



Introduction to the Special Focus: The Affective Neuroscience of Poverty

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Abstract

■ Growing up in poverty is associated with a heightened risk for mental and physical health problems across the life span, and there is a growing recognition of the role that social determinants of health play in driving these outcomes and inequities. How do the social conditions of poverty get under the skin to influence biology, and through what mechanisms do the stressors of poverty generate risk for a broad range of health problems? The growing field examining the neuroscience of socioeconomic status (SES) proposes that the brain is an entry point or pathway through which poverty and adversity become embedded in biology to generate these disparities. To date, however, the majority of research on the neuroscience of SES

has focused on cognitive or executive control processes. However, the relationship between SES and brain systems involved in affective or emotional processes may be especially important for understanding social determinants of health. Accordingly, this Special Focus on *The Affective Neuroscience of Poverty* invited contributions from authors examining the relationship between SES and brain systems involved in generating and regulating emotions. In this editorial introduction, we (a) provide an overview of the neuroscience of SES; (b) introduce each of the articles in this Special Focus; and (c) discuss the scientific, treatment, and policy implications of studying the affective neuroscience of poverty. ■

Poverty is a powerful risk factor for mental and physical health problems across the life span. Socioeconomic status (SES) is associated with depression, anxiety, psychosis, and academic achievement, as well as heart disease, stroke, cancer, diabetes, and early mortality (McLaughlin, Costello, Leblanc, Sampson, & Kessler, 2012; Adler & Stewart, 2010; Kessler et al., 2005; Sirin, 2005). There is a growing recognition of the important role that social determinants play in driving these mental and physical health inequities (Braveman & Gottlieb, 2014). Social determinants of health are the conditions in the environment where people are born, live, learn, work, and play that affect a wide range of health and quality-of-life outcomes (Marmot et al., 2008). An important question is, how do the social conditions of poverty get under the skin to influence the biology of a developing child? And through what mechanisms do these social determinants generate risk for such a broad set of mental and physical health outcomes? Over the past decade, researchers have begun to examine the role of the brain in answering these questions (Noble & Giebler, 2020; Farah, 2017). From this perspective, the brain is an entry point or pathway through which poverty and adversity become embedded in biology to generate health disparities (McEwen & Gianaros, 2010). Collectively, this small but growing field examining relationships between the brain, poverty, and health is referred to as the neuroscience of SES.

SES is construed as a dimension that varies from “worse off” to “better off,” with those who are better off having more material resources (e.g., income) and nonmaterial resources, including education and neighborhood quality. Historically, SES has been relegated to the status of a covariate or confound in the field of neuroscience. However, there is increasing evidence that the stress of living in poverty affects the developing brain in a manner that deserves its own investigation (Noble, Engelhardt, et al., 2015; Brito & Noble, 2014). The growth of knowledge on this topic is apparent from the fact that there were only a handful of studies on the neural correlates of SES in early reviews (Raizada & Kishiyama, 2010; Hackman & Farah, 2009), compared with dozens of relevant studies today. We now know from neurophysiology and both structural and functional imaging studies that early exposure to poverty is associated with alterations in brain systems involved in a variety of cognitive processes, including executive control, memory, and language (see Johnson, Riis, & Noble, 2016, for a review). Some of these studies report that neural alterations mediate the linkage between poverty exposure and cognitive processes (Hair, Hanson, Wolfe, & Pollak, 2015; Noble, Houston, et al., 2015; Mackey et al., 2015), suggesting they are not simply correlates of SES, but possible mechanistic pathways to outcomes that matter.

Most research on the neuroscience of SES has focused on cognitive processes. This work builds on the cognitive neuroscience of language, memory, and executive function and holds promise for understanding the SES achievement gap, as well as later occupational success (Farah,

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2017). However, it may be especially important to examine the relationship between SES and brain systems involved in affective processes if we're to better understand social determinants of mental and physical health. Chronic adversity, including poverty, negatively affects the structure and function of brain regions involved in emotion, including the amygdala, insula, ventral striatum, and portions of the pFC (see McLaughlin, Weissman, & Bitran, 2019, for a review). These same brain regions are implicated in numerous mental health problems, including anxiety, depression, and various externalizing disorders (Keren et al., 2018; Baskin-Sommers, 2016; Shackman et al., 2016), all of which correlate with SES (McLaughlin et al., 2012; Lorant et al., 2003). They also regulate homeostatic processes implicated in chronic inflammation and stress-related physical diseases, including metabolic syndrome, coronary heart disease, and autoimmune conditions (see Nusslock & Miller, 2016 for a review). Thus, multiple lines of evidence suggest that emotional brain systems may be affected by the stressors of poverty and contribute to the mechanistic pathways through which SES affects health. Accordingly, this Special Focus on *The Affective Neuroscience of Poverty* is intended to showcase research on SES and brain systems involved in generating and regulating emotions. In keeping with the *Journal of Cognitive Neuroscience's* emphasis on basic science over diagnostic and/or treatment-relevant work, the articles in this Special Focus present findings from basic affective neuroscience rather than investigations of patient populations.

The first set of articles in this Special Focus examined the relationship between SES and neural responses to emotional stimuli. Alvarez, Rudolph, Cohen, and Muscatell (2022) report that individuals with a lower socioeconomic position displayed greater activity to both positive and negative images in brain regions involved in emotion processing and homeostasis. Next, White, Nusslock, and Miller (2022) report that low SES was associated with greater activation in brain regions involved in attention to both reward and loss cues and reduced differentiation in the brain between reward and loss feedback. Both of these results are consistent with the concept of the "scarcity mindset," which suggests that individuals living with minimal resources are sensitive to cues of both gain and loss and that the metabolic demand of this hypersensitivity can generate health problems overtime (Shah, Shafir, & Mullainathan, 2015).

The next set of studies examined mediators and moderators of the relationships between SES, brain structure and function, and emotional states and traits. Hao, Bertolero, and Farah (2022) tested whether healthy young adults of low SES generally experience more negative emotions and whether the volume of the amygdala and its reactivity to emotional stimuli mediate the association between SES and negative emotions. Weissman et al. (2022) investigated the neural mechanisms through which two dimensions of adversity—threat and deprivation—might contribute to SES disparities in psychopathology. They

report that greater exposure to threat, but not deprivation, was associated with higher activation in the dorsomedial pFC and precuneus to fearful faces and that precuneus activation mediated the association between SES and post-traumatic symptoms. Hackman et al. (2022) tested whether SES moderated the relationship between school climate, which is important for children's socioemotional development, and cortical thickness, cortical surface area, and subcortical volume. These three studies help us understand the neural mechanisms through which poverty may facilitate negative emotions and under what conditions these mechanistic associations may emerge.

The last set of articles is consistent with the growing recognition in human neuroscience of the importance of moving beyond only examining brain regions in isolation of each other and to also assess functional and structural connections between brain regions. This is premised on the fact that both normative and nonnormative mental states likely emerge from distributed neural networks, rather than any one particular area of the brain in isolation (Bassett, Xia, & Satterthwaite, 2018; Braun et al., 2018). Ip et al. (2022) present findings from the Adolescent Brain Cognitive Development study on the associations between socioeconomic disadvantage, resting-state functional connectivity between the medial OFC and amygdala, and internalizing symptoms in 9- to 10-year-old youth. Hardi et al. (2022) used diffusion imaging to examine the relationship between white matter structural connectivity within frontolimbic structures and material hardship at different ages along the developmental spectrum. They report that the associations between frontolimbic connectivity and material hardship differ across prefrontal regions and developmental periods, providing support for potential windows of plasticity for structural circuits that support emotion.

There are at least three ways that the research exemplified here can benefit science and society. First, by elucidating the mechanisms through which poverty becomes embedded in biology, we will better understand an important source of individual differences in thinking, feeling, and health. This research will inevitably bring attention to environmental factors and structural inequities in our society and to our collective responsibility to improve the environment for low SES individuals. Despite the biological nature of brain differences, it does not follow that they are genetically caused; a substantial proportion of SES effects on neuroanatomy can be attributed to environmental causes (Kweon et al., 2022). A biological approach to SES disparities in brain and health in no way blames the poor or implies that DNA is destiny. Second, like any basic or preclinical science addressing an important social or medical problem, we anticipate that this work will eventually inform prevention and intervention strategies. Although the neuroscience of SES is a young field, it has already been used to better understand the effects of interventions on low-income children (Farah et al., 2021; Brody et al., 2017). Third, this work may help

facilitate policies that target structural inequities in our society that contribute to or drive health disparities. As noted by Muna Abdi, an Education and Racial Equity Consultant, “instead of praising people for being resilient, change the systems that are making them vulnerable” (personal communication with R. N.). There is early evidence that such changes can have a salubrious effect on the brain and body. In one of the first studies of its kind, Troller-Renfree et al. (2022) demonstrated that a modest monthly cash transfer to low-income families had a causal impact on infant brain activity that has been associated with the development of subsequent cognitive skills. We believe that the articles in this Special Focus and the neuroscience of SES more broadly provide a new source of support for investment in the needs of individuals and families in poverty.

Many fields, including psychology, sociology, epidemiology, and economics, have tried to understand the causes and consequences of health disparities. We are not suggesting that neural explanations replace these important perspectives, but that they can provide a complimentary viewpoint that can help us understand the mechanisms through which the socioeconomic environment leads to socioeconomic disparities. Ultimately, the value of neuroscience for understanding SES and facilitating interventions and policies is an empirical question that needs to be investigated. To facilitate this, we recommend that studies of human neuroscience consider including measures of SES. We also recommend that we as a field move beyond simply considering SES as a nuisance variable or covariate and instead examine its associations with primary variables and test for moderation and/or mediation. Finally, we argue that it is time for us as a field to move beyond relying on predominately middle- to upper-class participants or undergraduate subject pools and to include more participants from lower SES backgrounds. This will help us generate questions and findings that are more generalizable and better position us to apply our work to important societal issues such as poverty and health disparities.

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Diversity in Citation Practices

Retrospective analysis of the citations in every article published in this journal from 2010 to 2021 reveals a persistent pattern of gender imbalance: Although the proportions

of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience (JoCN)* during this period were M(an)/M = .407, W(oman)/M = .32, M/W = .115, and W/W = .159, the comparable proportions for the articles that these authorship teams cited were M/M = .549, W/M = .257, M/W = .109, and W/W = .085 (Postle and Fulvio, *JoCN*, 34:1, pp. 1–3). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article’s gender citation balance.

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