



Poverty and the Developing Brain: Insights from Neuroimaging

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Abstract

Poverty is a significant social problem, affecting how individuals live and the resources available to them. For children, poverty represents a chronically suboptimal developmental environment as much as it reflects a state of economic stress. Neuroimaging techniques permit the non-invasive investigation of human brain development in adverse conditions such as poverty. Brain imaging has contributed to the understanding of economic disparity by identifying changes in the brain's structure and function associated with poverty. Noninvasive imaging of the developing human brain enables the testing of hypotheses about links between economic disadvantage and neural development. Due to the plasticity of neural pathways, some of the effects of poverty on the brain may be reversible. Empirical research investigating the neural correlates of income disparity will continue to enable the design of targeted interventions to prevent and ameliorate the effects of childhood poverty.

Keywords: poverty, brain, neuroimaging, health, functional magnetic resonance imaging

Introduction

Poverty has cascading effects on neurocognitive development, limiting the availability of educational opportunities and compromising social relationships required for socio-emotional development (1,2). Growing up in a low income household is associated with dietary and nutritional deficiencies, maternal malnutrition, environmental toxins, and insufficient early sensory stimulation (3,4,5). These differences may contribute to the achievement gap observed in academics and cognitive skills in low income and middle class children (6,7). Compared to children from middle-class families, low income children are three times as likely to experience developmental delays. Living in poverty is associated with poorer overall physical health, and low-income youths are also at higher risk of developing mental disorders affecting attention, anxiety, and mood (3,4,5).

Socioeconomic status (SES) represents a combination of economic resources as well as social aspects such as occupational prestige and social status (2,8). SES measurements typically include a combination of education, income, and occupation (9). Low SES families lack access to a wide range of resources to promote and support young children's health and education, as well as resources for social, emotional, and cognitive development.

In recent decades, noninvasive brain imaging tools such as functional magnetic resonance imaging (fMRI) have provided insight into brain systems underlying cognitive functions including language, memory, and attention (10). fMRI investigations have likewise indicated that poverty experienced in childhood may have profound and lasting effects on many of these systems. While much remains to be known about the neurobehavioral ramifications of low socioeconomic status, studies that have addressed

neurocognitive effects of poverty have led to the development of some promising interventions (11,12). Neuroimaging studies testing hypotheses about the relationships between poverty, neurobiology, and neurocognition may continue to have significant economic and sociopolitical implications. Understanding these complex relationships may also affect policy as these studies seem to provide empirical support for the utility of early-life education programs targeted toward low-income children, such as Head Start. The goal of this commentary is to provide a brief overview of advances in cognitive neuroscience that have informed recent interventions seeking to lessen the neuro-behavioral effects of poverty.

Socioeconomic status, stress, and the developing brain

Economic disparity is most harmful to developmental and educational outcomes when experienced early in life, and in conditions of deep poverty (1,7). The stress response is one pathway by which SES status affects neurophysiology and cognitive function of low-income youth (13-20). Stress activates several neuropeptide-secreting systems, primarily the hypothalamic-pituitary-adrenal (HPA) axis' "fight or flight" response. In the short term, the HPA axis mobilizes energy resources to deal with the stressor by producing corticosteroids, such as cortisol (14). Over time, however, chronic stress leads to the overproduction of cortisol, which can lead to hippocampal cell death (15-22).

The term "allostatic load" was coined by McEwen (23) to reflect the harmful cumulative effects of persistent physiological stressors (22). Allostasis, which literally means "stability through change," refers to the ability of the body to adapt to stressful situations. The negative effects of allostatic load include damage to neural feedback loops employed to regulate the production of cortisol. In particular, the hippocampus is vulnerable to the cumulative effects of stress and allostatic load (14-21). The hippocampus subserves memory and cognition and is the target of stress hormones; over time, high levels of glucocorticoids cause overexpression and oversecretion of hippocampal glutamate, which is toxic to surrounding brain cells (22). In addition to causing neuronal atrophy, prolonged stress increases the risk of mood disorders such as depression, anxiety, and post-traumatic stress disorder (22).

SES impacts health outcomes at all phases of life, operating on the individual physiological level (e.g. stress and

allostatic load, lack of exercise) as well as environmental (e.g. exposure to toxins and carcinogens, diet), and socio-cultural (e.g. access to education and health information) levels which not only have a profound impact on not only quality of life, but as researchers have recently examined, brain development and function as well (2). Evans and Schamberg found an inverse relationship between childhood poverty and working memory capacity that was mediated by allostatic load (20); Hanson and colleagues found a correlation between low SES and low hippocampal volume (18).

As the prefrontal cortex and hippocampus are implicated in various cognitive functions including learning, memory, attention, and emotion regulation, it remains unclear exactly how these brain structures relate to the deficits observed in low-SES youth. However, by examining the effects of poverty on specific brain systems, researchers have found that the low-income developing brain exhibits a characteristic pattern of deficits in specific neurocognitive systems that may be candidate targets for behavioral, pharmacological, or neurotechnological interventions.

The neurocognitive effects of poverty

Poverty does not affect all cognitive systems uniformly. In a sample of kindergarteners, Noble, Norman, and Farah (20) examined the effect of SES on five basic cognitive systems: language, executive function, memory, spatial cognition, and visual. Middle-class children performed better than low-income peers on language, memory, and executive function tasks, while no difference was observed between groups when performing tasks testing spatial cognition and vision (20).

The language subsystem, which involves the left perisylvian region as well as other regions of frontal and temporal cortex, is involved in semantic, phonological, and grammatical processing of language (24, 25). SES differences in language processing are observed as early as 18 months and are thought to be partly due to differences in the home linguistic environment (26-28). Perkins and colleagues report that "for each \$5,000 in extra income annually, vocabulary is raised an average of 2 points on a standard scale vocabulary measurement" (28).

The memory system includes medial temporal structures including the hippocampus which are crucial for memory consolidation and retrieval (26). Hermann and Guadagno reviewed several studies indicating that memory

performance is strongly and directly correlated with SES (29), although it is not known whether different types of memory are equally affected by SES disparity (26).

The executive function system, subserved by the prefrontal cortex, enables allocation of attentional resources as well as information retrieval and maintenance (27). The executive function system can be further divided into subsystems including the lateral prefrontal “working memory” system that enables the maintenance and retrieval of information over an interval, and the anterior cingulate “cognitive control” system which is involved when inhibiting irrelevant information to make a task appropriate response (20). A study by Noble, McCandliss, and Farah showed that SES explained 30% of the variance in linguistic abilities and a significant portion of the variance in executive function (20). Dysfunction of the executive system is a hallmark of many psychiatric and mood disorders (30).

Farah and colleagues performed a similar study investigating these cognitive subsystems in older children (31). Sixty children of middle-school age (10-13 years) were tested on each of the five neurocognitive systems. Consistent with the previous study, Farah and colleagues observed significant differences in language, memory, and executive function systems in these children. Within the executive system, working memory and cognitive control deficits were observed. Income disparity was not associated with differences in spatial or visual cognition, or in the processing of rewards.

These studies reveal selective deficits in the language, memory, and executive systems in low-SES children at various ages, suggesting that these particular systems are neurocognitive correlates of SES. In addition to exhibiting deficits in neurocognition, low-SES children develop fewer social ties and experience more stress and deficits in emotion regulation (15, 32). Such developmental differences may affect neurocognitive function into adulthood and could contribute to differences in self-regulation, problem-solving ability, and adult intelligence (6).

Brain development and poverty

Environment plays a critical role in brain development. Children living in poverty have compromised brain development compared to children growing up in non-poor environments, reflecting the suboptimal conditions in which they live (14,33). The developing brain is liter-

ally “shaped” by one’s environment (32). Stimulating environments promote neuronal growth, while stressful environments have negative effects on brain development on multiple levels, from the systems level, e.g., the neuroarchitecture connecting brain regions or the volume of a given brain structure, down to the cellular level, e.g., density of synapses on a single neuron (33). Neuroimaging investigations of the young brain in conditions of poverty may continue to reveal, on a macro scale, the neurobiological changes that occur in such conditions. A thorough understanding of SES-associated deficits in the developing brain may lead to brain-based interventions targeting affected neurocircuitry that is behavioral, pharmacological, or neurotechnological in nature, amplifying the function of affected circuits (14).

The prefrontal cortex is a region of the brain susceptible to the negative effects of SES disparity (33). The PFC subserves decision-making, judgment, and allocation of attentional resources and undergoes a protracted development into adulthood (34). Neuroimaging studies of brain function reveal that parental SES is associated with delayed maturation of the prefrontal cortex, impulsive decision-making (35), deficits in selective attention, and changes in brain activation indicative of deficits in a variety of cognitive abilities such as reading, language, and cognition which persist into adulthood (33,34). Low SES in youth, regardless of SES in adulthood, is associated with impulsive decision making as an adult (35).

Candidate mechanisms for brain differences in poor and middle-class children include differences in the home linguistic environment and stress exposure (6). High-SES families are more likely to speak to children more often and with more linguistic complexity, spend more time reading to their children, and provide increased access to books (2). The increased exposure to linguistic stimuli in high-SES families may be associated with developmental differences in language-supporting cortical regions in the left hemisphere. These disparities may be most strongly related to parental education, which strongly influences the linguistic environment at home (36).

Targeted interventions to reduce effects of poverty on the brain

Because the brain possesses a capacity to rapidly grow and change during the first five years of life, young children are unusually receptive to environmental influences (37, 38). Adequate nutrition as well as nurturance

and care in the early years provides the brain with the sensory stimulation and supportive social climate necessary for normal development (33, 39, 40). Exposure to enriched environmental conditions are associated with improved cognitive performance. Stimulating, enriching environments may cause neuroanatomical and biochemical changes in several brain regions, including frontal, parietal, and entorhinal cortices, as well as hippocampus and cerebellum (33).

In the future, targeted interventions to improve neurocognitive processes may be possible with the use of behavioral, neurotechnological, and/or pharmacological interventions focused on improving aspects of memory, and emotional regulation. Enriching activities such as learning and cognitive tasks increase the growth of new neurons in the hippocampus, while stressful experiences have been associated with the reduction of neurogenesis in this region (16,18,19,31). Environmental stress also has profound effects on the prefrontal cortex, implicated in allocation of attentional resources, emotional regulation, and decision-making, and amygdala, which plays a role in emotion, memory, and cognition as well (33,34,41). These structures are part of a larger, distributed neurocircuitry comprised of limbic cortical and subcortical regions which underlie many of the neurocognitive deficits seen in low-SES children, including memory, language, and executive function (2). Effective interventions may increase the functional and/or structural integrity of these regions.

A few interventions have proven effective for improving cognitive and academic performance in low-SES children. Comprehensive educational pre-school programs have been successful in increasing academic performance in low-SES children with long-term effects into the middle school years. *Head Start* is one such scholastic readiness program designed to reduce the income-achievement gap. A federal program promoting educational readiness in children of up to five years of age, *Head Start* was found to be effective in preventing developmental delay in low-SES youth, as reflected by improved academic performance (42). In the classroom setting, interventions which focus on building core skills including attention, memory, and problem-solving, may mitigate SES effects on brain development (33). *Tools of the Mind*, an educational program which encourages children to use executive functioning, is a promising program which is currently being tested in low-income preschool children (11,12). Other attention-training programs are currently being tested in collaboration with community resources

(32). Perhaps due to the enhancement of cognitive performance in children at a developmentally critical time, pre-school educational programs are associated with better probability of completing high school and college, and increased earnings in the workforce (43). Empirical research investigating the neural correlates of effective interventions in reducing the income-achievement gap are needed (41).

Different facets of early experience affect the developing brain differently: cognitive stimulation influences the development of language, while socioemotional stimulation affects the development of memory, and not language (2). Interventions seeking to eliminate the neural effects of poverty must be multifaceted, seeking to improve socioemotional processing as well as cognition. Socioemotional deficits associated with poverty are thought to derive from inadequate parental interaction and high-conflict and/or stressful home and neighborhood environments, and hippocampal volume is correlated with the amount of parental nurturance a young child receives (18, 44). Parent interventions are effective but can be difficult to execute due to conflicting demands on the parent's time, such as work obligations (11).

Targeted interventions may be a double-edged sword, as neurotechnologies promote "biopower," the ability to manage people as a group; in modern capitalist society, biopower manifests as a promotion of life and regulating the body (45). A government in which life is paramount to other values such as diversity and equality can lead to "unchecked justification" for harm, such as xenophobia or genocide (45). If neuro-centric data supports the belief that low-SES individuals with neurocognitive deficits harm income citizens with health issues harm society, this could be used as a justification for eugenics or euneuromics. Therefore, it is crucial to recognize the issues related to biopower and "embrace a cosmopolitan ethical stance" (45). Targeted interventions must therefore be a multifaceted effort taking into consideration the interests, values, and contexts of populations as well as governments (45).

Conclusion

Severe poverty can be systematic, perpetuating for generations. Investment in the neurocognitive development of low-SES youth may help avert this cycle: researchers estimate that a nationwide early education program could reduce poverty rates from five to 15 percent (46). A nationwide early education program could have additional

benefits including reduced grade retention and need for special education programs; less crime; and greater economic productivity (46). Neurocognitive interventions targeted to low-SES families are therefore an investment in the future of all Americans.

The harmful neurodevelopmental effects of poverty are significant and can persist into adulthood, affecting socioemotional development, education, and health. The scientific literature characterizing neurocognitive differences in low – and high-SES populations is a powerful example of how cognitive neuroscience can be used to examine the health ramifications of significant social problems. Indices of SES status may be associated with decreased function of executive control, memory and language processing networks and decreased development of brain regions involved in socio-emotional processing. Low SES is associated with negative physical and mental health outcomes which may increase susceptibility to neuropsychiatric illness and alter life trajectories. Because the brain continues to develop into adulthood, targeted interventions to ameliorate the neural effects of poverty may be effective in altering life trajectories. The use of government-sponsored SES-based interventions requires an understanding of the values and contexts of populations in order to prevent unjustified harm to citizens.

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Competing interests

The author declares no competing interests.

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