

# Neurobiological Impact of Living in Poverty

Effects on Fear Processing, Stress Regulation, and Decision-making

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## **INTRODUCTION**

According to the World Bank, nearly half of the world's population in 2018 lived on less than \$5.50 a day and struggled to meet basic everyday needs. The U.S. Census Bureau reports that in the United States alone, one in four children is born into poverty, up from one in five in 2011. Studying neurological fear processing in low-income populations can help researchers and policymakers better understand how people living in poverty respond to certain situations and why they may react differently than those with ample resources. Exploring biological bases for fear-related behavior is significant because it can both inform policy decisions related to poverty as well as reduce stigma and discrimination associated with low-income populations. This topic unites neuroscientific findings with the field of poverty studies, bringing seemingly disparate fields together under one common research endeavor. This review will integrate current research findings to address three questions related to the neuroscience of fear in low-income populations: (1) Does poverty during childhood impact future neurological fear processing? (2) Does living in poverty have an effect on the stress system, including the HPA axis and cortisol regulation? (3) Does low socioeconomic status impact the executive function of decision-making?

## **EFFECT OF CHILDHOOD POVERTY ON FEAR PROCESSING**

Neurological processing of fear most strongly involves limbic system structures such as the amygdala (AG) (Davis, 2000; LeDoux, 2000) and the hippocampus (HC), (Phillips & LeDoux, 1992; Lovett-Barron et al., 2014), as well as prefrontal cortex (PFC) areas such as the anterior cingulate cortex (ACC) (Gisella et al., 2011). Childhood poverty has been shown to have effects on the size of these fear-processing brain structures even up to 50 years later in life (Staff et al., 2012). Measures of childhood poverty such as low parental income have been consistently

found to be associated with smaller HC and AG volumes (Noble et al., 2012; Luby et al., 2013; Hanson et al., 2015) as well as with reduced gray matter volume in the ACC (Gianaros et al., 2007). The smaller size of these brain structures in low-income populations could imply abnormal fear-processing ability of these regions. Consistent with this prediction, one longitudinal functional magnetic resonance imaging (fMRI) study has shown that those with lower family income at age 9 exhibit reduced dorsolateral PFC (dlPFC) activity and failure to suppress AG activation during effortful regulation of negative emotion at age 24 (Kim et al., 2013). Regardless of income upon reaching adulthood, childhood poverty has also been found to be associated with higher adult AG reactivity to “threat faces” as opposed to “happy faces” (Gianaros et al., 2008; Javanbakht et al., 2015) as well as with lower functional connectivity between the left AG and the mPFC (Javanbakht et al., 2015). This decreased connectivity could imply an impaired ability of the PFC to downregulate an AG-driven threat response. Childhood poverty has also been found to result in reduced dlPFC recruitment for emotional regulation and decreased HC activation following exposure to acute stress (Liberzon et al., 2014). This collection of evidence suggests that childhood poverty can have a lasting impact on the ability to respond normally to fear-relevant stimuli. This raises the question as to what aspect of childhood poverty could be contributing to this dysregulation in fear-processing brain structure and function. A potential answer may be found in the relationship between poverty, allostatic load, and the neurobiological stress system.

## **EFFECT OF POVERTY ON STRESS SYSTEM**

As compared to children in a more stable living situation, children experiencing poverty grow up with a higher level of environmental stressors such as a chaotic home environment

(Work et al., 1990), lower parental caregiving (Miller & Davis, 1997), increased exposure to toxins (Bullard & Wright, 1993), overcrowding (Clauson-Kass et al., 1997), and less access to healthy foods (Moreland et al., 2002). In children of low SES, the simultaneous occurrence of these environmental stressors can result in an increased “allostatic load” (an accumulation of stress factors) acting on the hypothalamic-pituitary-adrenal (HPA) axis and a subsequent dysregulation of cortisol, leading to chronic “wear and tear” on the body (McEwan & Gianaros, 2010; Evans & Kim, 2012). An abnormally high allostatic load due to poverty and single parent status has been found to be related to blunted activity of the HPA axis and a subsequent decreased ability to regulate cortisol release (Zalewski et al., 2012). Poverty has been found to elevate basal cortisol levels of children from age 7 months to 48 months (Blair et al., 2011) with a greater number of years spent living in poverty corresponding to more elevated overnight cortisol levels (Evans & Kim, 2007). Increased allostatic load due to lower SES has not only been found to be linked to chronically dysregulated diurnal and basal cortisol rhythms as described but also to be associated with higher immediate cortisol reactivity to stressful situations (Hackman et al., 2012). Poverty-related stress during pregnancy due to overactivation of the mother’s HPA axis can even influence the stress system and other parts of the brain of the developing fetus (Lefmann & Combs-Orme, 2014). Dysregulated activation of the neurobiological stress system due to increased allostatic load and chronic “wear and tear” may potentially be what underlies abnormalities in other brain areas such as fear-processing regions in those of lower SES.

## **EFFECT OF POVERTY ON DECISION-MAKING**

Decision-making is a form of executive neural functioning that draws on the contribution

of several more basic psychological processes such as planning and working memory (Kersten & Szpakowicz, 1994; Bechara et al., 1998; Banfield et al., 2004). Those of low SES show decreased self-regulation (Bernheim et al., 2015) and dysfunctional decision-making behaviors (Spears, 2011), which could potentially be mediated by a deficit in the more basic psychological factors that govern decision-making. Consistent with this prediction, low SES has been found to significantly impair measures of executive function such as planning ability (Aran-Filippetti & Richaud de Minzi, 2012), inhibitory control and cognitive flexibility (Sarsour et al., 2011), and working memory in young adults (Finn et al., 2017) as mediated by elevated childhood chronic stress (Evans & Schamberg, 2009). The amygdala and the vmPFC, both implicated in fear-processing as previously mentioned, have also been found to be involved in decision-making (Bechara et al., 2003). The dysregulation of these structures in those of low SES as described previously could also be a contributing factor to impaired decision-making ability. In fact, low access to resources among those living in poverty has been found to manifest itself in a “scarcity mindset,” a shift in allocation of attention due to the perception of not having enough of what one needs (Shah et al., 2012). A scarcity mindset is associated with decreased dlPFC activity and a subsequent abnormality in proper goal-directed decision-making abilities (Huijsmans et al., 2019) as seen in the impairment of decision-making among those of low SES populations.

## **CONCLUSION**

This review highlights that living in poverty has distinct neurobiological effects, specifically on the neural systems of fear processing, regulation of the stress system, and the psychological components of decision-making. Poverty during childhood leads to both decreased volume and decreased functionality of fear-processing systems such as the amygdala and hippocampus well into adulthood, as well as the prefrontal regions that modulate them. This

effect could be due to the stressful environment of poverty, which contributes to an increased allostatic load, chronic overactivity of the HPA axis, and a subsequently impaired ability to regulate both long-term and immediate cortisol release, resulting in increased wear-and-tear on the body and potentially on fear-processing brain systems as well. This combination of maladaptive fear processing and abnormal stress regulation could lead to the dysfunctional decision-making and “scarcity mindset” that is seen amongst low-income populations. While poverty is a complex and multifaceted social phenomenon, its long-lasting and deep-reaching neurological effects demonstrate that brain anatomy and processing can be modulated by the environment and can contribute to the different behavior of the impoverished population.

## **FUTURE PERSPECTIVES**

Neuroscience is only one of many lenses through which the fraught social issue of poverty can be examined. While it may be impossible to eliminate socioeconomic inequality altogether, neuroscience can nevertheless help us understand how to potentially mitigate its impact. Because the neural effects of poverty take root at such a young age and have a lasting influence on fear processing, stress regulation, and decision-making, this suggests that early intervention is key in working to combat behavioral effects of poverty. Policy reform to support early involvement in childhood poverty alleviation programs such as education or after-school workshops as well as parental training could be beneficial to help reduce environmental stressors and dissolve the scarcity mindset. This may promote better working memory capacity and decision-making abilities amongst the impoverished. Future research should bolster current findings with larger sample sizes and more longitudinal studies, as the “neuroscience of poverty” field is relatively new within the last fifteen years. As the neurobiological link between

low SES and abnormal brain structure and function has been established, next steps should include examining the impact of these findings at a psychosocially relevant level. Further research in this field can help better conceptualize how people experiencing poverty behave differently, why they often find themselves “stuck” in the cycle of poverty, and what interventions can be instituted to take advantage of neurobiology in order to break that cycle.

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