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Poverty, Stress, and Brain Development: New Directions for Prevention and Intervention

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Abstract

We review some of the growing evidence of the costs of poverty to children's neuroendocrine function, early brain development, and cognitive ability. We underscore the importance of addressing the negative consequences of poverty-related adversity early in children's lives, given evidence supporting the plasticity of executive functions and associated physiologic processes in response to early intervention and the importance of higher order cognitive functions for success in school and in life. Finally, we highlight some new directions for prevention and intervention that are rapidly emerging at the intersection of developmental science, pediatrics, child psychology and psychiatry, and public policy.

Keywords

brain development; early childhood; executive function; infancy; parenting; poverty; stress

Scientific attention has focused on the toxic consequences of stress for brain function and mental and physical health. It is has become increasingly clear that one of the mechanisms through which poverty affects the health and well-being of children and adults is through the toxic effects of stress on the brain. A growing body of evidence indicates that effects of poverty on physiologic and neurobiologic development are likely central to poverty-related gaps in academic achievement and the well-documented lifelong effects of poverty on physical and mental health.[1–5]

Here we review studies delineating the substantial effects of poverty on children's biological and psychologic development, thus emphasizing the importance of early experience and the malleability of developmental processes that are shaped early in life to establish a foundation for later competence. We also review studies that demonstrate the efficacy of early intervention for children at risk, highlighting implications for policy.

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Poverty and Brain Development

Although examinations of direct relations between income and brain structure and function are relatively recent, 2 prominent reports demonstrate that effects are particularly large and seen early in development for children in poverty. One study examined a cross-sectional sample of 389 children aged 4 to 22 years and found that children in families in poverty had reduced gray matter volumes in the frontal and temporal cortex and the hippocampus. When families were at 150% of poverty, these reductions were 3% to 4% below developmental norms. For children in families at 100% of poverty or below, reductions in these regions were 8% to 9% below developmental norms. Given the association of these regions with school readiness and school achievement, this analysis further examined the extent to which these gray matter reductions account for the well-known effect of poverty on academic outcomes. Mediation analysis of standardized achievement test data indicated that the measures of gray matter in frontal and temporal regions accounted for between 15% and 20% of the income-related achievement gap.[2] In a similar analysis examining cortical surface area with 1099 children and young adults aged between 3 and 20 years, both parental education and income were found to be positively related to surface area. Associations were greatest in the frontal, temporal, and parietal regions.[6] As with the analysis of Hair et al,[2] this study also found that the effects of income on brain development are largest for children in families whose incomes fell below the poverty line.

Effects of poverty on brain development start early and are seen in infancy. In a longitudinal analysis of 77 children participating in the National Institutes of Health (NIH) MRI Study of Normal Brain Development and seen between the early postnatal period and age 4 years, those in low-income or poor families were found to have total gray matter volumes that were nearly half a standard deviation smaller than their better-off counterparts. These reductions were particularly large in the frontal and parietal regions associated with executive function abilities.[7] Growth modeling indicated that these associations are developmental, with reduced growth trajectories for total, frontal, and parietal gray matter volumes that were most pronounced for children in poverty. These results are consistent with an EEG study of 6- to 9-month-old infants that found reduced high-frequency electrical oscillations in the frontal cortex, the seat of executive function abilities, among children in poverty relative to their higher-income counterparts.[8] A second analysis from the NIH MRI study of 283 children aged 11 years found that parental education as an indicator of socioeconomic status was positively associated with regional gray matter in the left superior frontal gyrus and right anterior cingulate gyrus, both regions associated with executive function abilities.[4] Similar longitudinal findings were seen in an analysis of 145 children followed longitudinally from preschool and who underwent MRI when they were approximately 10 years old. In this analysis, household income-to-need was positively related to gray and white matter volumes; the quality of parenting that children received in early childhood and the number of stressful life events experienced were found to mediate some of the effects of income on the volume of the hippocampus.[9]

In combination, available evidence confirms that the shaping of children’s biology and behavior by experience starts early and happens rapidly. The burgeoning research evidence of the costs of poverty to children’s early development and the parallel evidence of the

benefits of early intervention have triggered a call to action on the part of many to "preserv[e] and support our society's most important legacy, the developing brain."[10] Here we outline some developmental foundations that underlie the effects of poverty on brain development and consequences for early learning. We underscore the importance of addressing the negative consequences of poverty-related adversity early in children's lives. In doing so, we also emphasize the need for an increased scientific focus on the malleability and plasticity inherent in development, particularly given the relatively slow time course of brain development in areas that underlie the higher-order self-regulation associated with executive function. Finally, we highlight some new directions for prevention and intervention that are rapidly emerging at the intersection of developmental science, pediatrics, child psychology and psychiatry, and public policy.

Adverse Effects of Poverty on Developing Brain

Traditionally research on child development in the context of poverty has focused on reduced stimulation and reduced opportunity for learning relative to children in higher-income homes. Increasingly, however, research in a variety of disciplines is converging on the idea that in addition to reduced opportunity for types of stimulation that positively affect development, such as a rich and varied language environment,[11] poverty is also characterized by an overabundance of types of stimulation that negatively affect development. Key mechanisms that link children's exposure to poverty-related adversity and brain development include the presence of chronic stressors such as noise, including background noise such as that associated with ongoing and unmonitored television, household chaos, and conflict among family members that alter the physiologic response to stress, leading to potentially teratogenic effects of stress-related hormones on the developing brain and to a range of negative cognitive, emotional, and behavioral sequelae.[12,13] Importantly, poverty-related stressors have been theoretically argued and empirically shown to tune or program the physiologic response to stress in ways that alter neuroendocrine activity and consequently neural activity, thereby influencing the course of brain development and function[14] (Text Box 1). Controlled experiments in rodents and to some extent nonhuman primates demonstrate that exposure to chronic stressors and the resulting corticosterone/cortisol increase from the prenatal period through adulthood is associated with alterations to the volume of the amygdala, atrophy of the hippocampus, and atrophy of pyramidal dendrites, neurons that are integral to prefrontal cortex function and communication between prefrontal cortex and numerous regions throughout the brain, including limbic structures that activate and terminate the stress response.[15–18] Further, patterns of neural activity in the brain are altered under conditions of stress, suggesting that experience-dependent neural and behavioral responses to stimulation will be progressively established over time, biasing the developing individual to be reactive and defensive, rather than to engage in reflective and approach-oriented responses to stimulation.[19,20]

A number of studies have shown that cortisol and other stress markers are elevated in children in poverty.[21–23] In addition, these studies have shown that effects of poverty on the stress response in part underlie the effects of poverty on the development of executive function and the regulation of emotion and attention. These effects are consistent with animal models demonstrating that glucocorticoids influence activity in, and thereby the

development of, brain structures and neural circuitry that are important not only for regulating the hypothalamic–pituitary–adrenocortical (HPA) response to stress but also for executive function abilities.[16] Executive function is essential for self-regulation and school readiness and is a basic building block of early cognitive and social competence. Available evidence indicates that effects of socioeconomic and early psychosocial disadvantage on cortisol and brain structure partially mediate effects of poverty on the development of executive function in childhood.[24–26] Effects of poverty on brain development and executive function are likely one key pathway, along with reduced stimulation for learning, through which poverty is associated with gaps in school readiness and achievement and positive life outcomes. These effects are consistent with, albeit perhaps less severe than, those seen in studies examining effects of extreme stress and trauma, such as that associated with institutional rearing. Findings from studies of traumatic early rearing experience indicate alterations to the volume of the amygdala and hippocampus and total gray and white matter volumes in brain areas that underlie executive function and emotion regulation capabilities.[27,28]

Caregiving as a Key Mechanism

The foregoing provides an initial neurobiologic model detailing the ways in which early experience affects the development of stress-response physiology and the brain in areas that underlie the development of executive function and the self-regulation of behavior. In supportive and resource-rich environments, stress-response systems are understood to shape brain development in ways that are conducive to executive function and high levels of self-control. In lower-resource, unpredictable environments, however, stress-response systems are understood to shape the brain in ways that promote highly reactive behavior and poor executive function ability—an adaptation appropriate for an unpredictable and threatening environment (Text Box 2).

A central aspect of this experience early in life is the quality of caregiving the infant receives.[20] Importantly, the presence of toxic aspects of poverty have been argued to have a corrosive effect on the quality of caregiving provided by adults in ways that can exacerbate rather than mitigate effects of stress on children’s brains.[29] Chronic stress in the context of poverty can adversely affect the style of caregiving that parents provide.[30,31] As well, the maternal brain during pregnancy and the postpartum period is highly plastic for both good and for ill: While it is undoubtedly adaptive for new caregivers to experience substantial shifts in brain and neuroendocrine function that allow for greater attunement to infant cues after the birth of a new child, this plasticity also means that caregivers’ brains are open to insult neurologically, physiologically, and psychologically by stress, including the stresses associated with parenting.[32,33] Here, early caregiving is understood to function as a mediator of the effects of stress on development. In rats, creation of poverty-like conditions by restricting the availability of material for nesting increases the likelihood of lower levels of caregiving competence that are in turn associated with problems with stress regulation and adverse cognitive and behavioral outcomes in offspring.[34] Also in rats, an influential epigenetic model of early experience has demonstrated that specific aspects of maternal behavior in what can be considered the typical range alter gene expression in the pup hippocampus, with widespread downstream consequences for social and cognitive

development, including intergenerational transmission of this maternal behavior.[35] This demonstration of epigenesis, albeit in rodents, makes clear that typical variation in context can affect the type of caregiving the infant receives, with meaningful implications for later development.[36]

In a body of emerging research with human caregivers, studies have shown that when families are faced with stressful psychosocial and physical conditions within the home, parents are at greater risk for becoming less sensitive and warm in their patterns of early caregiving. In turn, lower level of maternal sensitivity increases the likelihood that children will demonstrate elevated cortisol levels and lower executive function ability, with commensurate difficulty regulating emotion and behavior.[37] Notably, however, just as early caregiving functions in a mediating role as a conduit for stressors in the environment, it also functions as a moderator, or a buffer of stress.[38] To be sure, many families provide high levels of sensitive and nurturing caregiving despite the struggle to make ends meet. In families where parents are able to maintain positive, responsive styles of caregiving, the presence of the mother is inherently regulating for offspring, serving to dampen the HPA response to stress. This has been shown most clearly in experiments with rodents in which the presence versus absence of the mother can be manipulated and the effects on pups' HPA activity, gene expression, and later development examined. The physical presence of the mother as a buffer on the HPA axis is seen even in the instance of low-quality early care in which neural systems associated with approach behavior and with early learning override the HPA response and potential learned aversions to the primary caregiver.[39]

In both rats and monkeys, however, intermittent stress induced by brief periods of separation from the mother is associated with benefits to the regulation of the stress response. These benefits are seen not only in the activity of the HPA axis but also in terms of the regulation of behavior in response to challenge and in terms of cognitive abilities, including performance on measures of working memory and inhibitory control.[40,41] These data suggest the potential benefits of moderate stress and emphasize the importance of stress regulation as a central aspect of the influence of experience on development.

Opportunities for Repair

Given the inherently moderating or buffering effect of the primary caregiver on the HPA axis, efforts to ensure consistent high-quality care are particularly important for children in poverty. Extant research indicates that care-giving can serve as a key lever of change through which effects of disadvantageous experience on biology and behavior can be altered. The limits on malleability and the extent to which the developing child may be more or less sensitive to the effects of caregiving and experience at specific points in development—so-called sensitive periods—are not yet fully known (although parameters are being discerned from the study of infants reared in conditions of severe deprivation[42]). Acquiring this information about the process of development constitutes a major scientific goal. To date, however, a focus on early caregiving provides partial explanation for why we find resilient outcomes or high levels of competence among a significant proportion of children experiencing exposure to economic stressors and high levels of adversity. This recurrent finding of resilience in the face of high levels of stressors is seminal: It alerts our fields of

developmental science, neuroscience, pediatrics, and child psychology and psychiatry that it is time move past the broad-brush recognition of the negative sequelae of poverty-related adversity for brain development to a more complex model of biological, interpersonal, and sociologic mechanisms that lead to individual differences among infants and children exposed to adversity. In so doing, we will gain new insights to the ways that children's emotional and cognitive regulation can be supported in conditions of hardship and toxic stress.

Research examining optimal ways to support and ensure competent, contingent, responsive caregiving for children in poverty is paramount. Positive parenting behavior represents a tremendously valuable resource within communities affected by poverty. We would be well served to learn from parents and community leaders about ways to preserve, strengthen, and extend this care as a way to safeguard infant and child development in communities hard hit by economic disadvantage; several models of public health messaging to reinforce positive parenting practices have been recently launched to support parents' positive practices as an important, population-level effort to support early "brain health"[43] (eg, the Best Start Initiative in California, and the Talk to Your Baby, Their Brain Depends on It campaign in New York). Caregivers' own mental health and management of stress becomes a viable target for community and public health support.

Pediatric primary care settings have proven to be an excellent venue for initial screening of parent and child mental health risk and for service provision. Screening of infants and toddlers as well as parents for mental health risk and referral to services leads to improvements in mental health and parenting behavior.[44,45] The Reach Out and Read program, which provides parents with children's books along with guidance and modeling of reading interactions during the pediatric primary care visit, has demonstrated effectiveness on child language development and parent–child interaction.[46] An innovative expansion of this program using video-based feedback to enhance parent–child interaction has been shown to increase parent stimulation for development and to reduce maternal depression.[47]

In addition, several targeted parenting interventions delivered through social service agencies supporting children in the context of disadvantage are currently underway.[48,49] Few completed studies, however, have incorporated measures of child physiology or neuroimaging. Several have indicated in high-risk samples of children that programs that increased the quality of care that children received from parents were associated with beneficial alterations to diurnal activity of the HPA axis, HPA axis reactivity to challenge, or both.[50,51] Further, evaluation of a program focused on selective attention for preschool children in poverty demonstrated effects at the neural and behavioral levels from the combination of information for parents with attention training activities for children.[52] In our own ongoing research, home visitors use a program incorporating video feedback to alert and focus parents' attention to their own emotions, behaviors, and appraisals of their infants in the context of ongoing, everyday parenting tasks. Prior evaluations of this program, the Play and Learning Strategies (PALS) program, indicate that it is effective at increasing parenting stimulation and sensitivity with concomitant changes in child development indicators for families in poverty.[53] We are midway through our empirical test

of this model using a randomized controlled trial design examining physiologic and behavioral measures of parent and child outcomes.

This increased integration of service delivery (such as child-focused educational supports combined with services targeting parenting) is not new. For example, early education programs serving young children (such as Early Head Start) have long recognized the importance of involving and supporting parents to attain goals of economic self-sufficiency as well as maintaining and strengthening positive caregiving skills. Increased awareness of the costs of poverty to infant and toddler brain development has arguably sparked greater policy interest in ways that various platforms of service delivery can be more effectively integrated. For example, a new set of "2 Generation" programs that primarily target adult postsecondary education and workforce development have been reengineered to also support parent–child relationships and early child health and development (eg, the Ascend Initiative led by the Aspen Institute). Family-serving community-based agencies and policy partners are often at the forefront of the design and implementation of innovative models of integrated service delivery, where child and parent health, educational attainment, and economic and housing security are approached in holistic and comprehensive ways (eg, http://www.liveworkthrive.org/).

In addition, we note that single-platform interventions involving one policy area (eg, center-based early education and universal prekindergarten) can significantly reduce poverty's negative sequelae when delivered with levels of high quality. Classic early intervention studies of the latter half of the 20th century indicated initial effects on general mental ability with long-term effects on academic achievement and a number of indicators of health and well-being.[54] While neuroscientific methods have been of great value in highlighting the brain-based benefits of reading interventions with older children, we are aware of only one early education study that has directly examined treatment-related effects of preschool intervention targeting self-regulation on brain development using neuroimaging methods.[52] There are, however, relevant examples from reading and language interventions with older children.[55,56] Even without this neuroimaging evidence, the extant findings from behavioral studies are impressive and persuasive with regard to the benefits of high-quality preschool education. For example, several studies have demonstrated that high-quality early education is associated with significant gains on measures of neurocognitive ability, such as executive function, greater skill in perspective taking (or theory of mind), the ability to regulate attention in response to emotional stimuli, and speed of information processing.[57–61] Further, one evaluation of a high-quality kindergarten program found that children in treatment classrooms in high-poverty schools had moderately elevated levels of cortisol indicative of positive stress relative to their counterparts in control classrooms.[58] Finally, long-term follow-up of the Abecedarian early intervention program, which began in the 1970s, found that as adults, participants randomly assigned to the intervention group had greatly reduced prevalence of indicators of risk for stress-related disease.[62] Even though resilient functioning may come with some biological wear and tear for low-income children, greater self-regulation and cognitive control (or executive function) are robust predictors of greater health, greater wealth, lower substance use, and lower involvement in crime in adulthood[63]; investments in early classroom-based interventions that support executive

function and related self-regulation skills may pay major dividends across the life course for decades to come.

Conclusion

In supporting children's physiologic reactivity, cognitive control, and self-regulation through parenting- and classroom-based interventions, prevention scientists, policy makers, and practitioners are essentially working hard to alleviate the costs of poverty for human development. Yet it is equally imperative to work further upstream—to lower parents' and children's exposure to poverty and associated stressors in the first place. Two avenues of policy innovation include supporting families in building higher levels of human capital so as to increase earnings and increasing federal and state income and non-income transfers (such as the Earned Income Tax Credit or Section 8 housing subsidies) to families so that they are less likely to be poor. Legislation introduced into the US Senate and the House of Representatives in summer 2015 for America's College Promise Act would accomplish both of those things at once: It would potentially provide a federal match of $3 for every $1 invested by states to dramatically lower the cost of tuition for community, technical, and tribal colleges for low-income young adults (https://www.whitehouse.gov/sites/default/files/docs/progressreportoncommunitycollege.pdf). In so doing, such policy would not only strengthen the earning potential of college-going young parents but would also lower their debt, leaving them with more income, reduced levels of poverty-related stressors, and more time and energy for their children. Such policy approaches to lifting families out of poverty provides tremendous opportunity for scientists to test ways that reducing poverty may benefit public health through key mechanisms of lowered allostatic load, improved caregiving, and healthier brain development. Reversing the negative consequences of poverty for children's brain development is not just imaginable but is also actionable. Through home-, school-, pediatric medical home-, and community-based intervention, we can and must take those important steps—now.

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Text Box 1

Effects of early stress on development have been demonstrated most clearly in the hypothalamic–pituitary–adrenal (HPA) axis component of the stress response system. The HPA axis is characterized by a cascade of activity in which stimulation of the amygdala initiates the release of corticotropin releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus leading to the secretion of adrenocorticotropic hormone from the pituitary and resulting release of the glucocorticoid hormone cortisol (corticosterone in rodents) from the adrenals. Circulating glucocorticoid then feeds back on the HPA system to inhibit CRH and the resulting production of cortisol.[1] Although increase in circulating glucocorticoids resulting from HPA activity is relatively slow and enduring, on the scale of minutes and hours, CRH also stimulates the sympathetic adrenal system, the faster-acting component of the stress response (on a scale of seconds), leading to complex interactions between the HPA axis and the autonomic nervous system.[2–4] In addition to fast and slow effects of stress on brain function, glucocorticoids affect brain structure through epigenetic processes. Levels of circulating cortisol/corticosterone affect processes in the cell nucleus and influence DNA transcription and gene expression.[5,6] Interactions among the components of stress physiology are intricate, and more research is needed on how they are related to one another developmentally.

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Text Box 2

The adaptability of stress physiology in response to experience is referred to as allostasis or biased homeostasis.[1] Allostasis refers to the adjustment of resting levels of stress response physiology in response to experience. Unlike homeostatic systems that must maintain functioning within a relatively narrow band around a given set point to support the optimal functioning of the organism (eg, body temperature around 98.6°F), stress response systems are allostatic, able to adaptively adjust set points and ranges in response to experience as needed. In this process of allostatic adjustment, the brain plays the key mediating role as it is shaped by experience to adjust physiologic systems to meet an expected future.[2] The process of the interactive adjustment of biological development by experience is referred to as experiential canalization.[3] As applied to self-regulation development,[4] experiential canalization refers to the way in which experience shapes stress response physiology in ways that promote behaviors appropriate for the context in which development is occurring. In the context of poverty, in which resources are scarce and the future unpredictable, stress physiology is hypothesized to shape brain development in ways that promote fast reactive and automatic responses to stimulation. In contrast, in high-resource, supportive environments, experience is hypothesized to shape brain development in ways that promote executive function and the intentional, thoughtful regulation of behavior.

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