Early Life Stress and Psychopathology

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Abstract and Keywords

Exposure to chronic or severe stressful life events during childhood and adolescence—frequently referred to as early life stress (ELS) or childhood adversity—has powerful and lasting associations with psychopathology across the life course. This chapter reviews the growing body of research on ELS and psychopathology across the life course, with a particular focus on the mechanisms that explain the strong associations between ELS and psychopathology. To address these questions, I review evidence on the links between ELS and psychopathology and highlight divergent conceptual models of ELS that advocate different approaches to uncovering these mechanisms. I end by addressing different approaches to the measurement and analysis of ELS that have emerged from these conceptual frameworks.

Keywords: stress, early life stress, childhood adversity, adverse childhood experience, development, psychopathology

Exposure to stressful life events has long been recognized as a risk factor for the onset of psychopathology. Over the past two decades, however, evidence from diverse scientific disciplines has demonstrated that certain types of stressful life events that occur during childhood and adolescence have particularly powerful and lasting associations with psychopathology across the life course (Afifi et al., 2008; Edwards, Holden, Felitti, & Anda, 2003; Green et al., 2010; MacMillan et al., 2001; McLaughlin et al., 2012). These experiences are frequently referred to as early life stress (ELS) or childhood adversity. This chapter reviews the growing body of research on ELS and its associations with psychopathology. What is ELS and how does it differ from normative experiences of stress? How is ELS related to psychopathology and, critically, what are the mechanisms that explain these associations? To address these questions, I review evidence on the links between ELS and psychopathology and highlight divergent conceptual models of ELS that advocate different approaches to uncovering these mechanisms. I end by

addressing different approaches to the measurement and analysis of ELS that have emerged from these conceptual frameworks.

Defining Early Life Stress

Despite the burgeoning interest in the links between ELS and psychopathology, there has been a surprising lack of consistency in the literature with regard to the definition and measurement of the construct. Until very recently, ELS was a construct in search of a definition. Even the terminology used to refer to ELS varies widely across studies. Some of the most commonly used terms other than ELS include childhood adversity, adverse childhood experiences, and household dysfunction. But to what does the construct of ELS or childhood adversity actually refer, and is it different from other types of childhood stressors? As I have noted elsewhere (McLaughlin, 2016), a definition of ELS has remained elusive because the construct is both difficult to define concretely but fairly obvious to most observers, making it an example of the classic standard of *you know it when you see it*. Recent efforts to define ELS and distinguish it from other types of stressors—particularly more normative forms of stress exposure—grew out of two related lines of inquiry.

First, extensive research had been conducted on normative stressors occurring during childhood and adolescence. Stress has been defined by Monroe (2008) as reflecting the ongoing adaptation required by environmental conditions that change over time. Within this broad perspective, stress is comprised of three components that interact with one another: (1) environmental conditions that require adaptation (i.e., stressors); (2) the psychological and neurobiological response of an organism to these changes in environmental conditions (i.e., the stress response); and (3) the ongoing transaction between an organism and the environment that unfolds across time (i.e., adaptation to stress) (Monroe, 2008). The environmental component of stress aligns most closely with the concept of ELS. Even in early studies of environmental stressors, it was recognized that stressful experiences occurring in childhood might have particular significance for mental health (Bifulco, Brown, & Adler, 1991; Bifulco, Brown, & Harris, 1987). Indeed, extensive research documents associations between exposure to stressors in childhood and adolescence and the subsequent onset of psychopathology (Grant et al., 2003, 2006; Grant, Compas, Thurm, & McMahon, 2004; Hammen, 2008). Within this literature, a variety of distinctions among different types of stressors have been utilized based on (a) temporal characteristics of the stressor (i.e., differentiating chronic stressors such as marital conflict from acute life events like a car accident) (Adrian & Hammen, 1993); (b) stressor severity, such that major stressors like parental divorce are distinguished from more minor events and hassles like failing a test (Grant, 2003); (c) the role of the child in contributing to the occurrence of the stressor, with independent events (i.e., those that the child played no role in generating, like a car accident) considered separately from dependent events that a child contributed to in some way, such as peer conflict (Rudolph

& Hammen, 1999); and (d) the source of stress. With regard to sources of stress, a variety of distinctions have been made, with interpersonal stressors frequently differentiated from those occurring in the academic domain or from other noninterpersonal forms of stress (Rudolph et al., 2000). Studies of childhood stress exposure and psychopathology typically assess a wide range of stressors, some of which would be considered to be forms of ELS (e.g., exposure to violence) and many of which would not (e.g., peer-related stressors; academic stressors; daily hassles). This body of work has informed recent attempts to define ELS and to distinguish experiences of adversity that reflect ELS from the wide range of stressors that have been studied in relation to developmental psychopathology.

A second body of work informing modern conceptualization of ELS emerged from the seminal Adverse Childhood Experiences (ACE) study (Anda et al., 2006; Felitti et al., 1998), which examined ELS as a determinant of adult physical and mental health. In many ways, the genesis of the modern construct of ELS began with this study. Before the ACE study, most work on ELS focused on specific, individual types of adverse experiences, such as parental divorce, death of a parent, sexual abuse, or poverty (Amato & Keith, 1991; Duncan, Brooks-Gunn, & Kato Klebanov, 1994; Molnar, Buka, & Kessler, 2001). Research on specific forms of ELS had evolved as relatively independent lines of inquiry until the ACE study, which considered numerous types of adversity—including abuse, neglect, parental psychopathology and substance use, parental loss, domestic violence, and parental criminal behavior—to be indicators of the same underlying construct, termed “adverse childhood experiences” or “household dysfunction” (Dube et al., 2001; Felitti et al., 1998). The study documented high levels of co-occurrence among these multiple forms of ELS and strong associations between ELS exposure and adult health (Dong et al., 2004; Edwards et al., 2003; Felitti et al., 1998). Although the findings of the ACE study provided the impetus for many recent studies of ELS, a concrete definition of “adverse childhood experience” or “household dysfunction” was never provided. According to the Centers for Disease Control and Prevention (CDC) website for the ACE study, the ACE score—a count of the total number of adversities experienced, is designed to assess “the total amount of stress experienced during childhood.”

I have recently proposed a definition of childhood adversity (or ELS) that builds on definitions of life stress (Monroe, 2008) and models of experience-expectant brain development (Baumrind, 1993; Fox, Levitt, & Nelson, 2010). Specifically, adversity (or ELS) refers to *environmental circumstances that are either serious (i.e., severe) or ongoing over time (i.e., chronic); are likely to require significant adaptation by an average child; and represent a deviation from the expectable environment* (McLaughlin, 2016). The expectable environment refers to a wide range of environmental inputs that the human brain expects to encounter in order to develop normally. Expected environmental inputs range from sensory and perceptual experiences (e.g., variation in patterned light information that is required for normal development of the visual system) to social experiences, such as the presence of a sensitive and responsive caregiver and exposure to language (Fox et al., 2010). Deviations from the expectable environment often take two primary forms: an absence of expected inputs (e.g., the absence of a primary caregiver or

limited exposure to complex language) 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) (Farah et al., 2008; Humphreys & Zeanah, 2015; McLaughlin & Sheridan, 2016; McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). These deviations reflecting either deprivation or threat can 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). This provides a reasonable working definition of ELS: 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 clear boundary conditions about the types of stressful experiences that are and are not reflective of ELS. The most obvious boundary condition involves the developmental timing of exposure—ELS must occur early in development. Most research on ELS has taken a fairly broad definition of what constitutes early life and includes events occurring during either childhood or adolescence. An additional boundary condition centers on the specific component of stress to which ELS refers. In the proposed definition, ELS refers to a specific event or ongoing conditions *in the environment*. ELS thus refers to environmental circumstances or events and not a child's response to those events or adaptation over time. Perhaps the most meaningful boundary condition involves the types of environmental circumstances that qualify as ELS. The proposed definition restricts ELS to environmental conditions that are likely to require significant adaptation by an average child—this means that not all childhood stressors qualify as ELS. Transient events or minor hassles should not qualify, but neither should major life events that do not represent a deviation from the expectable environment such as the death of a grandparent, enrolling in a new school, or experiencing the end of a close friendship. These are all examples of events that would qualify as meaningful stressors and could certainly influence mental health, but they are not examples of ELS. This boundary condition provides the clearest distinction between ELS and more broad definitions of stressors that occur during childhood or adolescence (Grant et al., 2003, 2004; Rudolph & Hammen, 1999; Rudolph et al., 2000). At the same time, this condition is the most difficult to define clearly, as there is no absolute rule or formula that can be used to distinguish circumstances requiring significant adaptation from those that are less severe or impactful. Nevertheless, a guiding principle is that ELS should only include events that are likely to have a meaningful and lasting impact on developmental processes for most children who experience them. More specifically, only environmental experiences that are likely to alter fundamental aspects of emotional, cognitive, social, or neurobiological development should qualify as ELS.

An unresolved issue for the field is whether the definition of ELS should be narrow or broad. For example, many population-based studies have included parental psychopathology and parental divorce as forms of ELS (Felitti et al., 1998; Green et al., 2010). Because psychopathology and divorce are common, consideration of any form of parental psychopathology or any type of divorce as a form of ELS results in a fairly broad

definition. 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 produce adverse situations that would require adaptation by most children (e.g., economic adversity, emotional abuse) as meeting the threshold for ELS. Despite some unresolved questions regarding the scope of stressors that qualify as ELS, the field has reached a general consensus about the types of experiences that *do* reflect ELS. Although not exhaustive, the following experiences are frequently examined as indicators of ELS: physical, sexual, and emotional abuse; physical and emotional neglect; exposure to domestic violence; other forms of interpersonal violence exposure (e.g., in the community); separation or abandonment from caregivers (e.g., early institutional rearing, parental death, other loss of a parent); and chronic or extreme poverty and other indicators of economic adversity and material deprivation (e.g., food insecurity). For the remainder of this review, I focus specifically on these forms of ELS and do not review other forms of stress occurring in childhood and adolescence. I use the terms “ELS” and “adversity” interchangeably in this review.

Early Life Stress and Psychopathology

The link between ELS and psychopathology has been investigated extensively. Hundreds of studies have examined associations between ELS exposure and risk for psychopathology, and the evidence is consistent and clear. Exposure to ELS is associated with an increased risk for experiencing many commonly occurring forms of psychopathology (Evans, Li, & Whipple, 2013; McLaughlin, 2016). Here, I briefly review this evidence, focusing specifically on findings from epidemiological studies designed to allow inferences to be drawn at the population level and longitudinal studies that examine the onset of psychopathology after the occurrence of ELS.

Population-based and longitudinal studies have documented five general patterns with regard to ELS and the distribution of mental disorders in the population. First, epidemiological studies that are designed to estimate population prevalence have consistently found that exposure to ELS is common. In the original ACE study, about two thirds of US adults (67.3%) reported exposure to at least one major form of ELS (Dong et al., 2004). The ACE study focused on adults who were members of the Kaiser Health Plan in San Diego, California, but it did not use probability sampling or weighting, key features of epidemiological studies that allow inferences to be made at the population level. In epidemiological surveys of the US population that use probability sampling and weighting, the prevalence of exposure to ELS is estimated at about 50% (Green et al., 2010; Kessler, Davis, & Kendler, 1997; McLaughlin, Conron, Koenen, & Gilman, 2010; McLaughlin et al., 2012), although the prevalence of specific types of ELS (e.g., physical abuse) varies across studies. Similar prevalence estimates have been reported in other

high-income countries as well as in low- and middle-income countries worldwide (Benjet et al., 2009; Kessler et al., 2010; Rosenman & Rodgers, 2004). Population-based studies indicate clearly that children throughout the world frequently experience ELS.

A second general finding is that individuals who have experienced ELS are at elevated risk for developing a lifetime mental disorder compared to those without such exposure (Edwards et al., 2003; Green et al., 2010; Kessler et al., 1997, 2010; MacMillan, et al., 2001; McLaughlin, Conron, et al., 2010; McLaughlin et al., 2012). In epidemiological studies, the magnitude of this risk scales with the degree of exposure to ELS, such that the odds of developing a lifetime mental disorder increase as the degree of exposure to ELS increases (Edwards et al., 2003; Green et al., 2010; Kessler et al., 1997, 2010; McLaughlin, Conron, et al., 2010; McLaughlin et al., 2012). Greater risk for lifetime psychopathology among those with a history of ELS has also been observed in numerous longitudinal studies (Caspi et al., 2014; Cohen, Brown, & Smailes, 2001; Fergusson, Horwood, & Lynskey, 1996; Fergusson & Lynskey, 1996; Weich, Patterson, Shaw, & Stewart-Brown, 2009), and the magnitude of that risk also increases with greater exposure to ELS. Few consistent individual differences have been documented with regard to the association of ELS with lifetime psychopathology, including differences based on sex and race/ethnicity (Ahern, Karasek, Luedtke, Bruckner, & van der Laan, 2016; McLaughlin, Conron, et al., 2010; McLaughlin et al., 2012). For example, in a nationally representative sample of over 10,000 US adolescents, the associations of 10 forms of ELS with lifetime disorders—including mood, fear, behavioral, and substance disorders—did not vary across White, Black, and Hispanic/Latino adolescents (Ahern et al., 2016). In a population-based study of US adults, the associations between violence exposure and onset of mental disorders by adolescence did not vary for males and females (Dunn, Gilman, Willett, Slopen, & Molnar, 2012), although stronger associations between child abuse and some mental disorders were reported in a population-based sample of adults in Ontario, Canada (MacMillan et al., 2001). Cross-national variation in the association of ELS with lifetime psychopathology is also minimal; in a study of over 50,000 respondents from 21 countries worldwide, the association of ELS exposure with lifetime mental disorders exhibited little meaningful variation across countries (Kessler et al., 2010).

Third, exposure to ELS is associated with virtually all commonly occurring forms of psychopathology, and the associations of ELS with mental disorders are largely nonspecific. Individuals who have experienced ELS are more likely to develop mood, anxiety, substance use, and disruptive behavior disorders than those with no history of ELS exposure, with little meaningful variation in the strength of associations across disorder classes in epidemiological studies (Chapman et al., 2004; Dube et al., 2003; Green et al., 2010; Kessler et al., 1997, 2010; McLaughlin et al., 2012). Exposure to ELS is also associated with psychotic experiences (Janssen et al., 2004; McGrath et al., 2017) and suicidal ideation and attempts (Afifi et al., 2008; Bruffaerts et al., 2010; Dube et al., 2001; Molnar, Berkman, & Buka, 2001) in population-based studies, with the magnitude of association in the same range as for other forms of psychopathology. Multiple epidemiological studies have shown that associations between child maltreatment and

lifetime psychopathology 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 liability (Caspi et al., 2014; Keyes et al., 2012). Evidence from longitudinal studies is consistent with these general patterns (Cohen et al., 2001; Fergusson et al., 1996; Weich et al., 2009). Exposure to ELS is associated with elevated risk of depression, anxiety, behavior problems, substance abuse, suicidal behavior, and psychotic experiences at later points in development (Cohen et al., 2001; Enns et al., 2006; Fergusson et al., 1996; Fergusson & Lynskey, 1996; Jaffee, Caspi, Moffit, Polo-Tomás, & Taylor, 2007; Johnson et al., 2002; Kelleher et al., 2013; Phillips, Hammen, Brennan, Najman, & Bor, 2005; Varese et al., 2012; Widom, DuMont, & Czaja, 2007).

Fourth, exposure to ELS is associated with heightened vulnerability to psychopathology that persists across the life course. In epidemiological studies, ELS exposure is associated not only with risk of developing a mental disorder in childhood and adolescence (McLaughlin et al., 2012) but also in adulthood (Benjet, Borges, & Medina-Mora, 2010; Green et al., 2010; Kessler et al., 1997, 2010; McLaughlin, Conron et al., 2010). Greater odds of mental disorder onset in adulthood are observed among those with a history of ELS even after adjusting for the presence of psychopathology at earlier points in development (Chapman et al., 2004; Green et al., 2010; Kessler et al., 2010). Prospective studies confirm these patterns. For example, in a 45-year longitudinal study, exposure to ELS was associated with elevated risk for depression and anxiety disorders in adolescence, early adulthood, and middle adulthood, with no meaningful reduction in the magnitude of associations with increasing age (Clark, Caldwell, Power, & Stansfeld, 2010). Associations between ELS and psychopathology in middle adulthood remained even after accounting for earlier onset mental disorders. Similar findings have emerged from other long-term prospective studies, indicating that the risk for psychopathology associated with exposure to ELS persists well into adulthood (Collishaw et al., 2007; Koenen, Moffit, Poulin, Martin, & Caspi, 2007; McLaughlin et al., 2010; Weich et al., 2009).

Finally, exposure to ELS 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 et al., 2012). Approximately 30% of lifetime mental disorders in the population are attributable to exposure to ELS. This reflects both the high prevalence of exposure to ELS and the strong associations of ELS with the onset of psychopathology. Together, findings from epidemiological and longitudinal studies indicate clearly that exposure to ELS powerfully shapes risk for psychopathology in the population.

Common Approaches to Conceptualizing Early Life Stress

Discrete Stressors

Prior to the ACE study, most research on ELS focused on specific types of individual stressors. This approach continues in some lines of research even today. Common types of ELS that, until recently, had been studied in relative isolation from one another included physical abuse (Springer, Sheridan, Kuo, & Carnes, 2007; Sugaya et al., 2012), sexual abuse (Molnar, Buka, et al., 2001; Mullen, Martin, Anderson, Romans, & Herbison, 1993; Trickett, Noll, & Putnam, 2011), neglect (Dubowitz, Papas, Black, & Starr, 2002), parental death (Fristad, Jedel, Weller, & Weller, 1993), parental divorce (Chase-Lansdale, Cherlin, & Kiernan, 1995), and poverty (Duncan et al., 1994; McLeod & Shanahan, 1993; McLoyd, 1998).

The ACE study was the first to consider each of these distinct types of stressors as reflecting indicators of the same underlying construct: adverse childhood experiences or ELS. Each of the forms of ELS described earlier—along with several others, including parental psychopathology and substance abuse, parental criminal behavior, and emotional abuse—were assessed in the ACE study. One of the key findings from the ACE study was that most people who have experienced one form of ELS have also experienced multiple other adverse experiences (Dong et al., 2004). This finding has been replicated in population-based studies of children (Finkelhor, Ormrod, & Turner, 2007; McLaughlin et al., 2012) and adults (Green et al., 2010; Kessler et al., 2010). These findings raise serious concerns about approaches to studying ELS that focus on individual types of adversity and fail to account for the co-occurrence between different forms of ELS. This is a major limitation in studies focusing on a single type of ELS, as it is impossible to determine whether any observed associations between a particular form of ELS (e.g., physical abuse) and outcome (e.g., depression) represent true consequences of the focal adversity in question or the downstream effects of other co-occurring experiences (e.g., poverty) that may have different developmental consequences. Without measuring and controlling for co-occurring forms of ELS, the strong possibility of confounding by other forms of adversity cannot be ruled out. An additional limitation when considering discrete forms of ELS individually is an inherent assumption that the mechanisms linking each of these types of experiences with downstream developmental outcomes, including psychopathology, are completely distinct. In other words, this approach implicitly assumes that the mechanisms linking, for example, sexual abuse with depression are distinct from those linking physical abuse or domestic violence with depression. While there is certainly some variability in core underlying developmental mechanisms across

different forms of adversity, it is also quite likely that at least some mechanisms are shared across multiple types of ELS.

Recognition of the high co-occurrence of different forms of ELS led to a shift in the way that ELS is conceptualized, with many recent studies examining multiple forms of ELS jointly. These approaches are described in the next section on “Cumulative Risk.” However, examination of discrete individual forms of ELS remains common for some types of adversity. For example, institutional rearing is often studied as a discrete exposure, separate from other forms of ELS (Gunnar, van Dulmen, & The International Adoption Project Team, 2007; McLaughlin, Sheridan et al., 2014; Rutter et al., 2010; Tottenham et al., 2011). This reflects the fact that many children who participate in research following institutional rearing have been adopted into families who have a strong desire to be parents, are relatively well-off financially, and generally have low levels of co-occurring adversity, as well as the fact that institutional rearing is a well-defined exposure with a clear onset and offset but for which co-occurring adversities occurring in the institutional environment are typically not assessed systematically.

Cumulative Risk

The prevailing approach to assessing and conceptualizing ELS over the past decade has been the cumulative risk approach. This approach tallies the number of distinct forms of ELS experienced to create a risk score without regard to the type, chronicity, or severity of the experience and uses this risk score as a predictor of outcomes (Dube et al., 2001; Evans et al., 2013; Felitti et al., 1998). For example, a child who experienced physical abuse, sexual abuse, and domestic violence would have a risk score of three; a child who experienced poverty, neglect, and maternal depression would also have a risk score of three. Cumulative risk thus focuses on the *number* of distinct types of ELS a child has experienced rather than the severity of those experiences or type of adversity. A critical assumption in the cumulative risk approach is that discrete forms of ELS have additive effects on downstream developmental outcomes (Evans et al., 2013). That assumption implies that each additional form of ELS a child experiences will have statistically similar effects on developmental outcomes, regardless of the number of co-occurring adverse experiences the child has had.

A cumulative risk approach involves a variety of benefits as compared to studying discrete forms of ELS in isolation. The first concerns the co-occurrence of different forms of ELS; this issue was described in the previous section on discrete stressors. A second is that multiple risk exposures have consistently been shown to have more robust effects on developmental outcomes than single exposures (Evans et al., 2013). Indeed, this is a well-replicated and fairly intuitive finding (Dube et al., 2001; Dube et al., 2003; Green et al., 2010; Jaffee et al., 2007; McLaughlin et al., 2012). In addition, the cumulative risk approach can identify children that would benefit from intervention, as children with high degrees of exposure to multiple forms of ELS are clearly in greatest need of intervention (Evans et al., 2013). Finally, some have argued that a cumulative risk score confers

statistical advantages over other strategies for measuring and modeling ELS because they are parsimonious (i.e., they use one value for ELS rather than multiple metrics); they are unweighted (i.e., they consider all forms of ELS to be equal in their developmental effects); they are not sensitive to collinearity among predictors (i.e., multiple forms of ELS can be tallied and summed without introducing collinearity into a statistical model); they are more stable statistically than individual stressors; and they are easily understandable to policymakers and other relevant stakeholders outside the scientific community (Evans et al., 2013). The argument regarding parsimony and ease of understanding cumulative risk scores is borne out by recent trends. The cumulative risk approach has been widely adopted following the ACE study and has generated considerable interest in understanding the long-term consequences of ELS and preventing those effects. The importance of ELS has been acknowledged in policy briefs by major medical organizations, including the American Academy of Pediatrics and the American Heart Association. Risk scores can also be used as a screening tool to identify those children in greatest need of intervention; indeed, some medical clinics have begun to screen for ELS as a routine part of clinical practice (Burke, Hellman, Scott, Weems, & Carrion, 2011). Thus, the cumulative risk approach has broad appeal, particularly for clinicians and policymakers.

However, some of the assumptions used to justify the cumulative risk approach are not borne out in empirical data. In particular, existing evidence indicates that the effects of multiple ELS exposures on mental health outcomes are not additive. Instead, the associations of multiple forms of adversity with psychopathology have been shown to be subadditive in several population-based studies (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012), such that as the number of ELS exposures increases, the odds of developing a mental disorder also increase, but at a *decreasing rate*. Thus, the incremental effect of having a sixth ELS exposure on risk for psychopathology (compared to having five) is lower than the incremental effect of experiencing a second type of ELS relative to having one (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012). Advocates for a cumulative risk approach acknowledge that evidence for the assumption of additivity is highly mixed (Evans et al., 2013).

Several other limitations of the cumulative risk approach are worth noting. First, the use of dichotomous (1,0) indicators for each ELS experience means that only high-severity ELS experiences are counted in risk scores (Evans et al., 2013). While it is fairly obvious that extreme exposures (e.g., repeated, chronic physical abuse) will likely have larger developmental impacts than less severe experiences (e.g., occasional violence within the family), it is also clear that even low levels of exposure to ELS can have profound impacts on child development. Moreover, considering only extreme cases makes it impossible to evaluate how the severity or chronicity of exposure to ELS influences outcomes of interest. Second, as I have argued elsewhere (McLaughlin & Sheridan, 2016), the cumulative risk approach also has significant limitations when used to identify mechanisms linking ELS with developmental outcomes, including psychopathology. Most notably, risk scores fail to distinguish between distinct types of environmental experience and assume that all forms of ELS will have similar effects on developmental processes.

The assumption is that the wide range of experiences encompassed in the ELS construct —including abuse, domestic violence, poverty, neglect, institutional rearing, parental loss, and many others—will all have *identical* effects on child development. Implicit in this assumption is the notion that all forms of ELS influence development through the same underlying mechanisms that are largely universal or shared across many types of adverse experiences. In other words, risk scores assume that experiencing physical abuse, sexual abuse, and domestic violence (risk score of 3) will influence a child's development in exactly the same way as experiencing poverty, neglect, and maternal depression (also a risk score of 3). As I review next in the section on "Dimensions of Experience," this assumption is highly tenuous. Thus, cumulative risk models have serious limitations when used to identify mechanisms through which ELS increases risk for psychopathology.

Dimensions of Experience

My colleague Margaret Sheridan and I have developed an alternative to the cumulative risk model that conceptualizes ELS as a multidimensional construct and articulates specific developmental mechanisms linking particular dimensions of adverse environmental experience with psychopathology (McLaughlin & Sheridan, 2016; McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). Rather than counting the total number of adversities, our approach attempts to distill complex adverse experiences into core underlying dimensions that cut across multiple forms of ELS. The model posits that many forms of ELS can be aligned along dimensions, and that similar developmental mechanisms will be observed for experiences that align along the same dimension. This model is based on two principles. First, it is possible to extract core underlying dimensions of environmental experience that occur in numerous types of adversity that share common features. Two initial dimensions proposed in our model are *threat*, which encompasses experiences involving harm or threat of harm to the child, and *deprivation*, which involves an absence of expected inputs from the environment during development, such as cognitive and social stimulation (e.g., complex language directed at the child). Conceptually, these dimensions cut across numerous experiences that reflect the core feature of threat or deprivation to varying degrees. For example, threat of harm to the child is a key component of many forms of ELS, including physical and sexual abuse, witnessing domestic violence, and exposure to other forms of interpersonal violence (e.g., in the school or community). The degree of threat involved in chronic physical abuse is higher than the degree of threat involved in occasional exposure to violence occurring in one's community, but both experiences share a core feature of threat to the child. In contrast, deprivation involving low levels of cognitive stimulation is a core feature of neglect, institutional rearing, other forms of parental absence, and occurs more often in families living in poverty or with low levels of parental education, though not universally (Bradley & Corwyn, 2002; Bradley, Corwyn, McAdoo, & Coll, 2001; Hart & Risley, 1995). Our conceptualization of threat and deprivation as key dimensions of ELS aligns with other recent work arguing for distinctions between forms of adversity that represent the presence of harmful versus inadequate environmental input (e.g.,

abuse vs. neglect) (Humphreys & Zeanah, 2015). Critically, although threat and deprivation are dimensions that are reflected in many common forms of ELS, other dimensions also clearly exist. For example, environmental predictability is a dimension that has relevance for many forms of ELS. Thus, a two-dimensional model is simply a starting point for conceptualizing adverse environmental experiences based on underlying dimensions.

The second principle on which the model is based is that different dimensions of ELS have distinct influences on children's development. Specifically, our model argues that different dimensions of adversity have unique influences on emotional, cognitive, and neurobiological development, and that understanding these distinct developmental pathways is critical for identifying mechanisms linking ELS with psychopathology (McLaughlin, 2016; McLaughlin & Sheridan, 2016; McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). In other words, a key assumption in the dimensional model is that the mechanisms linking different forms of ELS with psychopathology are not universal. This idea is not novel. Early conceptual models of child maltreatment argued for the importance of considering distinct types of maltreatment as reflecting different dimensions of experience with unique developmental consequences (Manly, Cicchetti, & Barnett, 1994; Manly, Kim, Rogosch, & Cicchetti, 2001). Seth Pollak's seminal work on behavioral and neural differences in emotion perception and recognition among children who were abused versus neglected demonstrates clearly that distinct forms of ELS have different effects on developmental processes (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003). More recently, other groups have also articulated similar ideas regarding distinctions between different types of ELS (Farah et al., 2008; Humphreys & Zeanah, 2015). Both principles of our model—that ELS can be distilled into core underlying dimensions that cut across multiple forms of adversity and that the mechanisms linking distinct dimensions of adversity to the onset of psychopathology vary across different ELS dimensions—are conceptually similar to ideas that have long been articulated in the field but are often ignored in current approaches involving cumulative risk.

Finally, a key element of this model is not just that ELS can be distilled into core underlying features that can be conceptualized as dimensions, but that in order to identify these mechanisms it is essential to control for co-occurring types of exposures when examining the effects of a particular dimensions of ELS (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014, 2016; Sheridan, Peverill, & McLaughlin, 2017). This is essential, as many forms of ELS co-occur, as reviewed earlier in this chapter. The goal is not to identify children who have only experienced one particular form of ELS in isolation—that would obviously be a fool's errand. The goal is to demonstrate that despite the co-occurrence of ELS experiences, there is at least some specificity in the mechanisms that link particular forms of ELS with downstream outcomes. Such an approach is critical for isolating whether developmental processes that are impacted by adversity globally or are specific to particular forms of ELS. Moreover, it is essential to control for co-occurring exposures because if one

demonstrates, for example, an association between violence exposure and working memory without controlling for co-occurring forms of ELS known to be associated with working memory (e.g., poverty, neglect) (Finn et al., 2016; Leonard, Mackey, Finn, & Gabrieli, 2015; Noble, McCandliss, & Farah, 2007), it would be easy to find a spurious association with violence exposure that is in fact explained by co-occurring adversity. Understanding how particular developmental processes are influenced by ELS and whether these effects are specific or general is of critical importance for developing targeted and effective preventive interventions.

How do the assumptions and justification for a dimensional model diverge from those associated with cumulative risk? Both cumulative risk and the dimensional model share the assumption that multiple ELS exposures will have stronger effects on developmental outcomes than single exposures and that experiences that are more chronic or severe are also more likely to influence development (Evans et al., 2013; McLaughlin & Sheridan, 2016; McLaughlin, Sheridan, & Lambert, 2014), but the approach for addressing these issues differs. Cumulative risk advocates dichotomizing each exposure and creating one risk score; the dimensional model argues for assessing ELS across multiple dimensions and including separate variables for those dimensions in the same model. Dimensions can be modeled in a variety of ways, and the specific approach for representing a particular dimension (e.g., threat) is not prescribed. For example, a variable could be created to represent the frequency of exposure to threat (i.e., the frequency of exposure to multiple forms of violence); a variety score could also be used (i.e., reflecting the number of distinct types of threat a child has experienced). The specific approach can vary based on the assessments available in a given study; the key is to create a composite that represents a *dimension* of threat exposure, ranging from an absence of that experience to a high level of exposure and to control for co-occurring exposures along other dimensions (e.g., deprivation). The primary distinction between these approaches lies in the assumption that all forms of ELS are equal in their developmental effects—a core tenet of the cumulative risk model that justifies using an unweighted count of adversities (Evans et al., 2013). As reviewed in the sections that follow, accumulating evidence suggests that the developmental processes influenced by different dimensions of ELS are at least partially distinct (Busso, McLaughlin, & Sheridan, 2017; Everaerd et al., 2016; Lambert, King, Monahan, & McLaughlin, 2017; Lawson et al., 2017; Sheridan et al., 2017). This raises questions about the assumption of universal ELS effects on which the cumulative risk model is based with regard to understanding mechanisms linking ELS with psychopathology.

The dimensional model also diverges from cumulative risk with regard to assumptions regarding the statistical modeling of ELS. Cumulative risk scores provide a more parsimonious modeling strategy (i.e., a single variable) than using multiple composites reflecting different ELS dimensions. In contrast, the dimensional model advocates for the importance of including multiple indicators of distinct ELS dimensions in the same statistical model; an approach that cumulative risk assumes will introduce problematic collinearity into the statistical model. However, it is clear from population-based data that while different forms of ELS co-occur, that co-occurrence is not sufficiently high to

introduce problematic collinearity. Table 1 presents polychoric correlations among 10 distinct forms of ELS assessed in the National Comorbidity Survey—Replication Adolescent Supplement (NCS-A), a nationally representative study of over 10,000 US adolescents. Several patterns are notable. First, although most forms of ELS are correlated, the associations are mostly in the small to moderate range (r = 0.1–0.6). The strongest correlations are among experiences of maltreatment, including abuse and neglect, and domestic violence (r = 0.4–0.6), and most of the strongest associations are among indicators reflecting the same underlying dimension (i.e., physical, sexual, and emotional abuse and domestic violence are all indicators of threat, and thus would be reflected in a single threat composite). Cross-dimension associations between neglect and threat indicators are also moderate (r = 0.4–0.6), but not sufficiently high that they would be problematic when included in the same statistical model. Other indictors of deprivation, such as poverty and food insecurity, exhibit correlations of small magnitude with indicators of threat (r = 0.1–0.3). Prior work with the NCS-A demonstrates that collinearity is not a problem even in a model that includes separate variables for more than 10 distinct types of ELS in predicting psychopathology (McLaughlin et al., 2012). Thus, the co-occurrence of different forms of ELS in the population is meaningful, but not so dramatic that it is not possible to tease apart distinct associations between particular experiences and relevant developmental outcomes.

Table 1. Polychoric correlations among domains of early-life stress in the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A)

	1	2	3	4	5	6	7	8	9	10
1. Physical abuse	—									
2. Domestic violence	.58	—								
3. Sexual abuse	.30	.37	—							
4. Violent victimization	.28	.33	.87	—						
5. Witnessing violence	.20	.23	.21	.33	—					

6. Emotional abuse	.45	.39	.30	.32	.22	—				
7. Poverty	.20	.16	.01	.00	.11	.12	—			
8. Financial hardship	.32	.34	.23	.25	.21	.25	.29	—		
9. Food insecurity	.23	.21	.15	.15	.13	.17	.24	.30	—	
10. Neglect	.46	.48	.45	.44	.17	.59	.16	.34	.18	—

Note: Polychoric correlations were calculated after applying sample weights to adjust for non-response and differential selection probabilities as well as to make the sample socio-demographic distributions representative of the U.S.; indicators of threat are in rows 1–6 and indicators of deprivation are in rows 7–10.

Mechanisms Linking Early Life Stress With Psychopathology

Identifying developmental mechanisms that underlie the strong associations between ELS and psychopathology is a major goal of ongoing research. Determining how experiences of ELS influence development in emotional, social, cognitive, and neurobiological domains and how disruptions in specific developmental processes, in turn, confer greater risk for psychopathology is a pressing research priority (McLaughlin, 2016). As noted earlier, ELS exhibits strong patterns of multifinality, predicting elevated risk for the onset of virtually all commonly occurring mental disorders (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012). However, the mechanisms that explain how ELS influences this liability to psychopathology are only beginning to be understood. Greater understanding of these mechanisms is needed to inform the development of interventions to prevent the onset of mental health problems in children who have experienced ELS. In order to intervene effectively, it is necessary to understand the developmental processes that are altered by experiences of ELS and how they ultimately contribute to the etiology of mental disorders. Many of the later chapters in this volume are concerned explicitly with mechanisms linking stress to the onset of psychopathology. Here, I focus on two broad approaches for conceptualizing and studying these mechanisms that have emerged from prevailing models of ELS: cumulative risk and dimensions of adversity. Each model articulates a different perspective on the mechanisms linking ELS with psychopathology and whether those mechanisms are general or specific to particular types of ELS.

General/Universal Mechanisms

One of the key assumptions of the cumulative risk model is that different forms of ELS have largely similar effects on developmental outcomes (Evans & Kim, 2007; Evans et al., 2013). As noted earlier, this assumption implies that a child who has experienced physical abuse, sexual abuse, and domestic violence (and thus has a risk score of 3) will have similar developmental outcomes as a child who has experienced poverty, maternal depression, and neglect (and thus also has a risk score of 3). It is assumed that not only are the effects of these vastly different social and environmental experiences *qualitatively* similar but also *quantitatively* similar, such that each distinct type of ELS will have an effect of equal magnitude on any developmental process in question (Evans et al., 2013). This assumption of equivalence is fundamental to the cumulative risk score approach.

Is it possible that all forms of ELS influence development through the same underlying mechanisms? Around the same time that findings from the ACE study emerged, the concept of allostatic load was introduced as a comprehensive neurobiological model of the effects of stress (McEwen, 1998, 2000). Allostatic load provides a framework for explaining the neurobiological mechanisms linking multiple forms of ELS—as well as other forms of stress experienced later in life—to downstream health outcomes. Advocates of cumulative risk argue that allostatic load, and the associated disruptions in the regulation of stress response systems and other regulatory systems, represents a common mechanism that explains how numerous forms of seemingly disparate adverse experiences influence the wide range of developmental outcomes associated with ELS (Evans & Kim, 2007; Evans et al., 2013).

The concept of allostatic load and the links between stress exposure and allostatic load have been reviewed extensively elsewhere (Danese & McEwen, 2012; McEwen, 1998, 2012; McEwen & Gianaros, 2010; McEwen & Seeman, 1999). Briefly, the process of allostasis allows an organism to adapt to changing environmental demands through changes in regulatory systems, including the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS) (McEwen, 1998, 2000). These regulatory systems work to maintain homeostasis and promote recovery following environmental stressors (McEwen, 2000). These adaptations to stressors produce physiological changes that are adaptive in the short term but maladaptive in the long term (McEwen, 2000). For example, glucocorticoid release following a stressor produces rapid improvements in immunity, but chronic glucocorticoid release leads to maladaptive long-term changes in brain regions with high concentrations of glucocorticoid receptors, including the hippocampus, amygdala, and prefrontal cortex (PFC) (McEwen, 2000, 2012). This long-term wear-and-tear on the body’s regulatory systems resulting from chronic adaptation to stress is referred to as allostatic load (McEwen, 2000; McEwen & Seeman, 1999). Extensive evidence suggests that exposure to ELS disrupts stress response system functioning, including the ANS and HPA axis (Frodl & O’Keane, 2013; Gunnar & Quevedo, 2007; McCrory, De Brito, & Viding, 2010; Wilkinson & Goodyer, 2011), the primary regulatory systems that govern allostatic responses (McEwen, 2000). Indeed, greater

cumulative risk has been linked to higher levels of allostatic load, even in children (Evans, 2003; Evans, Kim, Ting, Tesher, & Shannis, 2007). These disruptions are the central mechanism explaining downstream consequences of ELS in the cumulative risk model.

Dysregulation in stress response systems and allostatic load represents a plausible pathway linking ELS with mental health. ELS has been associated consistently with alterations in stress response systems, including changes in diurnal regulation of the HPA axis (Alink, Cicchetti, Kim, & Rogosch, 2012; Doom, Cicchetti, & Rogosch, 2014; Doom, Cicchetti, Rogosch, & Dackis, 2013; Gunnar, Morison, Chisolm, & Schuder, 2001; van der Vegt, van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009; Zalewski, Lengua, Thompson, & Kiff, 2016) and reactivity of the HPA axis and ANS to laboratory stressors (Gunnar, Frenn, Wewerka, & Van Ryzin, 2009; Harkness, Stewart, & Wynne-Edwards, 2011; MacMillan et al., 2009; McLaughlin, Sheridan, Alves, & Mendes, 2014; McLaughlin et al., 2015). Similarly, disruptions in the regulation and reactivity of the HPA axis and ANS have also been linked with psychopathology in cross-sectional studies, and they have occasionally been shown to predict the subsequent onset of psychopathology, particularly depression (Adam et al., 2010; Wilkinson & Goodyer, 2011).

But are these stress pathways a universal mechanism that explains all—or even most—of the elevations in psychopathology that are associated with exposure to ELS? There are several problems with this assumption. First, the specific pattern of altered stress response system functioning associated with ELS varies across studies. The most commonly observed pattern involves blunted reactivity to laboratory stressors and globally reduced output (e.g., a flat diurnal rhythm) (Gunnar & Vazquez, 2001; MacMillan et al., 2009; McLaughlin, Sheridan, et al., 2015); this pattern has been observed in meta-analyses of chronic stress and HPA axis function (Miller, Chen, & Zhou, 2007). However, numerous studies document the opposite pattern—elevated reactivity to stressors or globally increased HPA axis output (Fries, Shirtcliff, & Pollak, 2008; Gunnar et al., 2001), and effects frequently vary by sex (Doom et al., 2013). This is true even for studies examining the same type of ELS exposure (e.g., institutional rearing). Thus, even when considering the effects of ELS on a relatively circumscribed set of neurobiological processes, there is wide variability across studies in the nature of these relationships.

Second, although disruptions in stress response systems and allostatic load have been consistently linked to the onset of physical health problems, such as cardiovascular disease (Heim, Ehlert, & Helhammer, 2000; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997), evidence for their role in the etiology of mental disorders is less clear. The literature on stress response system disruption and psychopathology has produced widely variable findings. For example, depression and posttraumatic stress disorder (PTSD) are two mental disorders that have been frequently studied in relation to cortisol regulation and reactivity. Meta-analysis indicates an absence of a meaningful relationship between diurnal cortisol and depression (Knorr, Vinberg, Kessing, & Wetterslev, 2010), and that adults with depression have similar cortisol response to laboratory stress paradigms as those without depression, but delayed recovery of the cortisol response; particularly among older adults and those with more severe depression (Burke, Davis, Otte, & Mohr,

2005). For PTSD, two meta-analyses have produced conflicting results, with one demonstrating an absence of an association between PTSD and HPA axis function (Klaassens, Giltay, Cuijpers, van Veen, & Zitman, 2012) and the other indicating moderately lower daily cortisol output among those with PTSD than trauma-exposed controls (Morris, Compas, & Garber, 2012). Prospective studies have also produced conflicting findings, with some demonstrating associations between cortisol awakening response and later onset of a depressive episode (Adam et al., 2010) and others failing to replicate this effect (Carnegie et al., 2014; Nederhof et al., 2015). Moreover, disruptions in stress response system functioning are clearly an insufficient mechanism to explain the wide range of other developmental outcomes that are commonly observed among children who experience ELS. For example, children exposed to neglect and poverty often exhibit difficulties in the domain of expressive and receptive language (Farah et al., 2006; Hildyard & Wolfe, 2002). There is no obvious link between stress response system functioning and language ability, which indicates that other mechanisms are involved.

Finally, allostatic load and disruptions in stress response system functioning provide little in the way of intervention targets for preventing the onset of psychopathology in children exposed to ELS. Aside from attempting to prevent exposure to ELS in the first place, how might we intervene to prevent the downstream consequences of ELS based on the allostatic load model? Few effective intervention approaches for children exposed to ELS have been developed as a result of cumulative risk models or stress dysregulation mechanisms. Although psychosocial interventions can influence cortisol regulation, the nature of these intervention effects varies widely across studies (Slopen, McLaughlin, & Shonkoff, 2014). Thus, although cumulative risk models are useful for screening and identifying children in need of intervention because of high levels of ELS exposure, they provide little guidance about *how* to intervene.

Specific Mechanisms

One of the guiding principles of the dimensional model of ELS is that unique emotional, social, cognitive, and neurobiological pathways underlie the links between different dimensions of adverse early experience and developmental outcomes, including psychopathology (McLaughlin, 2016; McLaughlin & Sheridan, 2016; McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). Specifically, the model argues that the mechanisms linking experiences of threat with psychopathology are at least partially distinct from those underlying the association between deprivation and psychopathology. Here, I briefly review predictions about the neurodevelopmental mechanisms that underlie the associations of threat and deprivation with psychopathology and existing evidence for these predictions.

With regard to threat, the dimensional model predicts that threatening experiences during childhood alter emotional development in ways that facilitate the rapid identification of potential threats in the environment (McLaughlin & Lambert, 2016; McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). This is an

adaptive response to being raised in an environment characterized by danger. Specifically, children whose early environment involves a high degree of threat should exhibit changes in emotion perception and recognition, attention and memory for emotional stimuli, emotional learning, emotional reactivity, and emotion regulation; these changes in social and emotional processing should be most pronounced for negative stimuli (e.g., angry or fearful faces) that could predict the presence of threat in the environment (McLaughlin & Lambert, 2016). Existing evidence supports these predictions. Enhanced processing of threatening social and emotional information has been consistently observed in children who have experienced physical or sexual abuse—environments characterized by a high degree of threat. For example, children exposed to these types of threatening environments exhibit attention biases toward threatening stimuli, including faster attentional engagement and slower attentional disengagement from anger, and are more likely to perceive neutral facial expressions as threatening (Gibb, Schofield, & Coles, 2008; Pollak et al., 2000; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003). Social information processing biases involving preferential attention to threatening cues and attributions of hostility in ambiguous situations are also common among children who have experienced abuse (Dodge, Bates, & Pettit, 1990; Dodge, Petit, Bates, & Valente, 1995).

With regard to emotional learning, emerging evidence indicates altered patterns of threat-safety learning in children who have experienced threat. Specifically, children exposed to violence exhibit skin conductance response of equal magnitude to both threat and safety cues in a fear conditioning paradigm (McLaughlin et al., 2016), which may reflect generalization of fear from the cue that predicts threat to the cue that predicts safety. Elevated emotional reactivity to threat following childhood threat exposure has been observed using a variety of metrics of emotional reactivity, including self-reported emotional responses (Heleniak, Jenness, Van der Stoep, McCauley, & McLaughlin, 2016; Heleniak, King, Monahan, & McLaughlin, 2018; Hennessy, Rabideau, Cicchetti, & Cummings, 1994), negative emotional reactions to daily stressors (Glaser, van Os, Portegijs, & Myin-Germeys, 2006; Wichers et al., 2009), amygdala activation to negative emotional cues (McCrory et al., 2011, 2013; McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015), and threat-related autonomic nervous system reactivity (Heleniak, McLaughlin, Ormel, & Riese, 2016; McLaughlin, Sheridan, Alves, et al., 2014). Emotion regulation difficulties are also well documented among children who have been exposed to violence, including in tasks assessing implicit and explicit forms of emotion regulation (Lambert et al., 2017; Marusak, Martin, Etkin, & Thomason, 2015; Powers, Etkin, Gyurak, Bradley, & Jovanovic, 2015) as well as adult reports of children’s emotion regulation abilities (Kim & Cicchetti, 2010; Kim-Spoon, Cicchetti, & Rogosch, 2013). In addition to behavioral indicators, atypical function in amygdala-prefrontal circuitry that supports these emotion regulation processes has also been observed consistently in children who have experienced abuse and other forms of interpersonal violence (Herringa et al., 2013; Marusak, Etkin, & Thomason, 2015; Marusak, Martin et al., 2015; McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015).

The disruptions in social and emotional processing that have been observed in children raised in threatening environments have been linked to multiple forms of psychopathology (see McLaughlin & Lambert, 2016, for a review). Briefly, enhanced perceptual salience and attention toward threatening stimuli, as well as social information processing biases (e.g., hostile attribution bias), have been associated with anxiety (Briggs-Gowan et al., 2015; Shackman, Shackman, & Pollak, 2007), PTSD (Briggs-Gowan et al., 2017), and conduct problems (Shackman & Pollak, 2014). Difficulty discriminating between threat and safety cues in fear conditioning paradigms is associated with externalizing psychopathology (Fairchild, van Goozen, Stollery, & Goodyer, 2008; McLaughlin et al., 2016). Heightened emotional reactivity to negative stimuli has been linked to internalizing and externalizing problems in cross-sectional studies (Heleniak, McLaughlin, et al., 2016; McLaughlin, Sheridan, Alves, et al., 2014) and predicts the future onset of psychopathology (Heleniak, Jenness, et al., 2016; McLaughlin, Kubzansky, et al., 2010). Elevated amygdala response to negative stimuli assessed prior to trauma exposure predicts the onset of PTSD symptoms following a traumatic stressor (McLaughlin, Busso, et al., 2014) as well as increases in internalizing symptoms following exposure to stressful life events (Swartz, Knodt, Radtke, & Hariri, 2015). Finally, difficulties with emotion regulation observed in children exposed to violence are associated with the onset of both internalizing and externalizing psychopathology (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007), as well as onset of PTSD symptoms following a traumatic event (Jenness et al., 2016). Multiple longitudinal studies demonstrate that emotion regulation difficulties are a mechanism linking childhood exposure to threat to the onset of internalizing and externalizing problems (Heleniak, Jenness, et al., 2016; Kim & Cicchetti, 2010).

In contrast to these social and emotional processing mechanisms in children exposed to threat, the dimensional model posits that deprivation influences development through a set of mechanisms that are at least somewhat distinct. Deprivation refers to an absence of social and cognitive stimulation and constrained opportunities for learning among children whose interactions with supportive caregivers are limited. This kind of deprivation has frequently been observed among children who experience neglect and institutional rearing. For example, children who are neglected experience low levels of sensitive, responsive, and stable caregiving, as do children raised in institutions whose interactions with caregivers are infrequent and lacking in sensitive and contingent responding (Bousha & Twentyman, 1984; Gaudin, Polansky, Kilpatrick, & Shilton, 1996; Kaufman Kantor et al., 2004; Smyke et al., 2007). Because most types of early learning occur in the context of interactions with caregivers, learning opportunities are constrained among children who experience less frequent and stable caregiving. The absence of consistent interactions with a caregiver deprives children of sensory, motoric, linguistic, and social experiences that caregivers provide that provide fodder for early learning. Children who are neglected or raised in institutional settings experience meaningful reductions in cognitive stimulation, learning opportunities, supervision by adults, and interactions with caregivers (Bousha & Twentyman, 1984; Hines, Kaufman

Kantor, & Holt, 2006; Kaufman Kantor, et al., 2004; Nelson, Furtado, Fox, & Zeanah, 2009; Smyke et al., 2007; Zeanah et al., 2003). Although low socioeconomic status (SES) is not inherently associated with this type of cognitive and social deprivation, many studies suggest that children being raised in poverty or by parents with low levels of education experience, on average, lower levels of cognitive stimulation, fewer learning opportunities at home and school, and less exposure to complex language than children raised in higher SES families (Bradley & Corwyn, 2002; Bradley et al., 2001; Crosnoe et al., 2010; Garrett, Ng'andu, & Ferron, 1994; Hart & Risley, 1995; Linver, Brooks-Gunn, & Kohen, 2002; Sheridan, Sarsour, Jutte, D'Esposito, & Boyce, 2012).

This type of early deprivation in cognitive and social stimulation can have pronounced effects on children's cognitive development, particularly in the domains of language and executive functioning (McLaughlin, 2016; Sheridan & McLaughlin, 2016). Indeed, poor performance on tasks of expressive and receptive language and executive functioning has been consistently observed among children who experience deprivation related to neglect (Allen & Oliver, 1982; Culp et al., 1991; Spratt et al., 2012), institutional rearing (Albers, Johnson, Hostetter, Iverson, & Miller, 1997; Bos, Fox, Zeanah, & Nelson, 2009; Colvert et al., 2008; Hostinar, Stellern, Schaefer, Carlson, & Gunnar, 2012; Loman et al., 2013; McDermott et al., 2013; Pollak et al., 2010; Rakhlin et al., 2015; Tibu et al., 2016; Windsor et al., 2011), and low SES (Blair, 2002; Farah et al., 2006; Fernald, Marchman, & Weisleder, 2013; Noble et al., 2007; Noble, Norman, & Farah, 2005; Raver, Blair, Willoughby, & The Family life Project Key Investigators, 2013; Weisleder & Fernald, 2013). SES-related differences in language and executive functioning are observable as early as infancy (Clearfield & Niman, 2012; Fernald et al., 2013). Deprivation-related adversity involving neglect is more strongly associated with these cognitive outcomes than experiences of threat, such as physical and sexual abuse (Hildyard & Wolfe, 2002). In addition to these behavioral differences, altered function in the neural networks that support language and executive functioning—particularly the lateral prefrontal cortex—have been observed in children who have experienced deprived early environments, including institutional rearing (Mueller et al., 2010) and low SES (Kishiyama, Boyce, Jimenez, Perry, & Knight, 2009; Raizada, Richards, Meltzoff, & Kuhl, 2008; Sheridan et al., 2012). Critically, these neural differences are not present at birth (Brito, Fifer, Myers, Elliott, & Noble, 2016), suggesting that they are the result of environmental experience.

This variability in language and executive functioning is likely shaped by early learning opportunities and environmental stimulation. For example, the degree of stimulation in the home as well as the amount and quality of maternal language predicts children's language skills (Farah et al., 2008; Hoff, 2003). Direct links have also been demonstrated between the degree of enrichment and stimulation in the early caregiving environment and children's cognitive outcomes, including executive functioning and school achievement (Crosnoe et al., 2010; Duncan, 2003; Sarsour et al., 2011). SES-related differences in both language ability and executive functioning are mediated by the complexity of language spoken at home and the degree of enrichment in the home environment (Hoff, 2003; Sarsour et al., 2011; Sheridan et al., 2012). Interventions that increase children's access to learning opportunities and provide more consistent and

structured interactions with adults have consistently been shown to improve cognitive development among children growing up in low-SES families in both experimental (Campbell, Pungello, Miller-Johnson, Burchinal, & Ramey, 2001; Schweinhart, Berrueta-Clement, Barnett, Epstein, & Weikart, 1985) and observational studies (Anderson et al., 2003; Gormley, Gayer, Phillips, & Dawson, 2005), providing additional support for the role of cognitive and social stimulation in shaping children's cognitive development.

Together, these disruptions in cognitive development are associated with later risk for externalizing psychopathology. With regard to language ability, meta-analysis indicates that language difficulties prospectively predict the onset of externalizing behavior (Chow & Wehby, 2016). Prospective studies also suggest that children with low language ability are at elevated risk for internalizing problems (Bornstein, Hahn, & Suwalsky, 2013; Salmon, O'Kearney, Reese, & Fortune, 2016). Poor executive functioning is a core feature of attention-deficit/hyperactivity disorder (ADHD; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Sergeant, Geurts, & Oosterlaan, 2002; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), and it has been shown to mediate the association between deprivation-related ELS and the onset of ADHD (Tibu et al., 2016). Some studies have observed executive functioning difficulties in children with other forms of externalizing psychopathology, including conduct disorder and oppositional defiant disorder (Clark, Prior, & Kinsella, 2002; Hobson, Scott, & Rubia, 2011), while others have found that poor executive functioning is specific to ADHD (Oosterlaan, Scheres, & Sergeant, 2005). However, poor executive functioning prospectively predicts 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). Executive functioning is a less well-established risk factor for internalizing problems, but difficulties with inhibition and cognitive flexibility that emerge in the context of emotional processing have been linked to risk for depression (Goeleven, De Raedt, Baert, & Koster, 2006; Joorman & Gotlib, 2010), potentially by increasing rumination (Joorman, 2006).

Altogether, substantial progress has been made in delineating the mechanisms that underlie the associations of ELS with psychopathology. While some general mechanisms have been identified that are common across many forms of ELS (e.g., disruptions in physiological stress response systems), many of these mechanisms appear to differ as a function of the specific type of ELS experienced. Exposure to trauma and forms of ELS involving threat have clear associations with emotional and social information processing and emotion regulation, whereas exposure to forms of ELS involving deprivation have much stronger associations with cognitive processes, including language and executive functioning. These emerging findings highlight mechanisms that are unique to particular forms of ELS but can also explain underlie the transdiagnostic associations of ELS with psychopathology.

Measurement and Modeling of Early Life Stress

In this last section, I discuss issues that arise in the measurement of ELS and approaches for modeling the associations of ELS with psychopathology and intermediate mechanisms. I end with a brief review of assessment tools that are commonly used to measure ELS.

Assessment of Early Life Stress

A general issue to consider with regard to the assessment of ELS concerns reliability and validity, particularly for retrospective assessments of ELS in adulthood. These issues have been reviewed in depth elsewhere (Hardt & Rutter, 2004; Widom, Raphael, & DuMont, 2004), so I address them here briefly. Methodological studies have generally concluded that ELS exposures can be assessed with reasonable reliability and validity, that prospective assessment in childhood is generally preferable to retrospective assessment in adulthood, and that the primary bias involved in ELS assessments is underreporting of exposure (Hardt & Rutter, 2004; Widom, Raphael, & DuMont, 2004). With regard to reliability, prospective studies have documented high reliability of reports of some ELS experiences (e.g., parental death) but modest reliability for most forms of ELS, including child maltreatment; this moderate reliability is largely driven by high rates of underreporting (i.e., false negatives) (Fergusson, Horwood, & Woodward, 2000; Finlay-Jones, Scott, Duncan-Jones, Byrne, & Henderson, 1981; Widom & Morris, 1997; Widom & Shepard, 1996). Across studies, little evidence has been found for false positives, or false reports of ELS. Underreporting of ELS exposure is relatively unsurprising, and there are numerous reasons why research participants decline to endorse ELS that they have experienced, ranging from a desire for privacy to concerns about protecting caregivers or other people involved. Other factors that contribute to low reliability of ELS reports include memory biases (e.g., forgetting, mood-congruent recall, and poor memory for events in very early childhood) and lack of knowledge of certain forms of ELS even at the time they occurred (e.g., parental SES) (Hardt & Rutter, 2004). The validity of ELS reports is more challenging to evaluate because there is no gold standard assessment to confirm or deny the presence of ELS. Reports of ELS exposure have often been compared to sibling or parent reports and to documented reports of child maltreatment (e.g., by child protective services). Although concordance of self-reports with those of other informants or documented records provides good evidence for the presence of exposure, a lack of concordance does not suggest an absence of exposure. Parent and sibling reports involve the same types of biases present in self-reports of ELS exposure, and only a minority of ELS cases come to the attention of authorities (Brown, Cohen, Johnson, & Salzinger, 1998). Thus, neither of these methods provides a definitive test of validity of ELS reports. On the whole, there is general agreement that ELS reports—particularly

when focused on the presence or absence of certain experiences—have sufficient reliability and validity, even when assessed retrospectively in adulthood (Hardt & Rutter, 2004).

ELS can be assessed using numerous methodological approaches, each of which involves relative strengths and weaknesses. The first issue to consider involves the informant. By far, self-report is the most common method for assessing ELS, followed by caregiver report. Self-reports are generally the preferred approach as many ELS experiences may be known only to the respondent. Moreover, caregivers may be motivated to underreport certain types of ELS experiences (e.g., abuse, harsh discipline). However, caregiver reports may be the preferred approach in some situations, particularly when participants are young children or when there is a possibility that ELS exposure occurred early in development. In studies of children, a combination of both child and caregiver report is advisable, with an “or” rule to combine across informants. Such an approach is the best way to mitigate underreporting on the part of either the child or caregiver. Substantiated records can also be used to assess certain types of ELS, particularly those that fall under the purview of child protective services—including physical and sexual abuse, neglect, and domestic violence. A benefit of this approach is that ELS reports are substantiated, reducing the risk of false positives. A downside is that documented cases of maltreatment reflect only a small proportion of actual cases, many of which never come to the attention of authorities, and reflect only the most severe or frequent exposures. When using documented cases of maltreatment, there is also substantial risk of false negatives in the control group. For example, a seminal longitudinal study of maltreatment defined cases by court-documented maltreatment and required that the control group have no court-documented maltreatment; maltreated children were matched to controls on sociodemographic factors. When maltreatment was assessed in this cohort in adulthood, 49% of the control group reported some form of maltreatment (Widom, Weiler, & Cottler, 1999). Thus, underreporting and false negatives are major concerns when only documented maltreatment is used to define exposure.

A second issue to consider is method of assessment. ELS can be assessed using self-report and caregiver-report surveys and interviews as well as official records from child protective services. Self-report and caregiver report questionnaires are typically either checklists (i.e., yes/no questions about whether the respondent experienced a particular exposure) or Likert-scale assessments of the frequency or severity of particular types of ELS. Checklists have the same inherent limitations as checklist assessments of more general types of stress, including lack of contextual detail and wide variability in the types of experiences that produce a positive endorsement (e.g., a respondent whose parent abstained from alcohol during her entire childhood due to alcohol abuse prior to the respondent’s birth and a respondent whose parent drank heavily throughout his entire childhood may each endorse having a parent with a drinking problem on a checklist) (Dohrenwend, 2006). On the other hand, there is general agreement that assessments about the presence or absence of ELS exposures are more valid than assessments of the timing, frequency, or severity of those experiences when the recall interval is long (Hardt & Rutter, 2004), suggesting that checklist-type assessments may

be preferable for retrospective assessment of ELS in adulthood. Questionnaires designed to assess the frequency or severity of ELS exposure are generally preferable to checklists for assessment in childhood as they constrain the degree of intracategory variability common in checklist methods, and they are better suited for designs utilizing a dimensional approach to conceptualizing ELS. ELS can also be assessed using interview-based assessments, which allow for greater details to be collected about the specific experiences being endorsed. Some of the limitations of ELS checklists can be mitigated by administering them in an interview format that allows for follow-up questions to probe the specific nature of experiences participants endorse. Contextual threat interviews can be adapted to assess ELS, although these interviews were generally not designed to assess ELS specifically. Contextual threat interviews have the advantage of more objective coding of the severity of ELS exposures (Rudolph & Hammen, 1999), but they are limited in the sense that maltreatment and other more severe types of ELS are not typically probed directly, which is likely to magnify underreporting. Contextual threat interviews also tend to focus on circumscribed periods of time (e.g., the past 6–12 months), limiting their utility if the goal is to assess exposures across a longer time interval.

Overall, many methods are available for assessing ELS, and the choice of a measure needs to be determined by the specific research question with attention to balancing the strengths and limitations of different approaches. I advocate the use of both questionnaires and interviews. We routinely encounter children in my lab who are reluctant to report ELS on a questionnaire but are more forthcoming in an interview, as well as children who will not disclose the details of ELS experiences to an unfamiliar interviewer but are willing to endorse items on a questionnaire. Inclusion of multiple assessment methods and multiple reporters typically reduces the likelihood of false negatives to the greatest degree possible.

Early Life Stress Measures

Numerous measures have been developed to measure ELS. Here I highlight the most widely used and psychometrically sound measurement tools for assessing this construct. Specifically, Table 2 summarizes commonly used and validated measures of ELS. This is provided as a reference for those interested in incorporating measures of ELS into their research. The table reviews the domains of ELS assessed by the measure, method of administration (i.e., questionnaire vs. interview), and informant (i.e., self-report or caregiver report) and provides citations for existing psychometric evidence. Additionally, I note how the measure can be used to model cumulative risk and/or dimensions of adversity. Many of the measures used to assess dimensions of adversity could also be utilized within a cumulative risk approach. To do so simply requires selecting a threshold to turn a dimensional measure into a dichotomous indicator of exposure (e.g., determining the threshold of emotional abuse that qualifies as exposure). In general, however, I would caution against study-specific or ad hoc determinations of what qualifies

as the presence or absence of exposure—a key limitation with cumulative risk models (Evans et al., 2013).

Table 2 Commonly used measures of early-life stress/childhood adversity

Measure	Informant	Type of Assessment	ELS Exposures Assessed	ELS Variable Types	Alignment with Cumulative Risk and Dimensional Models	Key Citations and Psychometric Evaluations
Childhood Trauma Questionnaire (CTQ)	• Child Report	• Questionnaire • Likert-scale assessment of frequency of exposure	• Physical abuse • Sexual abuse • Emotional abuse • Physical neglect • Emotional neglect	• Continuous variable representing frequency of exposure to each ELS domain • Validated cut-offs exist for generating dichotomous indicators for each domain	• Dimensional – assessment of both threat (abuse sub-scales) and deprivation (physical neglect sub-scale); emotional neglect sub-scale does not reflect either dimension	• Bernstein, et al. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry, 151*, 1132–1136.

				(Walker et al., 1999)	• Cumulative risk – each sub-scale can be dichotomized based on validated thresholds	• Bernstein et al. (1997). Validity of the Childhood Trauma Questionnaire in an adolescent psychiatric population. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*, 340–348.
Juvenile Victimization Questionnaire (JVQ)	• Child Report • Parent Report	• Questionnaire • Checklist	• Conventional crime (e.g., theft)	• Dichotomous variable representing exposure to		

			• Physical abuse • Sexual abuse/ assault • Emotional abuse • Neglect • Indirect victimization /witnessing violence (including domestic violence) • Peer and sibling victimization	each ELS domain • Dichotomous exposure variables are summed to create variety scores (i.e., number of exposures) in different domains (e.g., child maltreatment, direct and indirect violence exposure)	• Dimensional – assessment of both threat (abuse sub-scales) and deprivation (physical neglect sub-scale); emotional neglect sub-scale does not reflect either dimension • Cumulative risk – each sub-scale can be dichotomized based on validated thresholds	• Finkelhor, D., Ormrod, R., Turner, H., & Hamby, S. L. (2005). The victimization of children and youth: A comprehensive, national survey. *Child Maltreatment, 10*, 5–25. • Hamby, S. L., & Finkelhor, D. (2004). The Comprehensive Juvenile Victimization Questionnaire. Durham, NH: University of

						New Hampshire.
Adverse Childhood Experiences (ACES) Questionnaire	• Self-Report (designed for adult reporting)	• Questionnaire • Checklist	• Physical abuse • Sexual abuse • Emotional abuse • Neglect • Parental divorce • Domestic violence • Parental psychopathology and substance abuse • Parental criminal behavior	• Dichotomous variable representing exposure to each ELS domain • Dichotomous exposure variables are summed to create cumulative ACE score	• Cumulative risk – a cumulative risk score reflecting a count of the number of ELS domains experienced	

						• Anda, R. F., Felitti, V. J., Bremmer, J. D., Walker, J. D., Whitfield, C. L., Perry, B. D., … Giles, W. H. (2006). The enduring effect of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical*

						Neuroscience, 256, 174–186.

						• Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., … Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of*

						Preventive Medicine, 14, 245–258.
Childhood Experiences of Care and Abuse Interview or Questionnaire	• Child Report	• Interview • Assesses presence of exposure as well as frequency and severity	• Physical abuse • Sexual abuse • Emotional abuse • Neglect • Parental loss or separation • Domestic violence**	• Dichotomous variable representing exposure to each ELS domain • Continuous variable for frequency of emotional abuse and neglect • Continuous variable for severity of physical and sexual abuse	• Dimensional – assessment of both threat (abuse sub-scales) and deprivation (neglect sub-scale) • Cumulative risk – each ELS domain can be dichotomized based on validated thresholds (see www.ceca.in	• Bifulco, A., Brown, G. W., & Harris, T. O. (1994). Childhood Experiences of Care and Abuse (CECA): a retrospective interview measure. *Journal of Child Psychology and Psychiatry, 35*, 1419–1435.

			The original measure does not assess domestic violence, but items have been added to the interview and are available upon request by the author of this chapter		terview.com for scoring details)	• Bifulco, A., Bernazzani, O., Moran, P. M., & Jacobs, C. (2005). The childhood experience of care and abuse questionnaire (CECA.Q): Validation in a community series. *British Journal of Clinical Psychology, 44*, 563–581.
Traumatic Events Screening Inventory (TESI)	• Child Report • Parent Report	• Questionnaire or Interview	• Physical abuse • Sexual abuse	• Dichotomous variable representing	• Dimensional – assessment of threat	

		• Checklist followed by severity assessment	• Domestic violence • Community violence • Accidents • Injuries and hospitalizations	exposure to each ELS domain	• Cumulative risk –a count of the number of ELS domains experienced	• Ippen, C. G., Ford, J., Racusin, R., Acker, M., Bosquet, M., Rogers, K., … Edwards, J. (2002). Traumatic Events Screening Inventory - Parent Report Revised.

						• Ribbe D. Psychometric review of Traumatic Event Screening Instrument for Children (TESI-C) In: Stamm BH, editor. Measurement of stress, trauma, and adaptation. Lutherville, MD: Sidran Press; 1996. pp. 386–387. https://www.ptsd.va.gov/professional/assessment/child/tesi.asp

Conflict Tactics Scale (Parent-Child version)	• Parent report (parent-child version)	• Questionnaire • Likert-scale assessment of frequency of exposure	• Physical abuse • Sexual abuse • Emotional abuse/verbal aggression • Corporal punishment • Neglect	• Continuous variable for frequency of exposure to each ELS domain	• Dimensional – assessment of both threat (abuse sub-scales) and deprivation (neglect sub-scale)	• Straus, M. A., Hamby, S. L., Finkelhor, D., Moore, D. W., & Runyan, D. (1998). Identification of child maltreatment with the Parent-Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. Child abuse

						& neglect, 22(4), 249–270. • Bennett DS, Sullivan MW, Lewis M. Relations of parental report and observation of parenting to maltreatment history. Child Maltreatment. 2006; 11:63–75.
Traumatic Experiences Checklist (TEC)	• Child Report	• Questionnaire • Checklist	• Physical abuse • Sexual abuse • Emotional abuse	• Dichotomous variable representing exposure to	• Dimensional – assessment of threat dimension	

			• Emotional neglect • Parental loss or separation (including divorce) • Parental psychopathology • Injuries and accidents	each ELS domain • Continuous severity scores	• Cumulative risk – count of the number of ELS domains experienced	• Nijenhuis, E. R., Van der Hart, O., & Kruger, K. (2002). The psychometric characteristics of the Traumatic Experiences Checklist (TEC): First findings among psychiatric outpatients. Clinical Psychology & Psychotherapy, 9(3), 200–210.
Violence Exposure Scale		• Interview				

for Children - Revised	• Child Report • Parent Report	• Likert-scale assessment of frequency of exposure	• Direct violence exposure/ victimization • Indirect violence exposure/ witnessing	• Continuous variable for frequency of exposure to each ELS domain	• Dimensional – assessment of threat dimension	• Raviv, A., Erel, O., Fox, N. A., Leavitt, L., Raviv, A., Dar, I., … Greenbaum, C. W. (2001). Individual measurement of exposure to everyday violence among elementary schoolchildren across various settings. *Journal of Community Psychology, 29*, 117–140.

						• Raviv, A., Raviv, A., Shimoni, H., Fox, N. A., & Leavitt, L. A. (1999). Children's self-report of exposure to violence and its relation to emotional distress. *Journal of Applied Developmental Psychology, 20*, 337–353.
Screen for Adolescent Violence Exposure (SAVE)	• Child Report	• Questionnaire • Likert-scale	• Direct violence exposure/ victimization	• Continuous variable for frequency of exposure to	• Dimensional – assessment of threat dimension	

KID-SAVE (for children 3rd–7th grade)		assessment of frequency of exposure	• Indirect violence exposure/ witnessing • Physical abuse • Emotional/ verbal abuse	each ELS domain		• Hastings, T. L., & Kelley, M. (1997). Development and validation of the Screen for Adolescent Violence Exposure (SAVE). Journal of Abnormal Child Psychology, 25(6), 511–520.

						• Flowers, A. L., Hastings, T. L., & Kelley, M. L. (2000). Development of a screening instrument for exposure to violence in children: The KID-SAVE. Journal of Psychopathology and Behavioral Assessment, 22(1), 91–104.

Statistical Analysis of Early Life Stress

Most existing work on ELS and psychopathology has used a between-person approach to study these associations. In other words, ELS is assessed, and some type of ELS score is generated for each subject. That score is entered into a statistical model—typically, some type of regression model—to examine whether the level of ELS exposure is associated with psychopathology or some other outcome. This type of between-person approach can reveal whether participants who have higher levels of ELS exposure are also more likely to exhibit psychopathology. This basic approach is widely used in studies of both the cumulative risk and dimensional model of ELS. The primary distinction is in how the ELS variable is constructed. As reviewed earlier in the chapter, the ELS variable used in studies of cumulative risk represents a count of the number of distinct types of ELS a participant has experienced. To create this count, ELS exposure is dichotomized into present versus absent (i.e., 1 vs. 0) across multiple distinct forms of ELS, and the number of exposures that are classified as present are summed to create the risk score (see Evans et al., 2013). The resulting count score is then entered as a predictor into the regression model. Within a dimensional approach, continuous variables for ELS are constructed within each dimension being examined (e.g., threat, deprivation). These variables can be created either from a single measure of adversity or by standardizing composite scores taken from multiple measures of adversity and averaging or summing the standardized scores. This variable would be entered into a regression model, first independently (i.e., without variables for other dimensions of ELS) and then in a model that also includes a continuous variable representing another dimension of adversity (e.g., deprivation). This approach allows one to evaluate the associations of each dimension with psychopathology or other outcomes when examined in isolation (i.e., not controlling for co-occurring exposures) and after adjustment for co-occurring forms of ELS, the latter of which allows inferences to be made about developmental outcomes that are specifically associated with a particular dimension of adversity over and above other co-occurring types of ELS.

Person-level approaches have also been utilized in studies of ELS and psychopathology, typically latent class analysis or some other type of cluster-based modeling (Ford, Elhai, Connor, & Frueh, 2010; McChesney, Adamson, & Shevlin, 2015). The goal of these types of person-level approaches is to identify how different forms of ELS cluster within children and to identify meaningful groups of children who have experienced the same types of adversity. When applied to experiences of ELS, latent class models typically generate a handful of groups (or classes) that differ both in terms of the types and severity of ELS experiences. One must then evaluate and describe how the clusters differ from one another. For example, a latent class analysis of trauma exposure in a nationally representative sample of US adolescents revealed four classes that were described as low risk (i.e., relative absence of trauma exposure), sexual assault, nonsexual trauma, and high risk (i.e., exposure to multiple traumatic events) (McChesney et al., 2015). Class membership can then be associated with mental health or developmental outcomes. Person-level approaches are considerably less common than regression-based between-person models. They can be useful for determining how different types of ELS cluster

within children in a particular sample, but the solutions are inherently study specific and generally do not replicate in other samples. Clustering is often based on multiple ELS characteristics, and thus clusters can reflect combinations of the type of ELS experienced and the frequency and severity of exposure simultaneously. This can create challenges for studying the links between clusters and developmental mechanisms. In general, person-level modeling is useful for understanding the within-person clustering of ELS but is a less useful strategy for examining associations between ELS experiences and downstream outcomes, including psychopathology.

An emerging analytic approach that has not yet appeared in published studies of ELS and psychopathology but is being pursued in several research groups is network analysis. Network analysis methods are used in a wide range of scientific disciplines (Barabasi, 2011) and have increasingly been applied to the study of psychopathology (Borsboom & Cramer, 2013; McNally, 2016). These methods allow relationships to be quantified within complex systems. Although detailed description of network analysis is beyond the scope of this chapter, at core a network is a set of elements—referred to as nodes—that are related to one another through connections, referred to as edges. As applied to psychopathology, nodes are typically specific types of symptoms and the associations among these symptoms (i.e., edges) can be modeled in a variety of ways to understand the relationships among symptoms in the network (Borsboom & Cramer, 2013; McNally, 2016). Network analysis relies on an association matrix of the nodes in a network and provides numerous tools for understanding that underlying set of associations. Such an approach provides a tool for examining some of the predictions of a dimensional model of ELS, which posits that certain dimensions of ELS will be associated with particular developmental processes, but not others (McLaughlin, Sheridan, & Lambert, 2014; Sheridan & McLaughlin, 2014). These questions can be examined by treating ELS exposures and the developmental mechanisms of interest as nodes of the same network. Community detection algorithms can be used to determine whether the predicted associations of particular dimensions of ELS with specific developmental outcomes are observable within the network, while simultaneously adjusting for co-occurring dimensions of ELS and other mechanisms. Such an approach has the potential to generate innovative new directions in understanding the developmental mechanisms linking ELS with psychopathology outcomes.

Conclusion

Early life stress is among the most potent risk factors for the onset of psychopathology. Children who experience ELS are more likely to develop virtually all commonly occurring forms of psychopathology than children who have never been exposed to ELS. Identifying the developmental mechanisms that underlie the strong links between ELS and mental health outcomes is an active area of research within the ELS literature. Recent advances in the conceptualization and modeling of ELS are poised to facilitate improved

understanding of these developmental pathways. Greater knowledge of these mechanisms is required in order to develop more effective interventions to prevent the onset of psychopathology in children who have experienced ELS, a pressing issue for the field.

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