). Traumatic events occurring in childhood represent one potential form of childhood adversity, but not all types of childhood adversity are traumatic. Examples of adverse childhood experiences that would not be considered traumatic are neglect; poverty; and the absence of a stable, supportive caregiver.

*The first concrete recommendation for future research is that the field must utilize a consistent definition of childhood adversity* (see Table 1). A useful definition must have clarity about what childhood adversity is and what it is not, provide guidance about decision rules for applying the definition in specific contexts, and increase consistency in the measurement and application of childhood adversity across studies. The definition proposed here—that childhood adversity involves experiences that are likely to require significant adaptation by an average child and that represent a deviation from the expectable environment—represents a starting point in this endeavor, although consideration of alterative definitions and scholarly debate about the relative merits of different definitions is encouraged.

## Integrating Studies of Typical and Atypical Development

A developmental psychopathology perspective emphasizes the reciprocal and integrated nature of our understanding of normal and abnormal development (Cicchetti, 1996; Cicchetti & Lynch, 1993; Lynch & Cicchetti, 1998). Normal developmental patterns must be characterized to identify developmental deviations, and abnormal developmental outcomes shed light on the normal developmental processes that lead to maladaptation when disrupted (Cicchetti, 1993; Sroufe, 1990). Maladaptive outcomes—including psychopathology—are considered to be the product of developmental processes (Sroufe, 1997, 2009). This implies that in order to uncover mechanisms linking childhood adversity to psychopathology, the developmental trajectory of the candidate emotional, cognitive, social, or neurobiological process under typical circumstances must first be characterized before examining how exposure to an adverse environment alters that trajectory. This approach has been utilized less frequently than would be expected in the literature on childhood adversity.

Recent work from Nim Tottenham's lab on functional connectivity between the amygdala and medial prefrontal cortex (mPFC) highlights the utility of this strategy. In an initial study, Gee, Humphreys, et al. (2013) demonstrated age-related changes in amygdala–mPFC functional connectivity in a typically developing sample of children during a task involving passive viewing of fearful and neutral faces. Specifically, they observed a developmental shift from a pattern of positive amygdala–mPFC functional connectivity during early and middle childhood to a pattern of negative connectivity (i.e., higher mPFC activity, lower amygdala activity) beginning in the prepubertal period and continuing throughout adolescence (Gee, Humphreys, et al., 2013). Next, they examined how exposure to institutional rearing in infancy influenced these age-related changes, documenting a more mature pattern of negative functional connectivity among young children with a history of institutionalization (Gee, Gabard-Durnam, et al., 2013).

Utilizing this type of approach is important not only to advance knowledge of developmental mechanisms underlying childhood adversity–psychopathology associations but also to leverage research on adverse environmental experiences to inform our understanding of typical development. Specifically, as frequently argued by Cicchetti (Cicchetti & Toth, 2009), research on atypical or aberrant developmental processes can provide a window into typical development not available through other means. This is particularly relevant in studies of some forms of childhood adversity that involve an *absence* of expected inputs from the environment, such as institutional rearing and child neglect (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). Examining the developmental consequences associated with deprivation in a particular type of input from the environment (e.g., the presence of an attachment figure, exposure to complex language) can provide insights into the types of environmental inputs that are required for a system or set of competencies to develop normally.

Evidence on the developmental trajectories of children raised in institutional settings provides an illustrative example. Institutions for abandoned and orphaned children vary widely, but a common feature across them is the absence of an attachment figure who provides sensitive and responsive care for each child (Smyke et al., 2007; Tottenham, 2012; Zeanah et al., 2003). Developmental research on children raised in institutional settings has

provided ample evidence about the importance of the attachment relationship in early development for shaping numerous aspects of development. Unsurprisingly, most children raised in institutions fail to develop a secure attachment relationship to a caregiver; this is particularly true if children remain in institutional care past the age of 2 years (Smyke, Zeanah, Fox, Nelson, & Guthrie, 2010; Zeanah, Smyke, Koga, Carlson, & The Bucharest Early Intervention Project Core Group, 2005). Children reared in institutional settings also exhibit social skills deficits, delays in language development, lasting disruptions in executive functioning skills, decrements in IQ, and atypical patterns of emotional processing (Almas et al., 2012; Bos, Fox, Zeanah, & Nelson, 2009; Nelson et al., 2007; Tibu et al., 2016; Tottenham et al., 2011; Windsor et al., 2011). Institutional rearing also has wide-ranging impacts on patterns of brain development, including neural structure and function (Gee et al., 2013; McLaughlin, Fox, Zeanah, & Nelson, 2011; McLaughlin, Sheridan, Winter, et al., 2014; Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012; Tottenham et al., 2011). Although children raised in institutional settings often experience deprivation in environmental inputs of many kinds, it is likely that the absence of a primary attachment figure in early development explains many of the downstream consequences of institutionalization on developmental outcomes. Indeed, recent evidence suggests that disruptions in attachment may be a causal mechanism linking institutional rearing with the onset of anxiety and depression in children. Specifically, in a randomized controlled trial of foster care as an intervention for orphaned children in Romania, improvements in attachment security were a mechanism underlying the preventive effects of the intervention on the onset of anxiety and depression in children (McLaughlin, Zeanah, Fox, & Nelson, 2012). By examining the developmental consequences of the absence of an expected input from the environment—namely, the presence of a primary attachment figure—studies of institutional rearing provide strong evidence for the centrality of the early attachment relationship in shaping numerous aspects of development.

**Sensitive Periods—**The integration of studies on typical and atypical development may be particularly useful in the identification of sensitive periods. Developmental psychopathology emphasizes the cumulative and hierarchical nature of development (Gottlieb, 1991a, 1991b; Sroufe, 2009; Sroufe, Egeland, & Kreutzer, 1990; Werner & Kaplan, 1963). Learning and acquisition of competencies at one point in development provide the scaffolding upon which subsequent skills and competencies are built, such that capabilities from previous periods are consolidated and reorganized in a dynamic, unfolding process across time. The primary developmental tasks occurring at the time of exposure to a risk factor are thought to be the most likely to be interrupted or disrupted by the experience. Developmental deviations from earlier periods are then carried forward and have consequences for children's ability to successfully accomplish developmental tasks in a later period (Cicchetti & Toth, 1998; Sroufe, 1997). In other words, early experiences constrain future learning of patterns or associations that represent departures from those that were previously learned (Kuhl, 2004).

This concept points to a critical area for future research on childhood adversity involving the identification of sensitive periods of emotional, cognitive, social, and neurobiological development when inputs from the environment are particularly influential. Sensitive periods

have been identified both in sensory development and in the development of complex social–cognitive skills, including language (Hensch, 2005; Kuhl, 2004). Emerging evidence from cognitive neuroscience also suggests the presence of developmental periods when specific regions of the brain are most sensitive to the effects of stress and adversity (Andersen et al., 2008). However, identification of sensitive periods has remained elusive in other domains of emotional and social development, potentially reflecting the fact that sensitive periods exist for fewer processes in these domains. However, determining how anomalous or atypical environmental inputs influence developmental processes differently based on the timing of exposure provides a unique opportunity to identify sensitive periods in development; in this way, research on adverse environments can inform our understanding of typical development by highlighting the environmental inputs that are necessary to foster adaptive development.

Identifying sensitive periods of emotional and social development requires detailed information on the timing of exposure to atypical or adverse environments, which is challenging to measure. To date, studies of institutional rearing have provided the best opportunity for studying sensitive periods in human emotional and social development, as it is straightforward to determine the precise period during which the child lived in the institutional setting. Studies of institutional rearing have identified a sensitive period for the development of a secure attachment relationship at around 2 years of age; the majority of children placed into stable family care before that time ultimately develop secure attachments to a caregiver, whereas the majority of children placed after 2 years fail to develop secure attachments (Smyke et al., 2010). Of interest, a sensitive period occurring around 2 years of age has also been identified for other domains, including reactivity of the autonomic nervous system and hypothalamic-pituitary-adrenal (HPA) axis to the environment and a neural marker of affective style (i.e., frontal electroencephalogram asymmetry; McLaughlin et al., 2011; McLaughlin, Sheridan, et al., 2015), suggesting the importance of the early attachment relationship in shaping downstream aspects of emotional and neurobiological development.

The second concrete recommendation for future research is *to integrate studies of typical development with those focused on understanding the impact of childhood adversity*; in particular, *research that can shed light on sensitive periods in emotional, social, cognitive, and neurobiological development is needed*. Identifying the developmental processes that are disrupted by exposure to particular types of adverse environments will be facilitated by first characterizing the typical developmental trajectories of the processes in question. In turn, studies of atypical or adverse environments should be leveraged to inform our understanding of the types of environmental inputs that are required—and *when*—for particular systems to develop normally. Given the inherent problems in retrospective assessment of timing of exposure to particular environmental experiences, longitudinal studies with repeated measurements of environmental experience and acquisition of developmental competencies are likely to be most informative. Alternatively, the occurrence of exogenous events like natural disasters, terrorist attacks, and changes in policies or the availability of resources (e.g., the opening of the casino on a Native American reservation; Costello, Compton, Keeler, & Angold, 2003) provides additional opportunities to study sensitive periods of development. Identifying sensitive periods is likely to yield critical insights into the points in

development when particular capabilities are most likely to be influenced by environmental experience, an issue of central importance for understanding both typical and atypical development. Such information can be leveraged to inform decisions about the points in time when psychosocial interventions for children exposed to adversity are likely to be maximally efficacious.

## Explaining Multifinality

The principle of multifinality is central to developmental psychopathology (Cicchetti, 1993). Multifinality refers to the process by which the same risk and/or protective factors may ultimately lead to different developmental outcomes (Cicchetti & Rogosch, 1996). It is has been repeatedly demonstrated that most forms of childhood adversity are associated with elevated risk for the onset of virtually all commonly occurring mental disorders (Green et al., 2010; McLaughlin, Green, et al., 2012). As noted earlier, recent evidence suggests that child maltreatment is associated with a latent liability for psychopathology that explains entirely the associations of maltreatment with specific mental disorders (Caspi et al., 2014; Keyes et al., 2012). However, the mechanisms that explain how child maltreatment, or other forms of adversity, influence a generalized liability to psychopathology have not been specified. To date, there have been few attempts to articulate a model explaining how childhood adversity leads to the diversity of mental disorders with which it is associated (i.e., multifinality). What are the mechanisms that explain this generalized vulnerability to psychopathology arising from adverse early experiences? Are these mechanisms shared across multiple forms of childhood adversity, or are they specific to particular types of adverse experience?

Identifying general versus specific mechanisms will require changes in the way we conceptualize and measure childhood adversity. Prior research has followed one of two strategies. The first involves studying individual types of childhood adversity, such as parental death, physical abuse, neglect, or poverty (Chase-Lansdale, Cherlin, & Kiernan, 1995; Dubowitz, Papas, Black, & Starr, 2002; Fristad, Jedel, Weller, & Weller, 1993; Mullen, Martin, Anderson, Romans, & Herbison, 1993; Noble, McCandliss, & Farah, 2007; Wolfe, Sas, & Wekerle, 1994). However, most individuals exposed to childhood adversity have experienced multiple adverse experiences (Dong et al., 2004; Finkelhor, Ormrod, & Turner, 2007; Green et al., 2010; McLaughlin, Green, et al., 2012). This presents challenges for studies focusing on a single type of adversity, as it is unclear if any observed associations represent the downstream effects of the focal adversity in question (e.g., poverty) or the consequences of other co-occurring experiences (e.g., exposure to violence) that might have different developmental consequences. Increasing recognition of the co-occurring nature of adverse childhood experiences has resulted in a shift from focusing on single types of adversity to examining the associations between *number* of adverse childhood experiences and developmental outcomes—the core strategy of the ACE approach (Arata, Langhinrichsen-Roling, Bowers, & O'Brien, 2007; Dube et al., 2003; Edwards et al., 2003; Evans et al., 2013). There has been a proliferation of research utilizing this approach in recent years, and it has proved useful in documenting the importance of childhood adversity as a risk factor for a wide range of negative mental health outcomes. However, this approach implicitly assumes that very different kinds of experiences ranging from violence exposure

to material deprivation (e.g., food insecurity) to parental loss influence psychopathology through similar mechanisms. Although there is likely to be some overlap in the mechanisms linking different forms of adversity to psychopathology, the count approach oversimplifies the boundaries between distinct types of environmental experience that may have unique developmental consequences.

An alternative approach that is likely to meet with more success involves identifying dimensions of environmental experience that underlie multiple forms of adversity and are likely to influence development in similar ways. In recent work, my colleague Margaret Sheridan and I have proposed two such dimensions that cut across multiple forms of adversity: threat and deprivation (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). *Threat* involves exposure to events involving harm or threat of harm, consistent with the definition of trauma in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013). Threat is a central dimension underlying multiple commonly studied forms of adversity, including physical abuse, sexual abuse, some forms of emotional abuse (i.e., that involve threats of physical violence and coercion), exposure to domestic violence, and other forms of violent victimization in home, school, or community settings. *Deprivation*, in contrast, involves the absence of expected cognitive and social inputs from the environmental stimuli, resulting in reduced opportunities for learning. Deprivation in expected environmental inputs is common to multiple forms of adversity including emotional and physical neglect, institutional rearing, and poverty. Critically, we do not propose that exposure to deprivation and threat *occurs* independently for children, as these experiences are highly co-occurring, or that these are the only important dimensions of experience involved in childhood adversity. Instead we propose, first, that these are two important dimensions that can be *measured* separately and, second, that the mechanisms linking these experiences to the onset of psychopathology are likely to be at least partially distinct (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). I describe some of these key mechanisms in the transdiagnostic model presented later. Recently, others have argued for the importance taking this type of dimensional approach as well (Hamby & Grych, 2013; Humphreys & Zeanah, 2015).

Specific recommendations are for future research to (a) *identify key dimensions of environmental experience* that might differentially influence developmental outcomes and (b) *measure multiple such dimensions in studies of childhood adversity* to distinguish between general and specific underlying mechanisms linking different forms of adversity to psychopathology. Fine-grained measurement of the dimensions of threat and deprivation has often not been conducted within the same study. Studies focusing on specific types of exposure (e.g., abuse) without measuring or adjusting for co-occurring exposures (e.g., neglect) are unable to distinguish between common and specific mechanisms linking different dimensions of adverse experiences to psychopathology. The only way to determine whether such specificity exists is to measure and model these dimensions of experience together in future studies.

## Characterizing the Interplay of Risk and Protective Factors

Although psychopathology is common among children exposed to a wide range of adverse environments, many children exhibit adaptation and resilience following adversity (Masten, 2001; Masten, Best, & Garmezy, 1990). For example, studies of resilience suggest that children who have a positive relationship with a caring and competent adult; are good at learning, problem solving, and self-regulation; are socially engaging; and have positive self-image are more likely to exhibit positive adaptation after exposure to adversity than children without these characteristics (Luthar, Cicchetti, & Becker, 2000; Masten, 2001; Masten et al., 1990). However, in contrast to the consistent pattern of associations between childhood adversity and psychopathology, evidence for protective factors varies widely across studies, and in most cases children exposed to adversity exhibit adaptive functioning in some domains but not others; even within a single domain, children may be functioning well at one point in time but not at others (Luthar et al., 2000). This is not surprising given that the degree to which a particular factor is protective depends heavily upon context, including the specific risk factors with which it is interacting (Cicchetti & Lynch, 1993; Sameroff, Gutman, & Peck, 2003). For example, authoritative parenting has been shown to be associated with adaptive outcomes for children raised in stable contexts that are largely free of significant adversity (Steinberg, Elmen, & Mounts, 1989; Steinberg, Lamborn, Dornbusch, & Darling, 1992; Steinberg, Mounts, Lamborn, & Dornbusch, 1991); in contrast, authoritarian parenting appears to be protective for children being raised in environments characterized by low resources and/or high degrees of violence and other threats (Flouri, 2007; Gonzales, Cauce, Friedman, & Mason, 1996). The degree to which variation in specific genetic polymorphisms moderates the impact of childhood adversity on development outcomes is also highly variable across studies; although genetic variation clearly contributes to developmental trajectories of adaptation and maladaptation following childhood adversity, this topic has been reviewed extensively elsewhere (Heim & Binder, 2012; McCrory, De Brito, & Viding, 2010; Uher & McGuffin, 2010) and is not discussed further. This complexity has contributed to the widely variable findings regarding protective factors and resilience.

Progress in identifying protective factors that buffer children from maladaptive outcomes following childhood adversity might be achieved by shifting the focus from downstream outcomes to more proximal mechanisms known to underlie the relationship between adverse childhood experiences and psychopathology. Research on resiliency has often focused on distal outcomes, such as the absence of psychopathology, the presence of high-quality peer relationships, or good academic performance as markers of adaptive functioning in children with exposure to adversity (Bolger, Patterson, & Kupersmidt, 1999; Collishaw et al., 2007; Fergusson & Lynskey, 1996; Luthar, 1991). Just as there are numerous mechanisms through which exposure to adverse environments lead to psychopathology and other downstream outcomes, there are likely to be a wide range of mechanisms through which protective factors buffer children from maladaptation following childhood adversity. Indeed, modern conceptualizations of resilience describe it as a developmental process that unfolds over time as an ongoing transaction between a child and the multiple contexts in which he or she is embedded (Luthar et al., 2000). Rather than examining protective factors that buffer children from developing psychopathology following adverse childhood experiences, an alternative

approach is to focus on factors that moderate the association of childhood adversity with the developmental processes that serve as mechanisms linking adversity with psychopathology (e.g., emotion regulation, executive functioning) or that moderate the link between these developmental processes and the onset of psychopathology. Deconstructing the pathways linking childhood adversity to psychopathology allows moderators to be examined separately at different stages of these pathways and may yield greater information about *how* protective factors ultimately exert their effects on downstream outcomes, including psychopathology.

Accordingly, a fourth recommendation is that future research should focus on identifying protective factors that buffer children from the negative consequences of adversity at two levels: (a) *factors that modify the association between childhood adversity and the maladaptive patterns of emotional, cognitive, social, and neurobiological development that serve as intermediate phenotypes linking adversity with psychopathology*; and (b) *factors that moderate the influence of intermediate phenotypes on the emergence of psychopathology*, leading to divergent trajectories of adaptation across children. To understand resilience, we first need to understand the developmental processes that are disrupted following exposure to adversity and how certain characteristics either prevent or compensate for those developmental disruptions or reduce their impact on risk for psychopathology.

## A TRANSDIAGNOSTIC MODEL OF CHILDHOOD ADVERSITY AND PSYCHOPATHOLOGY

The remainder of the article outlines a transdiagnostic model of mechanisms linking childhood adversity with youth psychopathology. Two core developmental mechanisms are proposed that, in part, explain patterns of multifinality: emotional processing and executive functioning. The model builds on a framework described by Nolen-Hoeksema and Watkins (2011) for identifying transdiagnostic processes. Of importance, the model is not intended to be comprehensive in delineating all mechanisms linking childhood adversity with psychopathology but rather focuses on two candidate mechanisms linking childhood adversity to multiple forms of psychopathology. At the same time, these mechanisms are also specific in that each is most likely to emerge following exposure to specific dimensions of adverse early experience. The model is specific with regard to the underlying dimensions of adverse experience considered and identifies several key moderators that might explain divergent developmental trajectories among children following exposure to adversity. Future research is needed to expand this framework to incorporate other key dimensions of the adverse environmental experience, developmental mechanisms linking those dimensions of adversity with psychopathology, and moderators of those associations.

### Distal Risk Factors

Within the proposed model, core dimensions of environmental experience that underlie multiple forms of adversity are conceptualized as distal risk factors for psychopathology. Specifically, experiences of threat and deprivation constitute the first component of the proposed transdiagnostic model of childhood adversity and psychopathology (see Figure 1).

Experiences of threat and deprivation meet each of Nolen-Hoeksema and Watkins's (2011) criteria for a distal risk factor. They represent environmental conditions largely outside the control of the child that are linked to the onset of psychopathology only through intervening causal mechanisms that represent more proximal risk factors. Although they are probabilistically related to psychopathology, exposure to threat and deprivation do not invariably lead to mental disorders. These experiences influence proximal risk factors primarily through learning mechanisms that ultimately shape patterns of information processing, emotional responses to the environment, and higher order control processes that influence both cognitive and emotional processing.

## Proximal Risk Factors

The developmental processes that are altered following exposure to adverse environmental experiences represent proximal risk factors, or intermediate phenotypes, linking them to the onset of psychopathology. These proximal risk factors represent the second component of the proposed transdiagnostic model. Nolen-Hoeksema and Watkins (2011) argued that proximal risk factors are within-person factors that mediate the relationship between distal risk factors—including aspects of environmental context that are difficult to modify, such as childhood adversity—and the emergence of psychopathology. Proximal risk factors directly influence symptoms and are temporally closer to symptom onset and often easier to modify than distal risk factors (Nolen-Hoeksema & Watkins, 2011). Identifying modifiable within-person factors that link adverse environmental experiences with the onset of symptoms is the key to developing interventions to prevent the onset of psychopathology in children who have experienced adversity.

The model includes two primary domains of proximal risk factors: emotional processing and executive functioning. Emotional processing refers to information processing of emotional stimuli (e.g., attention, memory), emotional reactivity, and both automatic (e.g., habituation, fear extinction) and effortful (e.g., cognitive reappraisal) forms of emotion regulation. These processes all represent responses to emotional stimuli, and many involve interactions of cognition with emotion. Executive functions comprise a set of cognitive processes that support the ability to learn new knowledge and skills; hold in mind goals and information; and create and execute complex, future-oriented plans. Executive functioning comprises the ability to hold information in mind and focus on currently relevant information (working memory), inhibit actions and information not currently relevant (inhibition), and switch flexibly between representations or goals (cognitive flexibility; Miyake & Friedman, 2012; Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001). Together these skills allow the creation and execution of future oriented plans and the inhibition of behaviors that do not serve these plans, providing the foundation for healthy decision making and self-regulation. Many of the diverse mechanisms linking childhood adversity to psychopathology are subsumed within these two broad domains.

**Emotional processing—**Stable patterns of emotional processing, emotional responding to the environment, and emotion regulation represent the first core domain of proximal risk factors. Experiences of uncontrollable threat are associated with strong learning of specific contingencies and overgeneralization of that learning to novel contexts, which facilitates the

processing of salient emotional cues in the environment (e.g., biased attention to threat). Given the importance of quickly identifying potential threats in the environment for children growing up in environments characterized by legitimate danger, these learning processes should produce information processing biases that promote rapid identification of potential threats. Indeed, evidence suggests that children with abuse histories—an environment characterized by high levels of threat—exhibit attention biases toward facial displays of anger, identify anger with little perceptual information, have difficulty disengaging from angry faces, and display anticipatory monitoring of the environment following interpersonal displays of anger (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003; Pollak, Vardi, Putzer Bechner, & Curtin, 2005; Shackman, Shackman, & Pollak, 2007). Given the relevance of anger as a signal of potential threat, these findings suggest that exposure to threatening environments results in stable patterns of information processing that facilitate threat identification and maintenance of attention to threat cues. These attention biases are specific to children who have experienced violence; for example, children who have been neglected (i.e., an environment characterized by deprivation in social and cognitive inputs) experience difficulty discriminating facial expressions of emotion but do not exhibit attention biases toward threat (Pollak, Klorman, Thatcher, & Cicchetti, 2001; Pollak et al., 2005). In addition to attention biases, children who have been the victims of violence are also more likely to generate attributions of hostility to others in socially ambiguous situations (Dodge, Bates, & Pettit, 1990; Dodge, Pettit, Bates, & Valente, 1995; Weiss, Dodge, Bates, & Petit, 1992), a pattern of social information processing tuned to be overly sensitive to potential threats in the environment. Finally, some evidence suggests that exposure to threatening environments is associated with memory biases for overgeneral autobiographical memories in both children and adults (Crane et al., 2014; Williams et al., 2007).

Children with trauma histories also exhibit meaningful differences in patterns of emotional responding that are consistent with these patterns of information processing. For example, children who have experienced interpersonal violence exhibit greater activation in the amygdala and other nodes of the salience network (e.g., anterior insula, putamen, thalamus) to a wide range of negative emotional stimuli (McCrory et al., 2013; McCrory et al., 2011; McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015), suggesting heightened salience of information that could predict threat. These findings build on earlier work using evoked response potentials documenting amplified neural response to angry faces in children who were physically abused (Pollak, Cicchetti, Klorman, & Brumaghim, 1997; Pollak et al., 2001) and suggests that exposure to threatening experiences heightens the salience of negative emotional information, due to the potential relevance for detecting novel threats. Heightened amygdala response to negative emotional cues could also reflect fear learning processes, whereby previously neutral stimuli that have become associated with traumatic events begin to elicit conditioned fear responses, or the result of deficits in automatic emotion regulation processes like fear extinction and habituation, which are mediated through connections between the ventromedial prefrontal cortex and amygdala. Recent findings of poor resting-state functional connectivity between the ventromedial prefrontal cortex and amygdala among female adolescents with abuse histories provide some evidence for this latter pathway (Herringa et al., 2013).

In addition to heightened neural responses in regions involved in salience processing, consistent associations between exposure to threatening environments and elevations in self-reported emotional reactivity to the environment have been observed in our lab and elsewhere (Glaser, Van Os, Portegijs, & Myin-Germeys, 2006; Heleniak, Jenness, Van Der Stoep, McCauley, & McLaughlin, in press; McLaughlin, Kubzansky et al., 2010). Atypical physiological responses to emotional cues have also been documented consistently among children who have experienced trauma, although the specific pattern of findings has varied across studies depending on the specific physiological measures and emotion eliciting paradigms employed. We recently applied a theoretical model drawn from social psychology on adaptive and maladaptive responses to stress to examine physiological responses to stress among maltreated youths. We observed a pattern of increased vascular resistance and blunted cardiac output reactivity among youths who had been physically or sexually abused relative to participants with no history of violence exposure (McLaughlin, Sheridan, Alves, & Mendes, 2014). This pattern of autonomic nervous system reactivity reflects an inefficient cardiovascular response to stress that has been shown in numerous studies to occur when individuals are in a state of heightened threat and is associated with threat appraisals and maladaptive cognitive and behavioral responses to stress (Jamieson, Mendes, Blackstock, & Schmader, 2010; Jamieson, Nock, & Mendes, 2012; Mendes, Blascovich, Major, & Seery, 2001; Mendes, Major, McCoy, & Blascovich, 2008). Using data from a large population-based cohort of adolescents, we recently replicated the association between childhood trauma exposure and blunted cardiac output reactivity during acute stress (Heleniak, Riese, Ormel, & McLaughlin, 2016).

Together, converging evidence across multiple levels of analysis indicates that exposure to trauma is associated with a persistent pattern of information processing involving biased attention toward potential threats in the environment, heightened neural and subjective responses to negative emotional cues, and a pattern of autonomic nervous system reactivity consistent with heightened threat perception. This heightened reactivity to negative emotional cues may make it more difficult for children who have been exposed to threatening environments to regulate emotional responses. Indeed, a recent study from my lab found that when trying to regulate emotional responses using cognitive reappraisal, children who had been abused recruited regions of the prefrontal cortex involved in effortful control to a greater degree than children who had never experienced violence (McLaughlin, Peverill, et al., 2015). This pattern suggests that attempts to modulate emotional responses to negative cues require more cognitive resources for children with abuse histories, meaning that effective regulation may break down more easily in the face of stress. Evidence that the negative emotional effects of stressful events are heightened among those with maltreatment histories is consistent with this possibility (Glaser et al., 2006; McLaughlin, Conron, et al., 2010).

In addition to alterations in patterns of emotional reactivity to environmental cues, child trauma has been associated with maladaptive patterns of responding to distress. For example, exposure to threatening environments early in development is associated with habitual engagement in rumination, a response style characterized by passive focus on feelings of distress along with their causes and consequences without attempts to actively resolve the causes of distress (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). High

reliance on rumination as a strategy for responding to distress has been observed in adolescents and adults who were abused as children (Conway, Mendelson, Giannopoulos, Csank, & Holm, 2005; Heleniak et al., in press; Sarin & Nolen-Hoeksema, 2010), adolescents who experienced victimization by peers (McLaughlin, Hatzenbuehler, & Hilt, 2009), and both adolescents and adults exposed to a wide range of negative life events (McLaughlin & Hatzenbuehler, 2009; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013), although the latter findings are not specific to threat per se.

Although evidence for disruptions in emotional processing come primarily from studies examining children exposed to environments characterized by high degrees of threat, deprived environments are also likely to have downstream effects on emotional development that are at least partially unique from those associated with threat. As noted previously, children who have been neglected experience difficulties discriminating facial displays of emotion (Pollak et al., 2001; Pollak et al., 2005), although some studies of neglected children have found few differences in neural responses to facial emotion in early childhood (Moulson, Fox, Zeanah, & Nelson, 2009; Slopen, McLaughlin, Fox, Zeanah, & Nelson, 2012). However, recent work suggests that children raised in deprived early environments exhibit elevated amygdala response to facial emotion and a mature pattern of functional connectivity between the amygdala and mPFC during emotional-processing tasks (Gee et al., 2013; Tottenham et al., 2011). Finally, children who were neglected or raised in deprived institutions tend to exhibit blunted physiological responses to stress, including in the autonomic nervous system and HPA axis (Gunnar, Frenn, Wewerka, & Van Ryzin, 2009; McLaughlin, Sheridan, et al., 2015).

Much of the existing work on childhood adversity and emotional responding has focused on responses to negative emotional cues. However, a growing body of evidence also suggests that responses to appetitive and rewarding cues are disrupted in children exposed to adversity. For example, children raised in deprived early environments exhibit blunted ventral striatal response to the anticipation of reward (Mehta et al., 2010), and a similar pattern has been observed in a sample of adults exposed to abuse during childhood (Dillon et al., 2009). In a recent study, an increase in ventral striatum response to happy emotional faces occurred from childhood to adolescence in typically developing children but not in children reared in deprived institutions (Goff et al., 2013). In recent work in our lab, we have also observed blunted reward learning among children exposed to institutional rearing (Sheridan, McLaughlin, et al., 2016). Although the mechanisms underlying the link between diverse forms of childhood adversity and responsiveness to reward have yet to be clearly identified, it has been suggested that repeated activation of the HPA axis in early childhood can attenuate expression of brain-derived neurotrophic factor, which in turn regulates the mesolimbic dopamine system that underlies reward learning (Goff & Tottenham, 2014). These reductions in brain-derived neurotrophic factor expression may contribute to a pattern of blunted ventral striatum response to reward anticipation or receipt. Alternatively, given the central role of the mesolimbic dopamine system in attachment-related behavior (Strathearn, 2011), the absence or unpredictability of an attachment figure in early development may reduce opportunities for learning about the rewarding nature of affiliative interactions and social bonds; the absence of this type of stimulus-reward learning early in development, when sensitive and responsive caregiving from a primary attachment figure is

an expected environmental input, may ultimately contribute to biased processing of rewarding stimuli later in development. If social interactions in early life are either absent or unrewarding, expectations about the hedonic value of social relationships and other types of rewards might be altered in the long term, culminating in attenuated responsiveness to anticipation of reward. Future research is needed to identify the precise mechanisms through which adverse early environments ultimately shape reward learning and responses to rewarding stimuli.

**Links between emotional processing and psychopathology—**An extensive and growing body of work suggests that disruptions in emotional processing, emotional responding, and emotion regulation represent transdiagnostic factors associated with virtually all commonly occurring forms of psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Specifically, attention biases to threat and overgeneral autobiographical memory biases have been linked to anxiety and depression, respectively, in numerous studies (Bar-Haim, Lamy, Bakermans-Kranenburgh, Pergamin, & Van Ijzendoorn, 2007; Williams et al., 2007), and attributions of hostility and other social information processing biases associated with trauma exposure are associated with risk for the onset of conduct problems and aggression (Dodge et al., 1990; Dodge et al., 1995; Weiss et al., 1992). Heightened emotional responses to negative environmental cues are associated with both internalizing and externalizing psychopathology in laboratory-based paradigms examining self-reported emotional and physiological responses to emotional stimuli (Boyce et al., 2001; Carthy, Horesh, Apter, Edge, & Gross, 2010; Hankin, Badanes, Abela, & Watamura, 2010; McLaughlin, Kubzansky, et al., 2010; McLaughlin, Sheridan, Alves, et al., 2014; Rao, Hammen, Ortiz, Chen, & Poland, 2008), fMRI studies examining neural response to facial emotion (Sebastian et al., 2012; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007; Stein, Simmons, Feinstein, & Paulus, 2007; Suslow et al., 2010; Thomas et al., 2001), and experience sampling studies that measure emotional responses in real world situations (Myin-Germeys et al., 2003; Silk, Steinberg, & Morris, 2003). Habitual engagement in rumination has also been linked to heightened risk for anxiety, depression, eating disorders, and problematic substance use (McLaughlin & Nolen-Hoeksema, 2011; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Together, evidence from numerous studies examining emotional processing at multiple levels of analysis suggests that disruptions in emotional processing are a key transdiagnostic factor in psychopathology that may explain patterns of multifinality following exposure to threatening early environments.

**Executive functioning—**Disruptions in executive functioning represent the second key proximal risk factor in the model. A growing body of evidence suggests that environmental deprivation is associated with lasting alterations in executive functioning skills. Poor executive functioning—including problems with working memory, inhibitory control, planning ability, and cognitive flexibility—has consistently been documented among children raised in deprived environments ranging from institutional settings to low socioeconomic status (SES) families. Children raised in institutional settings exhibit a range of deficits in cognitive functions including general intellectual ability (Nelson et al., 2007; O'Connor, Rutter, Beckett, Keaveney, & Kreppner, 2000), expressive and receptive language (Albers, Johnson, Hostetter, Iverson, & Miller, 1997; Windsor et al., 2011), and executive

function skills (Bos et al., 2009; Tibu et al., 2016). In contrast to other domains of cognitive ability, however, deficits in executive functioning and marked elevations in the prevalence of attention-deficit/hyperactivity disorder (ADHD)—which is characterized by executive functioning problems—are persistent over time even after placement into a stable family environment (Bos et al., 2009; Tibu et al., 2016; Zeanah et al., 2009). Similar patterns of executive functioning deficits have also been observed among children raised in low SES families, including problems with working memory, inhibitory control, and cognitive flexibility (Blair, 2002; Farah et al., 2006; Noble et al., 2007; Noble, Norman, & Farah, 2005; Raver, Blair, Willoughby, & The Family life Project Key Investigators, 2013), as well as deficits in language abilities (Fernald, Marchman, & Weisleder, 2013; Weisleder & Fernald, 2013). Poor cognitive flexibility among children raised in low SES environments has been observed as early as infancy (Clearfield & Niman, 2012). Relative to children who have been abused, children exposed to neglect are at greater risk for cognitive deficits (Hildyard & Wolfe, 2002) similar to those observed in poverty and institutionalization (Dubowitz et al., 2002; Spratt et al., 2012).

The lateral PFC is recruited during a wide variety of executive functioning tasks, including working memory (Wager & Smith, 2003), inhibition (Aron, Robbins, & Poldrack, 2004), and cognitive flexibility (Rougier, Noelle, Braver, Cohen, & O'Reilly, 2005), and is one of the brain regions most centrally involved in executive functioning. In addition to exhibiting poor performance on executive functioning tasks, children from low SES families also have different patterns of lateral PFC recruitment during these tasks as compared to children from middle-class families (Kishiyama, Boyce, Jimenez, Perry, & Knight, 2009; Sheridan, Sarsour, Jutte, D'Esposito, & Boyce, 2012). A similar pattern of poor inhibitory control and altered lateral PFC recruitment during an inhibition task has also been observed in children raised in institutional settings (Mueller et al., 2010).

These studies provide some clues about where to look with regards to the types of environmental inputs that might be necessary for the development of adaptive executive functions. In particular, environmental inputs that are absent or atypical among children raised in institutional settings, as well as among children raised in poverty, are promising candidates. Institutional rearing is associated with an absence of environmental inputs of numerous kinds, including the presence of an attachment figure, variation in daily routines and activities, access to age-appropriate enriching cognitive stimulation from books, toys, and interactions with adults, and complex language exposure (Smyke et al., 2007; Zeanah et al., 2003). Some of these dimensions of environmental experience have also been shown to be deprived among children raised in poverty, including access to cognitively enriching activities—including access to books, toys, and puzzles; learning opportunities outside the home (e.g., museums) and within the context of the parent–child relationship (e.g., parental encouragement of learning colors, words, and numbers, reading to the child); and variation in environmental complexity and stimulation—as well as the amount and complexity of language input (Bradley, Convyn, Burchinal, McAdoo, & Coll, 2001; Bradley, Corwyn, McAdoo, & Coll, 2001; Dubowitz et al., 2002; Garrett, Ng'andu, & Ferron, 1994; Hart & Risley, 1995; Hoff, 2003; Linver, Brooks-Gunn, & Kohen, 2002).

Together, these distinct lines of research suggest that enriching cognitive activities and exposure to complex language might provide the scaffolding that children require to develop executive functions. Some indirect evidence supports this notion. For example, degree of environmental stimulation in the home and amount and quality of maternal language each predict the development of language skills in early childhood (Farah et al., 2008; Hoff, 2003), and children raised in both institutional settings and low SES families exhibit deficits in expressive and receptive language (Albers et al., 1997; Hoff, 2003; Noble et al., 2007; Noble et al., 2005; Windsor et al., 2011) in addition to problems with executive functioning skills. Moreover, a recent study found that atypical patterns of PFC activation during executive function tasks among children from low SES families is explained by degree of complex language exposure in the home (Sheridan et al., 2012). Finally, children raised in bilingual environments appear to have improved performance on executive function tasks (Carlson & Meltzoff, 2008). These findings suggest that the environmental inputs that are required for language development (i.e., complex language directed at the child) may also be critical for the development of executive function skills. Language provides an opportunity to develop multiple such skills ranging from working memory (e.g., holding in mind the first part of a sentence as you wait for the speaker to finish), inhibitory control (e.g., waiting your turn in a conversation), and cognitive flexibility (e.g., switching between grammatical and syntactic rules).

Lack of consistent rules, routines, structure, and parental scaffolding behaviors may be another mechanism explaining deficits in executive functioning among children from low SES families. This lack of environmental predictability is more common among low SES than middle-class families (Deater-Deckard, Chen, Wang, & Bell, 2012; Evans, Gonnella, Mareynyszyn, Gentile, & Salpekar, 2005; Evans & Wachs, 2009). The absence of consistent rules, routines, and contingencies in the environment may interfere with children's ability to learn abstract rules and to develop the capacity for self-regulation. Indeed, higher levels of parental scaffolding—or provision of support to allow the child to solve problems autonomously—has been prospectively linked with the development of better executive function skills in early childhood (Bernier, Carlson, & Whipple, 2010; Hammond, Muller, Carpendale, Bibok, & Liebermann-Finestone, 2012; Landry, Miller-Loncar, Smith, & Swank, 2002). These findings suggest that environmental unpredictability is an additional mechanism linking low SES environments to poor executive functioning in children. However, given the highly structured and routinized nature of most institutional settings, environmental unpredictability is an unlikely explanation for executive functioning deficits among institutionally reared children.

Deficits in executive functioning skills have sometimes been observed in children with exposure to trauma (DePrince, Weinzierl, & Combs, 2009; Mezzacappa, Kindlon, & Earls, 2001) as well as children with high levels of exposure to stressful life events (Hanson et al., 2012), although some studies have found associations between trauma exposure and working memory but not inhibition or cognitive flexibility (Augusti & Melinder, 2013). There are two possible explanations for these findings. First, for children exposed to threat, it may be that deficits in executive functions emerge primarily in emotional contexts, such that the heightened perceptual sensitivity and reactivity to emotional stimuli in children exposed to threat draws attention to emotional stimuli (Shackman et al., 2007), making it

more difficult to hold other stimuli in mind, effectively inhibit responses to emotional stimuli, or flexibly allocate attention to nonemotional stimuli. Indeed, in a recent study in my lab, we observed that exposure to trauma (both maltreatment and community violence) was associated with deficits in inhibitory control only in the context of emotional stimuli (i.e., a Stroop task involving emotional faces) and not when stimuli were neutral (i.e., shapes), and had no association with cognitive flexibility (Lambert, King, Monahan, & McLaughlin, 2016). In contrast, deprivation exposure was associated with deficits in inhibition to both neutral and emotional stimuli and poor cognitive flexibility. Although this suggests there may be specificity in the association of trauma exposure with executive functions, greater research is needed to understand these links. Second, studies examining exposure to trauma seldom measure indicies of deprivation, nor do they adjust for deprivation exposure (just as studies of deprivation rarely assess or control for trauma exposure). Disentangling the specific effects of these two types of experiences on executive functioning processes is a critical goal for future research.

**Links between executive functioning and psychopathology—**Executive functioning deficits are a central feature of ADHD (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Sergeant, Geurts, & Oosterlaan, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Problems with executive functions have also been observed in children with externalizing psychopathology, including conduct disorder and oppositional defiant disorder, even after accounting for comorbid ADHD (Hobson, Scott, & Rubia, 2011). They are also associated with elevated risk for the onset of substance use problems and other types of risky behavior (Crews & Boettiger, 2009; Patrick, Blair, & Maggs, 2008), including criminal behavior (Moffitt et al., 2011) and the likelihood of becoming incarcerated (Yechiam et al., 2008). Although executive functioning deficits figure less prominently in theoretical models of the etiology of internalizing psychopathology, when these deficits emerge in the context of emotional processing (e.g., poor inhibition of negative emotional information) they are more strongly linked to internalizing problems, including depression (Goeleven, De Raedt, Baert, & Koster, 2006; Joorman & Gotlib, 2010). Executive functioning deficits also contribute to other proximal risk factors, such as rumination (Joorman, 2006), that are well-established risk factors for depression and anxiety disorders. Patterns of executive functioning in childhood have lasting implications for health and development beyond effects on psychopathology. Recent work suggests that executive functioning measured in early childhood predicts a wide range of outcomes in adulthood in the domains of health, SES, and criminal behavior, over and above the effects of IQ (Moffitt et al., 2011).

## Mechanisms Linking Distal Risk Factors to Proximal Risk Factors

How do experiences of threat and deprivation come to influence proximal risk factors? Learning mechanisms are the most obvious pathways linking these experiences with changes in emotional processing and executive functioning, although other mechanisms (e.g., the development of stable beliefs and schemas) are also likely to play an important role. Specifically, the impact of threatening and deprived early environments on the development of patterns of emotional processing and emotional responding may be mediated, at least in part, through emotional learning pathways. The associative learning

mechanisms and neural circuitry underlying fear learning and reward learning have been well characterized in both animals and humans and reviewed elsewhere (Delgado, Olsson, & Phelps, 2006; Flagel et al., 2011; Johansen, Cain, Ostroff, & LeDoux, 2011; O'Doherty, 2004). Exposure to threatening or deprived environments early in development results in the presence (i.e., in the case of threats) or absence (i.e., in the case of deprivation) of opportunities for emotional learning; these learning experiences, in turn, have lasting downstream effects on emotional processing. Specifically, early learning histories can influence the salience of environmental stimuli as either potential threats or incentives, shape the magnitude of emotional responses to environmental stimuli—particularly those that represent either threat or reward, and alter motivation to avoid threats or pursue rewards. Thus, fear learning mechanisms and their downstream consequences explain, in part, the association of threatening environments with alterations in emotional processing (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). Similarly, the effects of deprived early environments on emotional processing are likely to be partially explained through reward learning pathways. Pathways linking threatening early environments to habitual patterns of responding to distress, such as rumination, may also involve learning mechanisms including both observational (e.g., modeling responses utilized by caregivers) and instrumental (e.g., reinforcement of passive responses to distress when emotional displays are met with dismissive or punishing reactions from caregivers) learning.

Learning mechanisms may also be a central mechanism in the association between deprived early environments and the development of executive functioning. In particular, deprived environments such as institutional rearing, neglect, and poverty are characterized by the absence of learning opportunities, which is thought to directly contribute to later difficulties with complex higher order cognition. Specifically, reduced opportunities for learning due to the absence of complex and varied stimulus-response contingencies or the presence of consistent rules, routines, and structures that allow children to learn concrete and abstract rules may influence the development of both cognitive and behavioral aspects of self-regulation.

## Moderators of the Link Between Distal and Proximal Risk Factors

Children vary markedly in their sensitivity to environmental context. Advances in theoretical conceptualizations of individual differences in sensitivity to context can be leveraged to understand variability in developmental processes among children exposed to adverse environments. A growing body of evidence suggests that certain characteristics make children particularly responsive to environmental influences; such factors confer not only vulnerability in the context of adverse environments but also benefits in the presence of supportive environments (Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2007; Belsky & Pluess, 2009; Boyce & Ellis, 2005; Ellis, Essex, & Boyce, 2005). Highly reactive temperament, vagal tone, and genetic polymorphisms that regulate the dopaminergic and serotonergic system have been identified as markers of plasticity and susceptibility to both negative and positive environmental influences (Belsky & Pluess, 2009). These plasticity markers represent potential moderators of the link between childhood adversity and disruptions in emotional processing and executive functioning.

Developmental timing of exposure to adversity also plays a meaningful role in moderating the impact of childhood adversity on emotional processing and executive functioning. For example, in recent work we have shown that early environmental deprivation has a particularly pronounced impact on the development of stress response systems during the first 2 years of life (McLaughlin et al., 2015). These findings suggest the possibility of an early sensitive period during which the environment exerts a disproportionate effect on the development of neurobiological systems that regulate responses to stress. As noted in the beginning of this article, additional research is needed to identify developmental periods of heightened plasticity in specific subdomains of emotional processing and executive functioning and to determine the degree to which disruptions in these domains vary as a function of the timing of exposure to childhood adversity.

### Moderators of Trajectories From Proximal Risk Factors to Psychopathology

A key component of Nolen-Hoeksema and Watkins's (2011) transdiagnostic model of psychopathology involves moderators that determine the specific type of psychopathology that someone with a particular proximal risk factor will develop. Specifically, their model argues that ongoing environmental context and neurobiological factors can moderate the impact of proximal risk factors on psychopathology by raising concerns or themes that are acted upon by proximal risk factors and by shaping responses to and altering the reinforcement value of particular types of stimuli. For example, the nature of ongoing environmental experiences might determine whether someone with an underlying vulnerability (e.g., neuroticism) develops anxiety or depression. Specifically, a person with high neuroticism who experiences a stressor involving a high degree of threat or danger (e.g., a mugging or a car accident) might develop an anxiety disorder, whereas a person with high neuroticism who experiences a loss (e.g., an unexpected death of a loved one) might develop major depression (Nolen-Hoeksema & Watkins, 2011). Neurobiological factors that influence the reinforcement value of certain stimuli (e.g., alcohol and other substances, food, social rejection) can also serve as moderators. For example, individual differences in rejection sensitivity might determine whether a child who is bullied develops an anxiety disorder. Although a review of these factors is beyond the scope of the current article, greater understanding of the role of ongoing environmental context as a moderator of the link between proximal risk factors and the emergence of psychopathology has relevance for research on childhood adversity. In particular, environmental factors that buffer against the emergence of psychopathology in children with disruptions in emotional processing and executive functioning can point to potential targets for preventive interventions for children exposed to adversity.

## CONCLUSION

Exposure to childhood adversity represents one of the most potent risk factors for the onset of psychopathology. Recognition of the strong and pervasive influence of childhood adversity on risk for psychopathology throughout the life course has generated a burgeoning field of research focused on understanding the links between adverse early experience, developmental processes, and mental health. This article provides recommendations for future research in this area (see Table 1). In particular, future research must develop and

utilize a consistent definition of childhood adversity across studies, as it is critical for the field to agree upon what the construct of childhood adversity represents and what types of experiences do and do not qualify. Progress in identifying developmental mechanisms linking childhood adversity to psychopathology requires integration of studies of typical development with those focused on childhood adversity in order to characterize how experiences of adversity disrupt developmental trajectories in emotion, cognition, social behavior, and the neural circuits that support these processes, as well as greater efforts to distinguish between distinct dimensions of adverse environmental experience that differentially influence these domains of development. Greater understanding of the developmental pathways linking childhood adversity to the onset of psychopathology can inform efforts to identify protective factors that buffer children from the negative consequences of adversity by allowing a shift in focus from downstream outcomes like psychopathology to specific developmental processes that serve as intermediate phenotypes (i.e., mechanisms) linking adversity with psychopathology.

Progress in these domains will generate clinically useful knowledge regarding the mechanisms that explain how childhood adversity is associated with a wide range of psychopathology outcomes (i.e., multifinality) and identify moderators that shape divergent trajectories following adverse childhood experiences. This knowledge can be leveraged to develop and refine empirically informed interventions to prevent the long-term consequences of adverse early environments on children's development. Greater understanding of modifiable developmental processes underlying the associations of diverse forms of childhood adversity with psychopathology will provide critical information regarding the mechanisms that should be specifically targeted by intervention. Determining whether these mechanisms are general or specific is essential, as it is unlikely that a one-size-fits-all approach to intervention will be effective for preventing the onset of psychopathology following all types of childhood adversity. Identifying processes that are disrupted following specific forms of adversity, but not others, will allow interventions to be tailored to address the developmental mechanisms that are most relevant for children exposed to particular types of adversity. Identification of moderators that buffer children either from disruptions in core developmental domains or from developing psychopathology in the presence of developmental disruptions—for example, among children with heightened emotional reactivity or poor executive functioning—will provide additional targets for intervention. Finally, uncovering sensitive periods when emotional, cognitive, and neurobiological processes are most likely to be influenced by the environment will provide key information about when interventions are most likely to be successful. Together, these advances will help the field to generate innovative new approaches for preventing the onset of psychopathology among children who have experienced adversity.

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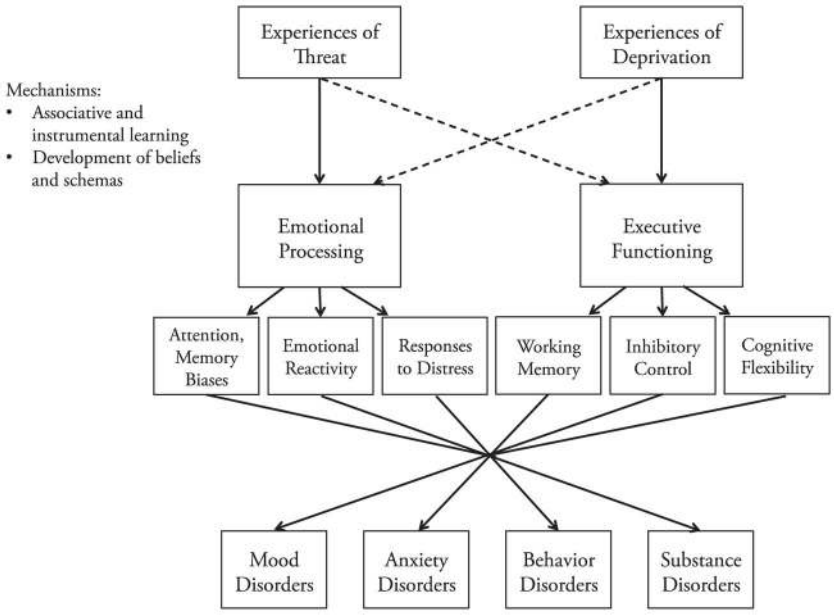


**FIGURE 1.**

A transdiagnostic model of childhood adversity and youth psychopathology. *Note*. Dashed lines represent pathways that are less well established and may represent indirect associations or associations that emerge only in interaction with other processes. For example, children exposed to threatening early environments may exhibit problems with working memory and inhibitory control only in emotionally arousing situations or when the stimuli to be remembered or to which a response must be inhibited are emotional in nature. Greater research is needed to understand these pathways and the conditions under which they operate.

**TABLE 1**

Recommendations for Future Research on Childhood Adversity and Youth Psychopathology

| |
|---|
| Develop and utilize a consistent definition of childhood adversity |
| Integrate studies of typical development with those focused on understanding the impact of childhood adversity; in particular, research that can shed light on sensitive periods in emotional, social, cognitive, and neurobiological development is needed |
| Identify developmental mechanisms linking adverse environmental experiences in childhood to the onset of multiple forms of psychopathology (i.e., mechanisms that explain multifinality) |
| Identify distinct dimensions of environmental experience that might differentially influence developmental mechanisms |
| Measure multiple dimensions of environmental experience in studies of childhood adversity to distinguish between common and specific underlying mechanisms linking different forms of adversity to psychopathology |
| Identify protective factors that buffer children from the negative consequences of adversity at two levels: (a) factors that modify the association between childhood adversity and the maladaptive patterns of emotional, cognitive, social, and neurobiological development that serve as intermediate phenotypes (i.e., mechanisms) linking adversity with psychopathology, and (b) factors that moderate the influence of intermediate phenotypes on the emergence of psychopathology, leading to divergent trajectories of adaptation across children |