CHILD DEVELOPMENT PERSPECTIVES

Childhood Poverty, Chronic Stress, Self-Regulation, and Coping

Gary W. Evans[1] and Pilyoung Kim[2]

[1]Cornell University and [2]University of Denver

ABSTRACT—***Poverty is a powerful factor that can alter lifetime developmental trajectories in cognitive, socioemotional, and physical health outcomes. Most explanatory work on the underlying psychological processes of how poverty affects development has focused on parental investment and parenting practices, principally responsiveness. Our primary objective in this article was to describe a third, complementary pathway—chronic stress and coping—that may also prove helpful in understanding the developmental impacts of early childhood poverty throughout life. Disadvantaged children are more likely than their wealthier peers to confront a wide array of physical stressors (e.g., substandard housing, chaotic environments) and psychosocial stressors (e.g., family turmoil, separation from adult caregivers). As exposure to stressors accumulates, physiological response systems that are designed to handle relatively infrequent, acute environmental demands are overwhelmed. Chronic cumulative stressors also disrupt the self-regulatory processes that help children cope with external demands.***

KEYWORDS—*poverty; stress; self-regulation; coping*

Gary W. Evans, Departments of Design & Environmental Analysis and of Human Development, Cornell University; Pilyoung Kim, Department of Psychology, University of Denver.

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Correspondence concerning this article should be addressed to Gary W. Evans, Departments of Design & Environmental Analysis and of Human Development, College of Human Ecology, Cornell University, Ithaca, NY 14853-4401; e-mail: gwe1@cornell.edu.

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Today, nearly one in four American children grows up in poverty (U.S. Census, 2011), with worldwide levels much higher. Poverty and other markers of disadvantage are powerful variables that can alter developmental trajectories, including cognitive development (Duncan & Brooks-Gunn, 1997; Heckman, 2006), socioemotional development (Bradley & Corwyn, 2002; Conger & Donnellan, 2007; Grant et al., 2003), and physical health (Chen, Matthews, & Boyce, 2002; Evans, Chen, Miller, & Seeman, 2012; Miller, Chen, & Parker, 2011), throughout life.

In the last two decades, many scholars have investigated the underlying psychological processes that explain why childhood poverty has such pervasive ill effects on human development. Most of this research has focused on two pathways that are complementary to the one we describe in this article. The first pathway to explain why poor children lag behind their more affluent peers is parental investment. One reason poverty is inimical for children is because children in poverty have less cognitively stimulating environments, with less available print media, fewer age-appropriate toys, fewer informal learning venues, fewer educational digital materials, and more exposure to television (Bradley & Corwyn, 2002; Duncan & Brooks-Gunn, 1997; Evans, 2004). Poor children also live in impoverished language environments where fewer words are spoken and parents read to them less often (Hoff, Laursen, & Tardif, 2002).

A second pathway linking poverty to human development is the tendency of lower income parents to engage in harsher and less responsive interactions with their children (Bradley & Corwyn, 2002; Conger & Donnellan, 2007; Grant et al., 2003). Low-income families have more conflict and hostility, and are more likely to rely on corporal punishment than more affluent families. Less responsive parenting in disadvantaged families includes less attention and social support to children's emotional needs as well as less instrumental support, such as helping children with school work or providing information or material assistance (e.g., less help finding part-time work and applying to college, less available money).

A third, but less well-studied pathway that may underlie childhood poverty and development is elevated chronic stress.

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According to the model we summarize, disadvantaged children must contend with a wide array of stressors that strain and eventually damage their biological and psychological regulatory systems. In the rest of this article, we summarize two major components of the chronic stress model of childhood poverty. In the first section, we show that some of the harmful effects of childhood poverty are caused by elevated environmental demands that strain the biological response systems that maintain organisms' equilibrium. In the second section, we show that childhood poverty also damages self-regulatory capacities that help children manage environmental demands.

CHILDHOOD POVERTY AND CHRONIC STRESS

Environment of Childhood Poverty

One reason poverty is stressful for children is because of the environments in which they grow up. Numerous social and physical stressors are correlated with income, including family conflict and turmoil, family dissolution, maternal depression, exposure to violence, as well as elevated parental harshness and diminished parental responsiveness (Bradley & Corwyn, 2002; Conger & Donnellan, 2007; Grant et al., 2003). Poor children are also more likely to live in homes that are more chaotic, with greater structural problems, noise, crowding, toxins, and allergens. Poor children live in neighborhoods that have less social capital; are exposed to more toxins and pollutants, crime, and street traffic; and have fewer places to engage in physical activity and less access to healthy foods (Evans, 2004).

Exposure to multiple stressors may be a signature feature of childhood poverty with far-reaching consequences. Not only are poor children likely to be exposed to higher levels of each of the many individual psychosocial and physical stressors listed previously, they are especially likely to be exposed to a confluence of multiple stressors. This is important because exposure to multiple risk factors outweighs the adverse developmental sequelae of being exposed to a single risk (Evans & Kim, 2010; Sameroff, Seifer, & McDonough, 2004). For example, accumulation of risk exposures throughout childhood mediates the link between social class at birth and middle-age social attainment (Schoon et al., 2002). Similarly, elevated cumulative risk exposure mediates the link between poverty and chronic physiological stress (e.g., resting blood pressure, overnight stress hormones) as well as multiple markers of psychological well being (e.g., symptoms of aggression, anxiety, and depression, self-regulatory ability, learned helplessness) (Evans & English, 2002; Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005).

Not only are low-income children themselves more likely to experience a greater array of physical and psychosocial stressors, their parents and peers are more apt to be confronted by many of the same environmental demands. Poverty-related stress affects parents' competencies as well as interpersonal relationships among family members (Conger & Donnellan, 2007; Grant et al., 2003). Furthermore, many urban poor children live in neighborhoods and attend schools made up mostly of low-income individuals (Brooks-Gunn, Duncan, & Aber, 1997). We know little at this time about the cross-level interactions of child and parent stressors or the potential interactions between stressors in the household and those in the low-income neighborhoods in which poor children live and the schools they attend.

One implication of the ecological context of childhood poverty is that disadvantaged children not only face a greater confluence of cumulative risk factors but do so across multiple domains of risk. Physical and psychosocial environments are more likely to be inadequate across the settings directly experienced by the child (e.g., home, child care, school, and neighborhood). Moreover, these settings are often embedded in relatively more impoverished contexts inhabited by other important adults and peers in the child's life. For example, parents are more likely to work in unhealthy settings that are also more stressful. Thus, disadvantaged children experience more suboptimal environmental conditions and do so in a wider array of developmentally salient contexts.

Chronic Physiological Stress

Another reason to consider chronic stress as an underlying mechanism linking poverty to child development is physiological outcomes. For most chronic diseases, early childhood deprivation predicts morbidity in adulthood, regardless of whether there is later upward social mobility. These outcomes might reflect the idea that deprivation is embedded early in life, permanently scarring individuals or leading to a history of multiple insults that accumulate to do damage (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Evans et al., 2012; Miller et al., 2011). Current research has suggested that both perspectives are possible.

A principal pathway for how poverty influences physical disease is elevated chronic physiological stress. Relative to their more advantaged peers, low-income children have more sympathetic nervous activity (e.g., elevated blood pressure), more elevated hypothalamic pituitary adrenal axis (HPA) activity (e.g., dysregulated cortisol), more dysregulated metabolic activity (e.g., elevated adiposity), and greater inflammation indicative of compromised immune function (e.g., elevated cytokines; Chen et al., 2002; Evans et al., 2012; Miller et al., 2011). These income-related alterations in markers of chronic stress with well-documented physical morbidity outcomes can begin early. For example, as shown in Figure 1, family poverty elevates basal cortisol measures beginning at 7 months through age 4 (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011).

A more useful indicator of chronic stress, allostatic load (McEwen & Gianaros, 2010; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010), reflects chronic wear and tear on the body caused by the mobilization of multiple systems as they respond to changing environmental demands. Allostatic

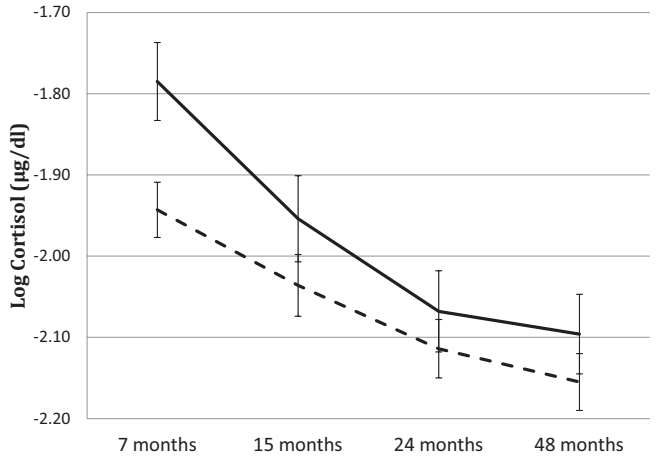

Figure 1. Resting cortisol levels among low- and middle-income children from age 7 months through age 4.
Note. Data adapted from Blair et al. (2011). The solid line denotes low income; the dashed line denotes middle income.

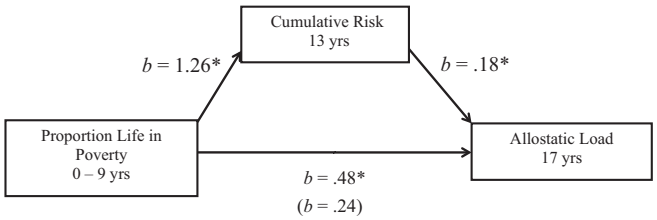

Figure 2. The mediating effects of cumulative risk at age 13 (additive composite of housing quality, crowding, noise, family turmoil, child separation from family, and exposure to violence) between proportion of life in poverty (birth to age 9) and allostatic load at age 17, statistically controlling for allostatic load at age 9.
Note. Data adapted from Evans and Kim (2012).

load, which is typically assessed by indices of cumulative physiological dysregulation across multiple response systems (e.g., elevated HPA, elevated SAM, poor metabolic control, elevated inflammation), is elevated among poor children (Evans & Schamberg, 2009; Goodman, McEwen, Huang, Dolan, & Adler, 2005; Worthman & Panter-Brick, 2008).

Furthermore, as seen in Figure 2, the prospective longitudinal relation between early childhood poverty and elevated allostatic load in young adults is mediated by cumulative risk exposure during adolescence (Evans & Kim, 2012). Similar findings on cumulative risk exposure mediation have been documented across a wide range of individual indices of physiological stress (Evans & Kim, 2010). Thus, not only does childhood poverty increase chronic physiological stress, some of these effects may be accounted for by elevated cumulative risk exposure among disadvantaged children.

Experiencing deprivation in early childhood may also influence the expression of genes involved in responses to chronic stress. In one study (Miller et al., 2011), genes controlled by the glucocorticoid receptor were less active among those who grew up in low-income households. Because cortisol levels were equivalent across groups, the study concluded that this was evidence of glucocorticoid insensitivity, which occurs when cells do not fully register cortisol signals through the glucocorticoid receptor, yielding a more reactive physiological stress response. In addition, the severity of asthma is related to the overexpression of genes that regulate inflammatory processes and to stress responses among children living in poor families, but not among those living in affluent families.

In addition to poverty-related stress directly altering genetic phenotypes, genes can also moderate children's vulnerability to the chronic physiological stress that goes along with disadvantage. Chronic stressors accompanying poverty, such as parental insensitivity, chaos, family instability, and financial pressure, each interacting with specific genetic polymorphisms to alter vulnerability (Kim & Evans, 2011). As an illustration, in one study, allostatic load for low-income African American adolescents was significantly elevated only among the subset of young adults with the short allele of a serotonin transporter gene (5-HTTLPR; Brody et al., in press). This variant of the gene is associated with hypervigilance and high reactivity to environmental events, including chronic stress. Low-income youth with the long allele did not manifest elevated allostatic load. In summary, low-income children experience elevated chronic physiological stress that's caused, in part, by the higher levels of cumulative risk factors they encounter.

CHILDHOOD POVERTY, SELF-REGULATION, AND COPING

Childhood poverty not only increases stress levels but interferes with regulatory systems that enable children to manage the many environmental demands typically accompanying poverty. Self-regulation and coping rely on multiple processes—attention control, working memory, inhibitory control, delay of gratification, and planning—that can be directly compromised by chronic stress (Blair, 2010; Blair & Raver, 2012).

Childhood Poverty and Self-Regulation

Low-income children have multiple self-regulatory deficits. Their parents and teachers rate them as less competent in self-control, they have more trouble delaying gratification, they manifest attentional-control problems, they exhibit weaker inhibitory control, and they have diminished capacity for working memory (Blair, 2010; Blair & Raver, 2012; Evans et al., 2005; Hackman, Farah, & Meaney, 2010). Moreover, some of the adverse impacts of poverty on children's self-regulatory skills are moderated by self-regulatory skills: Low-income children with better self-regulatory skills are more resilient to adverse psychological outcomes (Blair, 2010; Blair & Raver, 2012).

Childhood Poverty and Coping Strategies

Childhood poverty is also associated with maladaptive coping strategies among older children and youth. Coping strategies that engage environmental demands tend to be adaptive. For example, active engagement by solving problems or seeking support protects children from the adverse effects of stressors on mental health (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). In contrast, efforts to disengage, such as avoidance or withdrawal, are associated with elevated internalizing and externalizing symptoms in children exposed to various stressors (Compas et al., 2001).

Low-income adolescents are more likely to use disengagement coping strategies, which has been found to elevate internalizing, externalizing, and social difficulties among low-income children and adolescents (Wolff, Wadsworth, & Santiago, 2010). However, low-income children who rely primarily on engagement coping strategies, such as problem solving or cognitive reappraisal in response to poverty-related stress, exhibit fewer adverse psychological symptoms (Wolff et al., 2010). Reframing the meaning of stressors as less threatening and holding more optimistic/hopeful beliefs buffer the adverse impacts of low income on children's inflammatory levels and degree of asthma (Chen & Miller, 2012). This combination of shift and persist coping strategy also tempers the relation between early childhood deprivation and elevated allostatic load among middle-aged adults (Chen & Miller, 2012).

Poverty, Parenting, Self-Regulation, and Coping

The two additional pathways linking childhood poverty to adverse outcomes—parental investment, and parental responsiveness and warmth—are important in thinking about poverty, self-regulation, and coping. Exposure to poverty increases distress among parents, which negatively affects the quality of parent–child interactions (Conger & Donnellan, 2007; Grant et al., 2003). Less responsive and harsher parenting both contribute to deficits in self-regulation (Blair, 2010; Blair & Raver, 2012). Positive parent–child relations, in contrast, can buffer the impacts of childhood poverty on children's well-being. For example, maternal responsiveness was found to attenuate the impacts of childhood deprivation on inhibitory control (Sarsour et al., 2011), the metabolic syndrome, a precursor to diabetes (Miller et al., 2011), and multiple markers of inflammation (Miller et al., 2011). Parental investment may also be related to the development of self-regulatory skills in children. Low-income parents who talk less to their children tend to have children with poorer language skills, which limits children's ability to regulate their emotions because of deficits in emotional expression and communication skills (Hoff et al., 2002).

Poverty, Stress, and the Brain

Lastly, childhood poverty is associated with altered structure and function of brain regions involved in stress and self-regulation. Childhood deprivation has been linked to reduced hippocampal volume, exaggerated amygdala responses to adverse stimuli, altered prefrontal cortical (PFC) activity and structure (including reductions in volume), and disrupted connectivity between the PFC and amygdala. Chronic stress accompanying childhood poverty appears capable of disrupting top-down control of emotional responses while simultaneously heightening amygdala sensitivity to negative emotions such as anger (Hackman et al., 2010; McEwen & Gianaros, 2010).

SUMMARY AND NEXT STEPS

We are beginning to understand some of the reasons why childhood poverty is harmful for human development over the course of life. Prior work has focused on underlying pathways associated with parenting, particularly investments in cognitive enrichment along with parental responsiveness and warmth. Emerging research suggests a third, complementary pathway between childhood poverty and human well-being—elevated chronic stress. Disadvantaged children are more likely to have to deal with multiple, suboptimal physical and psychosocial conditions. The accumulation of multiple stressors brought on by childhood poverty pressures response systems to marshal resources to meet the numerous environmental demands that threaten bodily equilibrium. The elevated chronic stress accompanying childhood disadvantage also disrupts the development of robust self-regulatory and coping capacities necessary for growing children to learn how to manage environmental demands.

Among the gaps in our knowledge of childhood poverty, stress, and self-regulation, we highlight five issues. First, we have little data on developmental trajectories in relation to early childhood poverty. Most poverty stress studies are cross-sectional and the few longitudinal studies do not have data on three or more developmental periods necessary to examine developmental trajectories with growth curve modeling.

Second, how the timing and duration of exposure to poverty in childhood affect development has not been well integrated with any of the three underlying mediating processes (i.e., parental investment, parenting quality, and chronic stress) linking poverty to developmental outcomes. Whether early deprivation becomes embedded in the organism, in essence creating a scar that continues to fester, or whether repeated experiences of poverty over life influence human potential in a cumulative manner is a critical issue. These two perspectives are not mutually exclusive—there could be critical periods for poverty exposure as well as alterations in subsequent developmental trajectories in relation to subsequent experience of disadvantage and/or salutary phenomena (e.g., upward social mobility).

Third, some of poverty's ill effects on children appear to be caused by exposure to multiple stressors. The confluence of risk factors brought on by deprivation may be a unique and key feature of poverty for human development. More research is needed

to examine whether certain types of stressors (e.g., physical, psychosocial, familial, peer) or combinations of stressors across different domains (e.g., household, school, neighborhood) endemic to poverty account for some of the adverse consequences of childhood poverty. Alternatively, perhaps it is the sheer quantity of environmental demands—regardless of type or pattern of stressor exposures—that matters.

Fourth, we need more clarity on the specifics of poverty, chronic stress, and self-regulatory and coping processes. Under what circumstances does poverty alter self-regulatory processes so that they become a direct pathway linking poverty to development? Greater self-regulatory capacity can also buffer some of childhood poverty's ill effects. How and when do self-regulatory processes become sufficiently damaged so they function directly to mediate the ill effects of early poverty on subsequent development?

Finally, low-income parents experience considerable financial strain that adversely affects their parenting abilities. To what extent are the elevated stress and poorer self-regulatory and coping processes documented among disadvantaged children a function of parental stress and related suboptimal parenting practices? A fuller understanding of the effect of poverty and other forms of disadvantage on child development will likely come from a more contextualized perspective that explores underlying cognitive, socioemotional, and physiological processes influenced by the chronic strains faced by low-income children and their families at home, in child care and school, and in their communities.

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