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,

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.

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 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.

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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 stressrelated 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 function¹⁴ (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 hypothalamicpituitary-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

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