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Social Structure, Adversity, Toxic Stress, and Intergenerational Poverty: An Early Childhood Model

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Keywords

educational attainment, social inequality, allostatic load, child development, epigenetics, life course

Abstract

Why are children of poor parents more likely to be poor as adults than other children? Early-childhood adversities resulting from social structures and relationships impact children's bodily systems and brain development through recurrent stress. These socially patterned biological processes influence social reproduction. Social support and interventions can prevent or compensate for the early biological effects of toxic social environments. This article integrates sociological, neuroscience, epigenetic, and psychological evidence to build a model of early-childhood developmental mechanisms contributing to intergenerational poverty. This model captures ways in which social structures interact with biological characteristics and systems to shape life trajectories.

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INTRODUCTION

Children raised in impoverished families are much more likely than other children to be poor as adults (Corcoran 2001, Duncan et al. 1998, Putnam 2015, Sharkey 2008, Sharkey & Elwert 2011, Wagmiller & Adelman 2009). What accounts for this pattern and more generally for patterns of social replication that make parents' social class predictive of the social class destinations of their children? And because many poor children do not end up poor as adults, what differentiates their trajectories from those of others growing up under similar economic circumstances? Sociologists have addressed these issues in varied but weakly integrated ways (e.g., Bozick et al. 2010, Corcoran 1995, Small et al. 2010, Wilson 2010). Sociological approaches also fall short in specifying mechanisms by which social structures, community resources, and family backgrounds both create patterned differences and differentially affect individual life trajectories. This article deepens sociological understanding of intergenerational poverty by paying close attention to early-childhood development and by incorporating new research from neuroscience, epigenetics, and child development. This research demonstrates that crucial biological processes that influence life chances depend upon social structures and processes.

Research from the emerging field of "developmental neuroscience of early stress and disadvantage" makes clear that social conditions and interpersonal experiences produce adversities that generate toxic stress (Boyce et al. 2012, p. 17143). Such stress, in turn, affects early-childhood brain and body development in ways likely to be consequential for later educational and occupational attainment (Boyce et al. 2012). This research thus examines biological processes not as primary causes of social outcomes but rather as mechanisms that depend on social structures, relationships, and interactions.

Our review integrates this work with research drawn selectively from varied areas of sociology: stress and mental health, poverty, status attainment, education, family, neighborhood, and life course development. It elaborates an early-childhood toxic stress model that adds significantly to sociological accounts by further identifying mechanisms for intergenerational transmission of social inequality. In doing so, it challenges sociologists interested in the intersection between their discipline and biology to broaden their horizons beyond the current focus on genetic factors (e.g., Conley 2016, Guo & Stearns 2002, Pescosolido et al. 2008, Shanahan et al. 2008) to examine epigenetic processes involved with gene-environment interactions in brain development and function over the life course and their relationships with neuroendocrine systems.

CONNECTING ADULT ATTAINMENT TO EARLY CHILDHOOD

"Explanations for patterns of social inequality are only as strong as the models of educational attainment on which they depend" (Morgan 2005). Evidence abounds to support this perspective. Research has documented gradations by social class of origin in educational attainment, school performance, cognitive skill, behavioral problems, and IQ (Hackman & Farah 2009, McLeod & Kaiser 2004, McLoyd 1998). Recent evidence indicates that the gap in school performance between students from lower-income families and students from higher-income families is both large and growing significantly in the United States (Reardon 2011). Furthermore, using data from the Panel Study of Income Dynamics, Duncan and colleagues show "statistically significant and, in some cases, quantitatively large detrimental effects of early poverty on a number of [other] attainment-related outcomes" such as adult earnings and work hours (Duncan et al. 2010, p. 306; see also Alexander et al. 2014).

An understanding of differences in educational and adult attainment requires a focus on children's development during their first years in part because "cognitive ability is significantly shaped

early in the life course" (Sampson et al. 2008). Crucial noncognitive skills that have significant impact on life course outcomes are also developed in the early years of life (Farkas 2003, Heckman et al. 2014, Jones et al. 2015). Research confirms large disparities in school readiness of children, depending on parental education and income (Farkas & Hibel 2008), and the predictive power of first-grade school performance with regard to later educational attainment (Entwisle et al. 2005). Sociologists, however, have paid little attention to early-childhood development in theorizing about status attainment and intergenerational poverty and have only partially identified mechanisms that translate social structures into patterned but varying individual trajectories.

IDENTIFYING MECHANISMS LINKING EDUCATIONAL ATTAINMENT TO FAMILY SOCIAL POSITION

To explain intergenerational poverty, sociologists must go beyond identifying correlations "between structural positions . . . and personal attributes. To advance our understanding of causal processes, we must understand the proximal conditions or stimuli through which structural positions influence the individual" (House & Mortimer 1990, p. 75). Research connecting neighborhood poverty to life trajectories illustrates this need. Sharkey & Elwert (2011) reported that children's cognitive performance declines by half a standard deviation when their families have been exposed to neighborhood poverty for two generations. Sampson and colleagues (2008) demonstrated the negative effects on verbal ability of growing up in concentrated poverty, and Sastry & Pebley (2010) showed that average neighborhood income strongly predicts children's reading and math scores. Other research has established that sustained residence in disadvantaged neighborhoods significantly reduces the likelihood of high school graduation (Wodtke et al. 2011). These researchers describe four mechanisms working to create this pattern of outcomes: isolation from supportive social networks, loss of mutual trust and control over youth behavior, inadequate public and school resources, and harm from environmental hazards. Although important, these mechanisms miss key biological processes and do not clearly address individual variation within impoverished communities.

Sociologists have taken several routes to identifying mechanisms by which social structures and conditions translate into variable individual life chances. Reengagement with cultural explanations of poverty (e.g., Lareau 2011, Small et al. 2010, Wilson 2010) represents one such approach. The status attainment research tradition exemplifies another effort to identify mechanisms that "specify the causal sequence through which individuals reach their positions in status hierarchies" (Haller & Portes 1973, p. 55). Genetic inheritance (Adkins & Vaisey 2009) has recently been added to the social psychological mechanisms—educational and occupational expectations or aspirations—that are the usual focus of this approach (see also Conley 2016).

In this article, we propose a model that incorporates both biological mechanisms and social structural, cultural, and status attainment approaches to explaining social reproduction. Rather than weakening sociological explanations, the introduction of biological mechanisms relating to toxic stress strengthens these explanations by recognizing biological processes, for example, epigenetic mechanisms (Landecker & Panofsky 2013, Meaney 2010), through which social inequalities become embodied, especially in the early-childhood years (Krieger 2005). Although sociologists have long resisted examining biological accounts because of concerns about reductionism, some have begun to examine genetic evidence in empirical studies of gene-environment interaction (Guo & Stearns 2002, Pescosolido et al. 2008, Shanahan et al. 2008) in which genetic characteristics appear fixed but play out differently under different conditions. The emergence of epigenetics (Landecker & Panofsky 2013) and of neuroscientific research on brain development and brain plasticity, however, makes clear that social environments play important roles in shaping and

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reshaping biological characteristics that in turn influence social behavior. This review emphasizes this latter view of biological mechanisms.

THE TOXIC STRESS MODEL: AN OVERVIEW

Toxic stress is the central biological mechanism in an emerging neuroscientific theory of the ways in which social circumstances, experiences, and relationships shape and reshape brain and body development, especially in early childhood, with resultant effects on later educational and occupational attainment as well as on health. Toxic stress involves the frequent or sustained activation of the biological stress system and is prompted by chronic social conditions and repeated or accumulating adverse events when social support systems are weak and when early-life experiences acting epigenetically have impaired the development of neural circuits involved in self-regulation of emotions and behavior. Health-promoting and health-damaging behaviors resulting from such conditions activate these same biological systems and contribute to their consequences. Although toxic stress affects the body and brain throughout the life course (Lupien et al. 2009), its effects during early development are particularly profound and are the focus of our model (see **Figure 1**).[1]

Figure 1 shows that adversities resulting from social position—particularly sustained early childhood poverty—and from other sources[2] such as abusive treatment or mental illness of a parent can lead to sustained bodily stress reactions in infants and young children. Toxic stress overloads the body's capacity to maintain homeostasis and creates an unbalanced physiological state (allostatic load/overload). This overload affects crucial aspects of brain development in childhood and impacts other body systems, often in ways that last a lifetime, with implications for cognitive performance, self-regulation, and physical and mental health. The effects of toxic stressors and stress are influenced by social factors, in particular the nature of caregiving (for example, its warmth or harshness) as well as other social supports and resources. Both the susceptibility of individuals to toxic responses to stressors and the nature of brain development are also affected by inherited genetic characteristics and experientially induced changes in gene expression (epigenetic factors) that we describe below. This article unpacks this model, connects it to sociological research and theory, and explicates the assumptions or evidence underlying the causal arrows in **Figure 1**.

This toxic stress model relates to the stress process paradigm that has dominated sociological analyses of mental health (Pearlin et al. 1981, Wheaton et al. 2013). That model links chronic and acute stressors to individual psychosocial resources and mental health outcomes in the context of social and economic structures and statuses (Pearlin & Bierman 2013). Our toxic stress model significantly widens the behavioral outcomes of the stress process to include cognitive functioning and varied dimensions of self-regulation as well as health behaviors and physical and mental health that relate to later status attainment. Our model's focus on early-child development also differentiates it from the stress process paradigm.

The model also incorporates biological processes that sociologists in the stress process tradition have resisted including. Their resistance stems in part from the view "that biologists will have little interest in the source of stress" (Pearlin 1989, p. 243). Researchers in the new developmental

[1]This model considerably elaborates on one proposed by Evans et al. (2011): poverty → cumulative risk exposure → chronic stress → low achievement. It also links closely to Massey's (2014) biosocial model of racial stratification: socioeconomic inequality and racial segregation → concentrations of poverty and violence → high allostatic load → coronary heart disease, inflammatory disorders, and cognitive impairment.

[2]Many of the other stressors that appear to affect early-child development—e.g., maternal depression, child abuse and neglect, spousal violence—are more likely to exist in low-income households (e.g., Knitzer et al. 2008, Straus et al. 2006).

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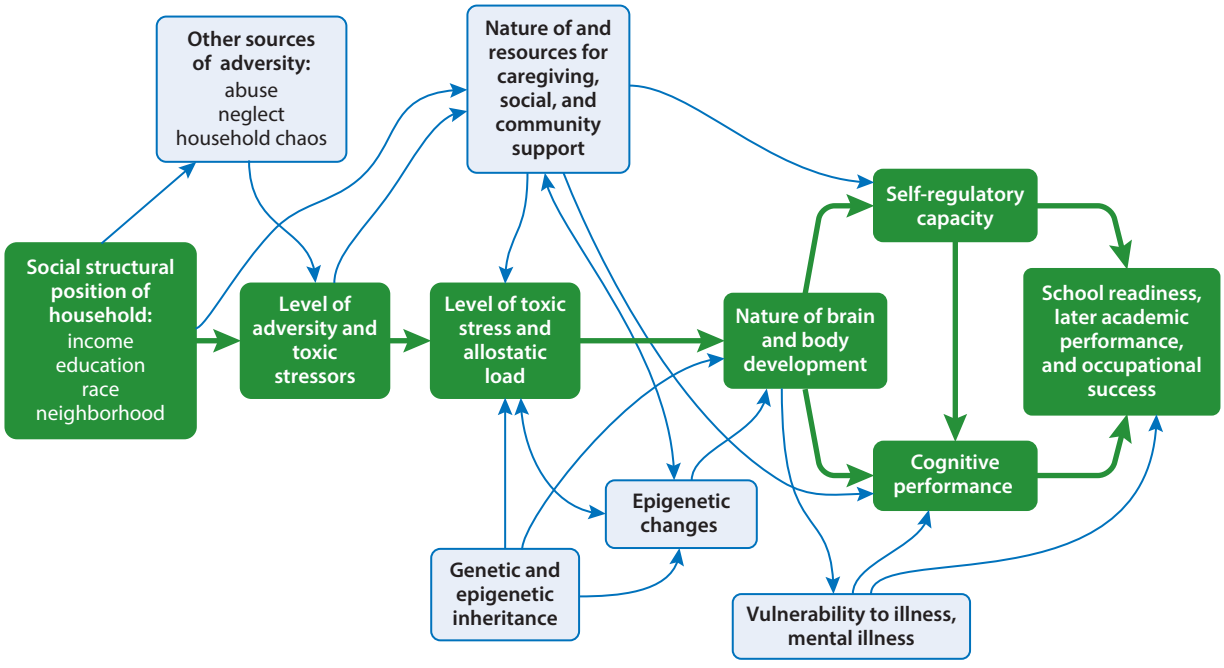

Figure 1

A model of social structure, social supports, adversity, toxic stress and brain/body development in early childhood. Social circumstances (economic, neighborhood, household, and other) affect levels of adversity, which in turn cause toxic stress that results in allostatic load if strong, positive social supports are unavailable. Toxic stress and resulting allostatic load affect brain and body development and function in childhood, partially through epigenetic changes mediated in part by the neuroendocrine system. Genetic and epigenetic factors also affect the degree of individual sensitivity to toxic stress. The effects on brain and body development diminish emerging self-regulatory capacity and cognitive performance and increase vulnerability to physical and mental illness. Together, these effects reduce school readiness and later academic performance. Given the plasticity of the developing brain, many of these effects are reversible through social intervention.

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science of childhood adversity, however, have shown great interest in the social sources of toxic stress (e.g., Boyce 2007, Boyce et al. 2012). Our model, unlike the stress process paradigm, reflects this emerging science and defines toxic stress and allostatic load in terms of biological indicators.

Attention to the biological mechanisms and consequences of toxic stress emphasizes the vital importance of early-childhood development—the social conditions shaping it and their direct and indirect consequences for circumstances such as maternal depression and household chaos. It also highlights relational resources such as (*a*) maternal attachment; (*b*) social support networks; and (*c*) consistent, high-quality child care that can protect against the effects of toxic stressors or that, because of the brain's plasticity, can later reverse some of the effects of such stressors.

BIOLOGICAL MECHANISMS OF TOXIC STRESS

Biological stress responses to challenges that we often term stressors are generally healthy and protective because they temporarily adjust bodily processes—resulting in increased heart and breathing rates, release of glucose and insulin for metabolism, and immune system activation or suppression—in response to environmental circumstances experienced as potentially threatening.

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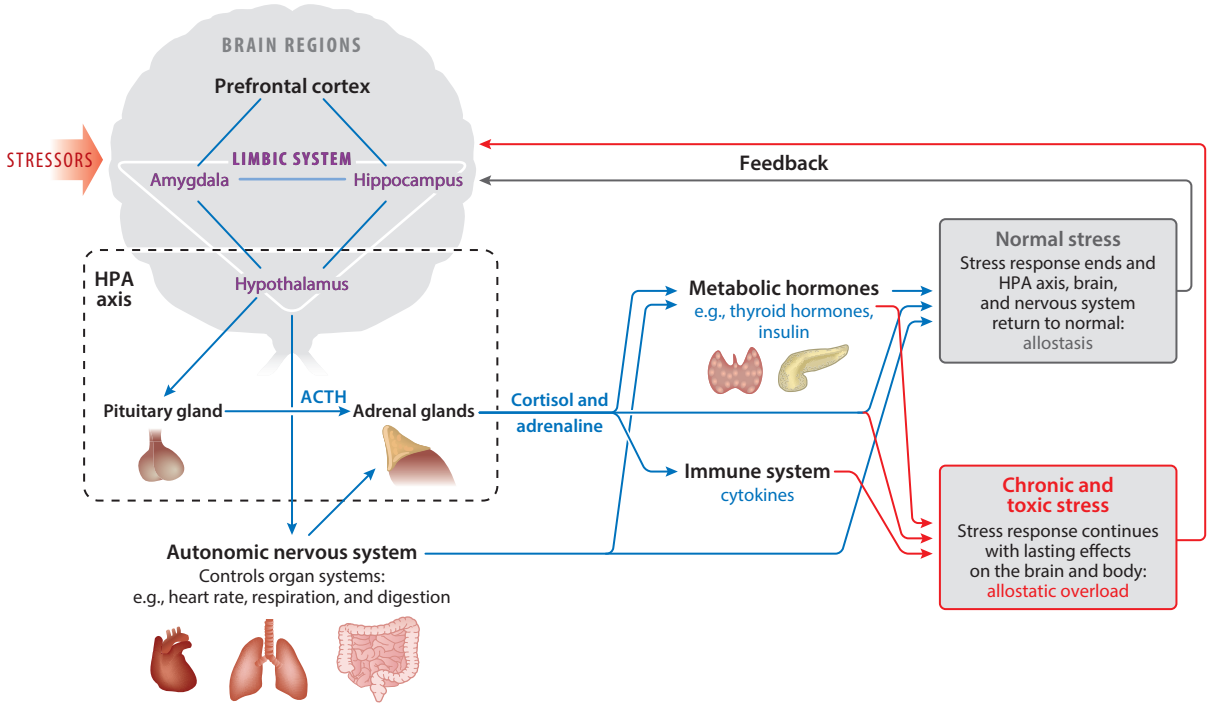

Figure 2

Normal and toxic stress processes and effects on brain and body. Acute physical and psychological stressors activate the autonomic nervous system via the amygdala, hypothalamus, prefrontal cortex, and hippocampus and also activate ACTH (adrenocorticotropic hormone) release from the pituitary gland and production of cortisol by the adrenal glands. These hormones affect the autonomic nervous system (e.g., heart rate and respiration increases). Stressors also alter the release of metabolic hormones and pro- and anti-inflammatory cytokines of the immune system and neurotransmitters in the brain. These responses are carefully coordinated and shut off when no longer needed; they collectively promote adaptation to events in daily life by a process referred to as allostasis. When stressors become chronic and particularly when the individual lacks adequate support and control, they become toxic and lead the same mediators that promote adaptation instead to cause wear and tear on the body and brain that can accelerate disease processes. This process is referred to as allostatic load and overload. The brain is a major target of these mediators. Neural architecture—particularly in the prefrontal cortex, hippocampus, and amygdala—is altered by acute and chronic stressors during early childhood, with consequences for behavior and for the regulation of allostasis (Lupien et al. 2009). HPA axis denotes hypothalamic–pituitary gland–adrenal gland axis.

Consciously and unconsciously perceived threats and resultant stress activate the hypothalamus, a brain center that regulates the neuroendocrine and autonomic nervous systems (see **Figure 2**).

The hypothalamus in turn signals the pituitary gland to release ACTH (adrenocorticotropic hormone), which stimulates the adrenal glands to produce cortisol. Cortisol works interactively with other hormones and biochemicals released into the bloodstream—adrenaline, immune system cytokines (protein messengers between cells), metabolic hormones such as insulin, and neurochemicals in the brain—to promote adaptation and survival (McEwen 1998). In this complex network, the effects of increases or decreases in one hormone or biochemical are never linear and depend, among other things, on the levels of other hormones and biochemical in this nonlinear regulatory network. The interactive system of feedback among the hypothalamus and the pituitary and adrenal glands is termed the HPA axis.

Toxic stress, unlike normal stress, involves the frequent or sustained activation of this HPA axis and of other interacting systems. Protective buffers provided by supportive and nurturing

interpersonal relationships and healthy lifestyle behaviors (for example, regular exercise and good diet) prevent or reduce toxic stress in the face of continuing stressors. Toxic stress is hugely consequential: "It disrupts brain architecture, affects other organ systems, and leads to stress-management systems that establish relatively lower thresholds for responsiveness that persist throughout life, thereby increasing the risk of stress-related disease and cognitive impairment well into the adult years." (Shonkoff et al. 2009, p. 2256).

When not counterbalanced, the effects of toxic stress can be seen in a wide array of diseases, such as cardiovascular disease, arthritis, asthma, diabetes, arthritis, and depressive illness (e.g., Boyce 2007, Wolfe et al. 2012, Ziol-Guest et al. 2012). The toll of toxic stress also includes a higher likelihood of drug abuse, depression, alcoholism, and smoking (Anda et al. 2002, Dube et al. 2003, Felitti & Anda 2010). But the implications of the relationships among toxic stress, poverty, and adversity extend well beyond health to altered brain architecture—especially early in life—that ultimately contributes to intergenerational poverty when there are no interventions to overcome those early effects.

The active process of adaptation in the HPA network (allostasis) maintains homeostasis in the body. Efficient turning on and off of responses to stress lead to optimal adaptation to anticipated and unanticipated events. However, excessive activation of this system caused by frequent or chronic stressors distorts the normal balance of the components of allostasis (e.g., too much or little cortisol, adrenaline, cytokines, metabolic hormones, or neurotransmitters). It leads to wear and tear on the brain and body that alters the architecture of the prefrontal cortex as well as other brain regions. This repetitive spiral is termed allostatic load and overload (overload refers to the most severe effects).

Allostatic load has many dimensions and should be measured by using indicators from various bodily systems affected by toxic stress—for example, the neuroendocrine, immune, autonomic, and metabolic systems (Seeman et al. 2010)—as well as markers of its secondary effects such as body mass index. Evidence clearly suggests that indicators of allostatic load correlate with neighborhood poverty independently of household socioeconomic status (SES) (Schultz et al. 2012) and that allostatic load is likely to remain higher among research subjects who experienced poverty during childhood independently of their current SES (Miller et al. 2009).

POVERTY, ADVERSITY, AND THE CONSEQUENCES OF TOXIC STRESS

Chronic and episodic environmental stressors have been of central interest to those concerned with toxic stress and allostatic load (Gianaros & Manuck 2010).[3] The unfolding theory of the relationship during early childhood between these stressors and the development of varied brain regions, cognitive function, and self-regulation carries significant implications for sociological understanding of intergenerational poverty.

Current research in neuroscience documents "the realities of SES disparities in neurocognitive function . . . and provide[s] more direct evidence of the involvement of [the] prefrontal cortex in the observed SES disparities" (Hackman & Farah 2009, p. 68). The prefrontal cortex controls decision making, working memory, impulsivity, and emotion (Casey et al. 2011, McEwen & Morrison 2013). Its slow development makes it especially vulnerable to toxic early-life stress but

[3]Racism, racial stratification, and segregation also appear to be chronic stressors (Brondolo et al. 2009, Clark et al. 1999, Massey 2014, Sternthal et al. 2011) but have received little attention in neuroscience research, although studies clearly link reported racism to the health and well-being of children and adolescents (Priest et al. 2013).

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also subject to rewiring through later interventions (Pechtel & Pizzagalli 2011). For example, Hanson and colleagues (2013) showed that the growth of gray matter in the brain is slower for infants from poor families than for infants from families with more resources. The neural circuitry in the prefrontal cortex plays a central role in managing responses to stress, but toxic stress alters its structure and functioning through the release of hormones and neurotransmitters by nerve cells (McEwen & Morrison 2013). The consequences appear to include compromised capacities for behavioral and emotional self-regulation, including working memory and executive function (Shonkoff 2012).

The capacities for self-regulation and cognitive performance constitute two key early-life outcomes identified in the toxic stress model that closely relate to school readiness and performance. Self-regulation includes attention, persistence, delay of gratification, short-term memory, and capacity to mobilize information to solve problems and accomplish goals (Berger 2011, Ursache et al. 2012). One general aspect of self-regulation is executive function: control of attention, planning, organizing and sequencing thinking and actions to accomplish goals, making transitions from one activity to another, and deploying working memory to accomplish cognitive tasks and problem solving.

A second general aspect of self-regulation involves control of impulses and emotions (Ursache et al. 2012). The capacity to employ cognitive approaches to control impulsivity, regulate emotional expression, and hold back inappropriate behavior plays a significant role in social competence and adjustment (Berger 2011). For example, children with weak regulatory skills tend to have difficulties with peers and are likely to challenge adults around them (Eisenberg et al. 1993, 2010).

Cognitive performance involves several neurocognitive systems, including language, working memory, reward processing, and spatial cognition (Farah et al. 2006). Using varied measures, researchers have documented SES differences in several neurocognitive systems. For example, evidence indicates on average lower working memory and language abilities in children from low-income families compared with children from middle-income households (Farah et al. 2006, Noble et al. 2007).

Early-childhood poverty affects the executive function that is so crucial to goal-directed behavior (Blair & Raver 2012, Blair et al. 2011b). A longitudinal study of a sample of rural, low-income New York children found that their working memories at age 17 had declined over time in "direct relation to the number of years [they] lived in poverty (from birth through age 13)" (Evans et al. 2011, p. 21; see also Kim et al. 2013). Importantly, this decline occurred only for children who had experienced high levels of toxic stress and allostatic load. In addition, the same research showed that poverty and adversity reduce the capacity of children to delay gratification (Evans & Rosenbaum 2008). Whatever the measures used, strong and consistent research evidence documents statistical relationships between early-life poverty and both reduced cognitive performance and limited capacity for self-regulation (Farah & Hackman 2012).

Evidence also strongly supports the proposition that cognitive performance and self-regulation relate to school readiness and later to school performance (e.g., Entwisle et al. 2005, Evans & Rosenbaum 2008, Farkas 2003, Shoda et al. 1990). For example, Evans & Rosenbaum (2008) concluded that the effects of self-regulation on school achievement are nearly as strong as those of cognitive enrichment in the home. Heckman et al. (2014) argued for the primary importance of what they term "character" (largely self-regulation) relative to cognition in accounting for adult achievement (see also Mischel et al. 1988, Posner & Rothbart 2000).

The self-regulation processes that support resilience and adaptation to adversities are hampered when the developing brain experiences toxic stress (Blair 2010). Deficits in executive function

limit cognitive performance and correlate with problems such as limited effortful attentional control (Dishion & Connell 2006, Liew 2012). Such control helps a child to screen out unrelated thoughts and external cues and to focus on a particular task or goal. Variable across individuals and developing in the first two years, executive function appears to have a genetic component but is also "embedded in family environments that promote self-regulation" (Dishion & Connell 2006, p. 127). A study of single parents by Zalewski et al. (2012) reported altered patterns of cortisol secretion and later lower effortful control among preschool children of single parents who were poor; the authors suggest a model in which poverty impacts parenting (greater negativity), which in turn affects the working of the HPA axis and the development of effortful control. Another study found that children with higher levels of salivary cortisol (an indicator of toxic stress) tended to have lower executive function and that what the authors define as positive parenting correlated with lower salivary cortisol levels (Blair et al. 2011a).

Examining the emotional and impulse control components of self-regulation requires attention to the brain's limbic system, which toxic stress also compromises. This system (see **Figure 2**) includes three brain regions—the hypothalamus, hippocampus, and amygdala—and plays a central role in the expression and regulation of emotion (LeDoux 2000, Phelps 2004). Clearly, the limbic system connects closely to the HPA axis because they share the hypothalamus. In the limbic system, the hypothalamus, with input from the amygdala, governs arousal and physiological responses to stress. The hippocampus helps manage spatial and episodic memory of events related to threats and other daily experiences and also plays a role in control of mood. The amygdala identifies environmental threats and signals the HPA system to activate stress responses. As the top-down agent of self-regulation, the prefrontal cortex helps manage the amygdala and works with the hippocampus to control impulsivity, emotions, and working memory. When the prefrontal cortex is less active, the amygdala becomes overactive, and emotional regulation diminishes. Increased activity of the amygdala, which also activates release of adrenaline and cortisol, can lead over time to allostatic load and overload as well as to emotional problems such as chronic anxiety and depression and behavioral issues stemming from reduced control of anger and aggression (McEwen & Gianaros 2011).

Mounting evidence connects overactivity of the amygdala—and relatedly its volume—with poverty and adversity. For example, research suggests that economic disadvantage correlates with "vigilance for threat," which appears to result from an overly responsive amygdala (Gianaros & Manuck 2010). Other research has shown that children raised in orphanages with resulting deficits in maternal care tended to have enlarged amygdalae (Tottenham et al. 2010), as did children of mothers who were chronically depressed over several years (Lupien et al. 2011). By contrast, Hanson and colleagues (2015) found reduced amygdala volume (as well as smaller hippocampus volume) in children who had experienced early-life stress arising from poverty or from abuse; these researchers point to the need to reconcile inconsistent findings. Toxic stress appears to play a key role in linking poverty and adversity to amygdala volume and amygdala overactivity. For example, the children of mothers depressed since giving birth also showed higher levels of salivary cortisol reactivity during a doctor's visit, one indicator of toxic stress (Lupien et al. 2011).

Like sociological research on stress and mental illness, research on child development in the context of toxic stress does not focus exclusively on poverty as the stressor (Evans et al. 2013). Sociologists have tended to look at varied individual stressors (George 2013) but have not "utilized the models of cumulative advantage and disadvantage that drive much contemporary research on health disparities" (McLeod 2013, p. 244). Such cumulative measures of adversity figure prominently in neuroscience research on toxic stress and allostatic load, however, and help direct attention to their links to social structures, processes, and relationships.

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CUMULATIVE RISK AND DISADVANTAGE

Poverty increases the likelihood of a wide range of adversities that affect the development of human brains when infants and children are not protected by consistent, strongly supportive, nurturing relationships with adults. However, adversities that help create toxic stress extend beyond the conditions of poverty, and "poverty" as a household or neighborhood condition does not inevitably lead to the many specific but variable social and experiential mechanisms that correlate with it and serve as proximal causes of toxic stress. Researchers have attempted in various ways to identify the adversities prompting allostatic overload and recognize that the effects of such adversities accumulate.

Pearlin & Bierman (2013) describe stress proliferation: the ways in which stressors accumulate, grow out of one another, and are sequenced. Proliferation can also be thought of as a cascade: Cumulative, even multiplying, adversity results when "earlier [childhood] disadvantages lead to later disadvantages and to an increasingly compromised capacity to respond to and manage new insults" (O'Rand & Hamil-Luker 2005, p. 117). This view, particularly prominent in studies of physical health over the life course, emphasizes the importance of examining stressors together, not one by one, and of including in cumulative measures those stressors resulting from chronic conditions such as poverty, discrimination, and racism along with disruptive life events or traumas (Pearlin & Bierman 2013).

Measures of cumulative risk help to specify the mechanisms by which broad social conditions such as poverty affect life course outcomes. These measures are based on classifying the presence or absence of each of a series of risk factors for individuals or communities. The risk score is a simple sum of the number of those factors determined to be present (Evans 2003, Wells et al. 2010).

The Adverse Childhood Experience (ACE) study employs this methodology and powerfully demonstrates the implications of cumulative early-life risk for adult physical and behavioral health (Anda et al. 2006, Natl. Cent. Inj. Prev. Control Div. Violence Prev. 2016). Begun in the 1990s with a research population of middle-class individuals, adult subscribers to Kaiser Permanente insurance, the continuing panel study asked more than 17,000 patients to indicate whether or not they had experienced any of ten adverse experiences as children.[4] As the resulting ACE index scores increase from 0 to 10, so also does the likelihood of a wide variety of illnesses such as heart disease, diabetes, and stroke (Felitti & Anda 2010). Similar gradients appear for depression (Chapman et al. 2004) and for health-related behaviors, including smoking (Anda et al. 1999) and drug abuse (Dube et al. 2003). In the years since the study's initiation, it has been recognized that the biological mechanisms translating adverse experiences into health outcomes revolve around toxic stress.

The ACE study is now widely disseminated through trainings of thousands of teachers and health care and human service providers as well as legislators and other public officials. The large body of ACE research, however, remains poorly integrated with the developmental science of childhood adversity. In particular, the focus of the ACE study on middle-class patients has limited the range of childhood adversities examined so that they do not include many adversities resulting from chronic social conditions such as poverty (for example, economic insecurity, unstable housing, fluid household composition, food insecurity, neighborhood violence) (Finkelhor et al.

[4]The ten adversities counted are physical, sexual, and emotional abuse; emotional and physical neglect; household substance abuse and mental illness; parental separation or divorce; domestic violence; and incarcerated family members.

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2013).[5] Unfortunately, in the extensive dissemination of ACE research to nonscientific audiences,[6] the ACE score has often become reified so that the ten ACEs come to stand for all childhood adversities, even though these ACEs incompletely represent the wide variety of childhood adversities. The ACE research resides in the CDC's Division of Violence Prevention (Natl. Cent. Inj. Prev. Control Div. Violence Prev. 2016). The framing of the research in terms of familial abuse and trauma disconnects childhood adversity from its broader social contexts that produce its many forms at varying rates. Attending to social context emphasizes that, in addition to therapeutic interventions, social policy and community reorganization are important preventive responses.

Scholars involved in the developmental science of childhood adversity have taken a much broader view of stressors than found in the ACE research, and their research examines varying elements of social disadvantage and chronic conditions while recognizing their cumulative effects (Burchinal et al. 2000, Evans et al. 2013). For example, the early Rochester Longitudinal Study—examining the transmission of mental health from parents to children who were followed from the perinatal period to age 18—attempted to locate mechanisms by which social class affects children's developmental outcomes (Sameroff et al. 1993). The result was a risk index composed of ten elements including, for example, maternal mental health, anxiety, and education; stressful life events; and family social support.

Research employing six cumulative risk factors (crowding in the home, noise levels, family turmoil, separation of child from parents, exposure to violence, and extent to which housing is substandard) identified biological stress as one mechanism connecting SES-related risks and children's developing cognitive capacities (Evans & Kim 2012). A longitudinal study in rural New York, for example, found that cumulative risk exposure, toxic stress, and allostatic load helped shape cognitive outcomes for children (Kim et al. 2013). The data show "that elevated cumulative risk exposure is a viable explanatory mechanism linking childhood poverty to subsequent chronic stress as children emerge into adulthood" and increased allostatic load in young adults (Evans & Kim 2012, p. 982).

Qualities of neighborhoods clearly figure into cumulative risk. Sampson et al. (2008) found that living in a neighborhood characterized by concentrated disadvantage significantly reduced later verbal test scores, even for children who had moved out of the area. Sociologist Amy Schultz et al. (2012) and colleagues documented a positive association between the extent of neighborhood poverty and allostatic load among Detroit adults. This link was mediated by an index of environmental stressors such as gang violence, theft, air pollution, and condition of housing (Schultz et al. 2012). The notion of cumulative risk has been used to capture school and neighborhood risk factors that mediate the relationship between neighborhood poverty and school-wide achievement (Whipple et al. 2010). Such research suggests that chronic stress and toxic stress are key mechanisms leading to the embodiment of environmental stressors experienced in neighborhoods (Diez Roux & Mair 2010).

Wachs & Evans (2010) theorized that chaos should be added to the cumulative list of mechanisms that impact child development. They argue that chaos has an effect independent of

[5]On the basis of recommendations of a Technical Expert Panel, the National Survey of Children's Health has created an Adverse Childhood Experience measure that more broadly represents childhood adversities. Its nine items include five ACE items along with four others—perceived discrimination, death of a parent, witness/victim of neighborhood violence, and socioeconomic hardship (Porche et al. 2016).

[6]The extent of ACE dissemination is suggested by its replication internationally, the inclusion of an ACE supplementary module for the CDC's Behavioral Risk Surveillance System telephone survey, legislation in Washington and Montana adopting the ACE definition of adversity and seeking support for prevention efforts, many state and city ACE coalitions, and the completion of an award-winning 2016 documentary about the study titled *Resilience*.

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SES, although it also is a mechanism through which poverty affects child development. Chaotic environments—not just in neighborhoods (Dunn et al. 2010) but also, for example, in schools (Maxwell 2010) and child care settings (Corapci 2010)—are unstable ones, often noisy and crowded, in which few or no routines or patterns can be counted on from day to day (Wachs & Evans 2010). The biological mechanisms by which chaos affects child development involve the stress physiology at the center of the toxic stress model (Ackerman & Brown 2010).

Although researchers generally recognize that adversities and their effects accumulate, they do not agree about what the most significant adversities are, how they relate to one another, or how they connect to social contexts and social structures. Some researchers have included poverty or its proxies in indices of adversity or risk, whereas others have tried to find proximate indicators derived from or correlated with poverty. Nonetheless, there is agreement that "traces many of the chronic morbidities, behavioral proclivities, and lasting afflictions of adulthood to experiences of adversity, maltreatment, and subordination sustained over the early years of life" (Boyce et al. 2012, p. 17144). Sociologists can play a key role in sharpening understanding of the range and social distribution of childhood adversities.

PROTECTIVE FACTORS AND RESILIENCE

Social structures, networks, and relationships not only are the sources of adversities and stressors but also can help cushion the impact of stress or reverse its effects. The nature of caregiving for young children and the availability of social network and community supports for them can prevent, moderate, or help overcome the effects of toxic stressors on brain development and on cognitive performance and self-regulation. Sociologists of mental health have long documented the powerful protective role of supportive social networks and nurturing, caregiving relationships (George 2013, Pearlin & Bierman 2013). These networks and relationships also protect against the toxic stressors resulting from social structural position and help sustain healthy brain development (Blair & Raver 2012, Meaney 2010). Protective factors diminish the effects of stressors on individuals at biological, psychological, and social behavioral levels (Luthar et al. 2006). For example, a study of five-year-old twins found that maternal warmth and provision of "stimulating activities," along with genetic inheritance, protect against the consequences of SES deprivation and help build cognitive and behavioral resilience (Kim-Cohen et al. 2004).

Protective factors relate closely to the concept of resilience as a joint product of individual qualities and social circumstances. In this sense, resilience is a set of interconnected processes that constitute the capacity of individuals challenged by adversity to secure the resources they need to experience well-being (Ungar et al. 2013). Positive, supportive interactions with adults in the context of adaptive institutional structures thus play a central role in healthy development (Blair & Raver 2012). On the basis of a review of resilience research, Ungar et al. (2013) hypothesized that the greater the adversity a child has experienced, the more that resilience (coping with adversity) depends on characteristics of the social environment rather than on individual qualities.

This bio-social-ecological perspective (Ungar et al. 2013) highlights multiple, interconnected systems: macrosystems of resource distribution; community, neighborhood, and institutional structures and their programmatic resources; relational networks of families, caregivers, friends, and neighbors; microsystems of interaction among children and between children and others in their social environments; and biological systems affected by and affecting these social systems. Because these systems connect with and depend upon one another, they are mutually reinforcing; access or lack of access to resources at one level often leads to access to or lack of resources at another level (Miller et al. 2011). Thus, resilience at the biological level—the capacity of a person supported by the environment to adapt under stressful circumstances in order to maintain

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homeostasis and minimize allostatic load—depends heavily on the nature of social relationships and resources (Karatoreos & McEwen 2013).

The examination of protective factors and resilience provides an important counterweight to any suggestions of biological or social determinism (Karatoreos & McEwen 2013, Ungar et al. 2013). The fates of children are not sealed in the first months and years of life, and as important as early experiences are to brain development, the plastic brain can be reprogrammed in response to new circumstances and experiences (Bavelier et al. 2010, McEwen & Gianaros 2011). At the same time, genetic factors cannot be ignored; they help shape individual personality characteristics such as temperament (Kim-Cohen et al. 2004) and increase or decrease sensitivity to the effects of both positive and negative experiences in the environment (Ellis & Boyce 2011). Clearly, however, protective factors and resilience for young children are strongly embedded in the nature of caregiving and social support experienced inside and outside the home.

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THE INTERACTION OF POVERTY, CHILD-REARING, AND BRAIN DEVELOPMENT

What goes on in homes, child care and community settings and their neighborhood contexts affects brain development of infants and young children and their later school readiness and performance as well as their mental and physical health (Boyce et al. 2012, Farah et al. 2008). Pierre Bourdieu underlined for sociologists the importance of the "total, early, imperceptible learning, performed within the family from the earliest days of life" (1984, p. 477). Indeed, the character of interactions between caregivers and very young children provides more or less stimulation to cognitive and language development at formative stages (Farah et al. 2008) and influences the emergence of self-regulation (Bernier et al. 2010, Sarsour et al. 2011). Beginning in infancy, so-called serve-and-return interactions with adults build the architecture of the brain; in these interactions, adults respond in kind to touching, babbling, and facial expressions and continue to interact by talking to, touching, and holding young children (Harvard Cent. Dev. Child 2013). However, the extent of serve-and-return interaction varies markedly across caregivers, with significant consequences for developmental outcomes. Similar variations occur in the degree of positive and nurturing relationships with adults; these relationships buffer children against the harmful effects of stressful experiences so that they become tolerable rather than toxic and thus minimize allostatic load.

Findings from the National Institute of Child Health and Human Development Early Child Care Research Network (NICHD) show that a significant proportion of the explanatory power of poverty for children's cognitive skills was mediated by indicators of maternal warmth and sensitivity as well as by measures of home enrichment resources (NICHD 2005). Access to stimulating and enriching educational toys, books, games, and activities has long been seen as important to children's early cognitive growth and later success in school (Bradley & Corwyn 2002, Chen et al. 2011, Miller & Chen 2010). Cross-national research in the United Kingdom and United States, however, also underlines the central importance of maternal warmth in helping to account for family income differences in children's cognitive performance (Waldfogel & Washbrook 2011). Other studies have found that the extent of parental negativity (e.g., hostility, criticism, annoyance, anger, physical punishment) toward children helps to mediate the inverse relationship between family SES and children's health. For example, Belsky et al. (2007) found that parents' ratings of the health of their six-year-olds was higher when the children experienced positive control and warmth but lower when they experienced parental negativity.

Self-regulation mediates the relationships between these dimensions of parenting and the later cognitive performance and mental and physical health of children. More intrusive and preemptive parenting styles give less room for the development of self-regulation than do approaches that

provide positive support for children's autonomous efforts at self-regulation (Berger 2011). Other research reports that self-regulatory capacity at age 8 to 9 was predicted by lower levels of physical punitive discipline and greater maternal warmth at age 4 to 5 (Colman et al. 2006). Chaos in the home appears to relate to inconsistent and harsh discipline and to hamper the development of self-regulatory skills (Dumas et al. 2005). Thus, although the development of self-regulation is not fully understood, it appears to depend importantly on parenting styles, which in turn relate to the social contexts of households.

The nature of parenting and parenting context also affects language development, which matters both in aspects of brain development and in later academic performance. Social class differences in language development are well documented (Durham et al. 2007, Farkas & Beron 2004). Hart & Risley (1995) observed large disparities in the number of words that children heard from their parents—more than three times as many in professional families as in lower-income families. As a result, these researchers found that three-year-olds from low-income families had less than half the vocabulary of their counterparts from professional families, with middle- and working-class children falling in between. Another study found that in crowded homes parents respond and speak less to their children (Evans et al. 1999). Recent research showed that lower-SES children at 24 months are 6 months behind their higher-SES peers in vocabulary and language processing speed and accuracy (Fernald et al. 2013). Evidence from other research suggests that such language development gaps will persist and predict later academic success (Farkas & Beron 2004, Fernald et al. 2013).

Because the brain is plastic, school- or preschool-based interventions can help make up differences between young children experiencing substantial adversity and those who are not. The HighScope Perry Preschool Program, Carolina Abecedarian Project, and Chicago Child-Parent Centers provided intensive preschooling of varying sorts and lengths for at-risk children who were studied at least into young adulthood, as were comparison groups (Temple & Reynolds 2007). Results include substantially reduced special education rates, fewer retentions in grade, and higher school attainment (Temple & Reynolds 2007). The Infant Health and Development Program provides high-quality day care with a language-based curriculum for low-birth-weight infants of all income levels. Duncan & Sojourner (2013) concluded that the 2-year program (for ages 1–3) had much more impact on low-income children than on high-income children, closed income-based gaps in cognitive performance and school readiness at age 3, and substantially reduced gaps at ages 5 and 8. An experimental evaluation of a classroom-based approach termed Tools of the Mind in kindergartens showed improvements in executive function and reasoning as well as reduced stress, especially in high-poverty schools (Blair & Raver 2014). Research about these varied programs and others thus indicates the positive impact of well-designed and well-implemented school and preschool interventions on at-risk children who may have been subject to toxic stress.

VARIATIONS IN PARENTING, SOCIAL CONTEXTS, AND RESOURCES

The likelihood of sensitive, warm, nonpunitive maternal child care and secure attachments to caregivers varies inversely with the amount of stress that parents undergo (Meaney 2010). Thus, in more stressful environments for parents, children not only experience less protection from environmental stressors but also are more likely to have stress-inducing relationships with caregivers (Blair & Raver 2012, Meaney 2010). We know that parents in households experiencing chronic poverty are especially vulnerable to high levels of stress (Linver et al. 2002). Using data from the Child Development Supplement, a component of the Panel Study of Income Dynamics, Yuan (2009) reported that decreased parental well-being and heightened stress resulting from economic hardship explain most of the rise in children's negative emotions and behavioral problems.

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Parental life course experiences, social and cultural capital, and material resources also shape parents' expectations of and interactions with children (Lareau 2011). Extended work hours, multiple jobs, changing work schedules, long commuting time, single parenting, social isolation, and overcrowded and chaotic homes diminish the amount of time that parents can spend with children and shade the character of their interactions, with psychological distress, anxiety, frustration, and exhaustion produced by circumstances (e.g., Ackerman & Brown 2010, McLoyd 1990, Repetti & Wang 2010). The Fragile Families & Child Wellbeing Study points to high levels of household instability and complexity as key factors shaping the experience of many never-married single parents and producing poor outcomes for children (McLanahan 2009). Neighborhoods also constrain parenting practices (Sampson et al. 2008) and can induce toxic stress. Parental overload can contribute to depression and other mental health issues, which in turn affect parenting resources, style, and behavior (Kiernan & Huerta 2008). Varying approaches to parenting are also likely to be significantly influenced by parental experience of toxic stress as a result of their own childhood adversities (Kim & Evans 2011, McLoyd 1998, Miller et al. 2011, Repetti et al. 2011).

In an influential, qualitative study of 12 families, Lareau (2011) distinguished between middle-class parenting directed at "concerted cultivation" of children and adolescents and parenting more common among poor and lower/working-class households that relies on "natural growth." According to Lareau, concerted cultivation involves "an emphasis on children's structured activities, language development and reasoning in the home, and active intervention in schooling" (2011, p. 32). Natural growth, in contrast, leaves children largely on their own and relies on parental directives rather than on negotiation. This characterization of class-correlated parenting approaches stems from research about school-age children and their parents.

Considerable evidence suggests that elements of the patterns that Lareau has identified extend back into parenting of infants and toddlers. For example, McLoyd (1990) described the higher likelihood that parenting by low-income parents will be inconsistent, punitive, and coercive. Another study using data from the children of the 1986 National Longitudinal Survey of Youth reports that poor mothers showed less responsive and harsher parenting than nonpoor mothers (McLeod & Shanahan 1993; see also Kelly et al. 2011). NICHD researchers found that sustained poverty and low maternal education predicted low maternal sensitivity, which in turn correlated with parent-rated behavioral problems and weaker cognitive development of children (NICHD 2005). Other research indicates that income relates directly to the frequency with which parents read to young children, and time use studies show large income-related differences in the amount of time that parents spend together with their children in novel environments but smaller differences in parental conversation time with children (Phillips 2011). Augustine (2014) concluded, using NICHD data, that less-educated single mothers do not generally have the support to "make the time investments, or engage in the kind of sensitive parenting, that help children experience early success in school" (p. 712). Consistent with Lareau's (2011) findings, Augustine's study thus indicates that lower-class and working-class parents tend to be far less engaged with their infants and toddlers than are middle-class parents, with significant potential consequences for early brain development. Further research by sociologists is needed to solidify our understanding of class-based patterns of early child-rearing and to identify sources of within-class variations.

Because social circumstances influence caregiving approaches, it is not surprising that caregivers' child-rearing practices can change in response to improved social and material resources as well as to enhanced knowledge about child development and informed and constructive feedback about approaches to interacting with infants and children (Blair & Raver 2012). We know least about how changing the economic circumstances of families alters approaches to parenting, but

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research does indicate that the quality of the home environment improves as families move out of poverty and childhood psychopathology can decrease (Costello et al. 2003, Garrett et al. 1994).

Interventions targeted at individual families in the United States have been more numerous and better studied. Such interventions also suggest the potential for changes in approaches to caregiving that parents understand are related to their children's life chances. The Baby College of the Harlem Children's Zone provides a powerful case study (Tough 2008). Another example comes from research demonstrating that the level of verbal interaction between caregivers and children can shift upward with some training and feedback from LENA recorders that monitor the frequency of speech interactions and the number of words used (Suskind et al. 2013). General reviews of a variety of parenting programs to increase parental sensitivity to children and enhance communication, learning, and cognitive development show some positive and some equivocal results in the context of very uneven research designs (Huberman & Mendelsohn 2012, Sweet & Appelbaum 2004, Welsh et al. 2014).

HEALTH, STRESS, AND SCHOOL SUCCESS

Health disparities across social classes have long been documented (Braveman et al. 2011, Phelan et al. 2010), and these differences extend to children. Clear links have been established between poverty and other adversity in childhood and adult ill health (Danese & McEwen 2012). But poverty also makes childhood illness more probable. For example, Bauman and colleagues (2006) demonstrate steep increases in parent-reported child ill health as the number of social disadvantages—poverty, single parenthood, less-than-college education—increases (see also Belsky et al. 2007, Flaherty et al. 2006). Poverty increases the risk of developmental delays, obesity, and asthma among children and makes chronic inflammation more likely (Pascoe et al. 2016). Adopting an "ecobiodevelopmental framework" for understanding the effects of poverty on children's health and well-being, the American Academy of Pediatrics in 2016 published a policy statement committing it to work toward eliminating child poverty and encouraging pediatricians to recognize in their practices the importance of poverty as one of the key social determinants of health (Counc. Community Pediatr. 2016).

Scholars have increasingly recognized that health disparities not only result from but also contribute to social and economic inequalities (Palloni et al. 2009). Health problems, whatever their origin, affect school readiness and longer-term educational performance (Crump et al. 2013, Currie 2009, Porche et al. 2016). Using data from a British cohort study following a sample from birth through middle age, Jackson (2010) reported not only the usual links between parental social status and child health, but also that early-childhood health has a long-term effect on educational and occupational achievement. The same is true for mental health issues, which have long been tied to stress (George 2013, Wheaton et al. 2013) and which commonly appear to have their origins in childhood (Kessler & Wang 2008, Porche et al. 2016). Behavioral issues in schools may be tied in part to brain development that does not support strong executive function and self-regulation. Research documents the substantially lower likelihood of high school completion among adolescents who have experienced mental health problems (McLeod & Fettes 2007). Thus, an understanding of social class differentials in school readiness and cognitive performance needs to incorporate the impact of toxic stress on physical and mental health early in life.

INDIVIDUAL VARIATION IN RESPONSE TO ENVIRONMENTS AND TO STRESS

The toxic stress model not only underlines the patterned ways in which social structures, resources, and circumstances affect brain and body development in early childhood, but it also acknowledges

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that individual variations depend on genetic factors in interaction with varying social environments. Differing genetic characteristics, for example, lead to differing relationships between the experience of childhood abuse and later depression as an adult (Caspi et al. 2003). Other research demonstrates that links between problematic behaviors and temperament are importantly influenced by genetic factors (Shiner et al. 2012). We also know that resilience in the face of adversity has genetic components that help shape a child's temperament, which in turn affects caregiver responses (Kim-Cohen et al. 2004). Freese (2008) underlined the importance of social processes that accentuate the impact of genetic traits on behavioral outcomes and life course trajectories.

The developing theory of biological sensitivity to context identifies reactive alleles: variants of genes that increase the responsivity of the individual to both adverse and positive experiences (Ellis & Boyce 2011, Obradović et al. 2010). This work proposes that environmentally sensitive infants, children, and adults respond disproportionately well to positive, supportive environments but disproportionately negatively to adversity. More biologically sensitive children in comparison to their not-sensitive peers respond more negatively to family conflicts, parental depression, and mental illness and more positively to demonstrations of parental warmth as well as to interventions to increase parental use of sensitive discipline (Bakermans-Kranenburg et al. 2008, Boyce et al. 2012).

Social environments help shape biology, not only in terms of direct effects on the HPA axis and limbic system, but also through epigenetic changes (Landecker & Panofsky 2013). Gene expression can be altered by environmental conditions through varied biochemical and molecular processes that do not alter the DNA sequence (Meaney 2010). Environmental signals and conditions, for example, modify the ways in which proteins act on the DNA double helix, bind to it, and affect the information that RNA messenger molecules take from the DNA without changing the genetic code (Mehler 2008, Szyf et al. 2008). These chemical processes that switch on and off various parts of the genome are characterized as epigenetic. For example, striking research results from a study using an animal model highlight the importance of early care by a caregiver in affecting gene expression (Meaney 2010). Those rat pups experiencing high licking and grooming during lactation controlled stress much better as adults than did other pups. These characteristics are then heritable but in the first instance are caused by an epigenetic modification that is induced by intense maternal care.

Evidence from humans suggests parallel phenomena connected to early-life adversities related to poverty, including abuse and neglect. These experiences in humans alter the expression of genes that respond to stress signals affecting heart rate and blood pressure. The result is a lifetime of increased sensitivity to stress that heightens the likelihood of inflammation and disease (Miller et al. 2009; see also Lam et al. 2012, Tehranifar et al. 2013). Other research shows higher rates of DNA methylation[7]—which alters gene expression—in adolescents exposed to high levels of early-life stress (Essex et al. 2013). The emerging understanding of epigenetics expands awareness of the highly consequential impact of social and physical environments on biological processes in individuals and their offspring (Grossniklaus et al. 2013). Thus, we end the explication of the model where we began—by underlining the vital biological impact of social structures, processes, and relationships on developing bodies and brains.

[7]Methylation of DNA is an epigenetic mechanism by which environmental events such as stressors alter the expression of genetic information without changing the actual genetic code. In methylation, a methyl group (a single carbon atom plus three hydrogen atoms) is covalently attached to a cytosine base in DNA. Other mechanisms of epigenetics also alter gene expression (see Allis et al. 2015).

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SUMMARY AND DISCUSSION

Early-childhood adversities play a significant role in the complex life course processes leading to intergenerational poverty. The model we have built from research in several disciplines highlights the powerful influence that social circumstances have on creating adversities and affecting early-child development and life trajectories through the impact of toxic stress on brain development, cognitive performance, and self-regulation. The research evidence strongly suggests that cumulative toxic stressors in the social environment—for example, conditions created by household and neighborhood poverty—during early childhood affect development of the brain's frontal lobes and limbic system. The effects of these adversities and stressors are ameliorated by strong, supportive adult relationships in a well-ordered social environment. In the absence of strong protective factors, however, the workings of the HPA axis together with other interacting brain and physiological systems that normally produce adaptation often become dysregulated in response to toxic stress. The toll that toxic stress takes appears to be felt most heavily in key processes of self-regulation that influence how effectively people use their cognitive skills to engage with their environments. With weak self-regulation and poor executive function, young children experience increased difficulty in paying attention, organizing and sequencing tasks, resisting impulses and immediate gratification, controlling anger and aggression, and engaging in proactive planning.

This toxic stress model highlights social and biological mechanisms that translate broader social structures into patterned life trajectories. At the same time, it makes room for individual variation. As a result, the model should be a rich resource for stimulating hypothesis building at various levels of sociological analysis and for opening up new areas of future research. For example:

- Cross-societal differences in levels of toxic stress and in school readiness among young children relate to varying degrees of public investment in child care, family leave, and income supports;
- strong immigrant networks and family systems protect against toxic stress from poverty and relate to group variations in rates of children's cognitive performance, self-regulation capacity, and school readiness; and
- individual variations in cognitive performance among children from impoverished families relate to differences in caregiver-child relationships and interactions, which in turn relate to varying levels of caregiver stress and social support.

The broad range of hypotheses suggested by the model indicates future directions of work for sociologists both in their own research traditions and in collaboration with those engaged in the developmental study of childhood adversity.

Clearly, sociologists have much to offer the developmental research enterprise, as the editors of *Nature* (2012, p. 143) recently pointed out: "Sociologists have been studying human environments for decades, and have tallied the social damage that stresses such as poverty or child abuse can cause. Biologists are now in a position to benefit from their insights, although they will need to learn the language of sociology."

And of course, the reciprocal is true: Sociologists have much to gain from learning some of the language and research of biology, developmental psychology, and neuroscience. By doing so, sociologists will not lose their focus on social structures and processes but will instead find their understanding of the mechanisms through which they work significantly enhanced.

Although the model we propose focuses sociologists' attention on early childhood, an underexplored topic in the discipline, a larger challenge for sociologists is to transform the model into one encompassing the life course and the increasing interactions of young children with peers and with institutions such as schools and employers as children age (Alexander et al. 2014). Emerging infant and child behavior traits influence the ways in which parents, siblings, peers, and teachers

respond to a child, and those responses in turn have consequences for their expectations of the child. These extraordinarily complex interactions unfold over the life course. Making the situation even more complicated are the ways in which parental life courses affect parenting and the life trajectories of the children involved (Evans et al. 2012).

This model and our cursory survey of voluminous research literatures across disciplines suggest to us four important areas for systematic sociological research. First, sociologists can play a lead role in sharpening understanding of the adversities that produce toxic stress as they relate to broader social conditions such as poverty, neighborhood, and other variables. Under what circumstances do such conditions produce adversities that create toxic stress? The rather haphazard collection of adversities that has been examined in research thus far needs thoughtful reexamination in the light of sociological knowledge and theory. Analysis needs to go beyond the fact and timing of poverty to the adversities it may or may not create in a context of social networks and community resources (see, e.g., Evans & English 2002). The considerable sociological research on stress and stressors can contribute to this enterprise, and such contributions in turn will challenge sociologists to look beyond mental health outcomes and to incorporate biological indicators of stress into sociological research and theory.

Second, sociologists in collaboration with developmental researchers need to strengthen understanding of the social relationships, networks, resources, and programs that successfully buffer the effects of toxic stressors in early childhood or help repair them later in life. Because of the brain's plasticity, both cognitive capacity and self-regulation can be reshaped in childhood, adolescence, and young adulthood with the right combination of social interventions at the macro and micro levels. Sociologists need to help examine and assess whether, when, and how such interventions—in communities, with families, by schools—make a difference in children's life trajectories.

Third, the vital importance of early-childhood experiences challenges researchers to bring together sociological perspectives on child-rearing dynamics and social class with the perspectives of developmental scholars interested in the impact of adversity. For example, low-income parents may have warm and strong bonds with infants but employ a natural growth approach (Lareau 2011) as the child ages. What social conditions lead to varying combinations of practices at different ages, and what are the implications for child development of such differing practices? And how do social networks (e.g., kin and neighbors) and resources (e.g., day care, preschool, and church groups) impact child rearing practices and child development? Research also needs to tease out the social sources of variability in parenting in early childhood among low-income households and within low-income neighborhoods.

Finally, sociologists can expand the current work comparing child well-being and outcomes across societies in relation to differing social policies and institutional arrangements. (e.g., Bradbury et al. 2015, Ermisch et al. 2012a, Waldfogel & Washbrook 2011). Recent research, for example, shows SES gradients for seven countries along several dimensions, including cognitive and socioemotional measures for children. The United States has the steepest gradients, and Ermisch et al. (2012b, p. 51) speculate that these are "established early on in a child's life." In these gradients within and across nations, the mechanisms at work appear to include differences in parenting, which are conditioned, in part, by resources and social networks (Waldfogel & Washbrook 2011). The toxic stress model can help frame new comparative research by sociologists and developmentalists in national settings with diverse policies toward children and families.

In calling for collaboration and cross-fertilization of research between sociologists and scholars from other disciplines, we—a sociologist and a neuroscientist—recognize many challenges. These come both from attitudes toward and lack of knowledge of other disciplines and from different conceptualizations, disparate research designs, and massive and minimally overlapping research

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literatures. However, there are more similarities than expected in research approaches, for example, secondary analysis of large data sets, small- and large-scale cohort studies, and heavy use of multivariate statistical analyses to sort through correlations among many variables. The challenges can be worked through, especially when we recognize what unites this work: a deep interest in solving a real puzzle of enormous importance for societies and for human lives. Specifically, why is poverty likely to be transmitted across generations, and what social processes and interventions mitigate the extent of that transmission?

We also believe that deepened understanding of both the social and biological mechanisms at work in the replication of poverty will make research evidence more useful in identifying policy and practice options to diminish poverty replication and in lending such evidence greater credibility in debates about policies. In the 2002 American Sociological Association presidential address, Barbara Reskin advocated "pursuing the mechanisms responsible for varying levels of inequality, [so] our scholarship can contribute to ameliorating these disparities" (2003, p. 17). Similarly, neuroscientists Daniel Hackman & Martha Farah (2009, p. 71) have called for enhanced understanding of the mechanisms by which SES affects the capacity of individuals to succeed in school and work because that knowledge "has the potential to reduce poverty and to prevent or ameliorate its burden." By embracing rather than resisting the examination of biological mechanisms that work in interaction with social structures, relationships, and networks, sociologists will strengthen their disciplinary understanding of intergenerational poverty and the relevance of that work to policy discussion.

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