Socioeconomic influences on brain function: implications for health

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Socioeconomic-based disparities in physical health outcomes are well established, with individuals from lower socioeconomic status (SES) backgrounds being more likely to experience chronic disease morbidity and early mortality compared to those from higher SES strata. While numerous studies in recent decades have focused on understanding the contextual, psychosocial, and biological mechanisms linking SES and health, the neural pathways that contribute to this relationship are currently underinvestigated. The present paper reviews and synthesizes the small number of published studies that have explored links between SES and health-relevant neural functioning. Specifically, current knowledge of the relationship between socioeconomic factors and neural systems that may be affected by low SES contexts, including those related to processing threat and stress, responding to reward, and engaging in emotion regulation, is reviewed. Gaps in our knowledge that could be filled by health neuroscience research are emphasized, in an effort to catalyze future studies in this area. Understanding the neural mechanisms linking SES and health is crucial for building comprehensive models of the pathways by which social inequalities become health inequalities and may help identify novel targets for intervention to prevent health disparities. Health neuroscience research has a critical role to play in this important area of research.

Keywords: socioeconomic status; social status; health disparities; health neuroscience; brain function

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Social and economic inequalities are currently at the forefront of the American cultural conversation, given increasing attention to economic disparities and heightened awareness of the existence of systemic social biases against certain racial groups. While historically neuroscientists have not engaged deeply with questions about how socioeconomic and other demographic factors influence brain function, a growing literature in health neuroscience¹ has begun to investigate how social factors get “under the skull” to influence neural processes that are relevant for physical and mental health outcomes.²⁻⁶ Indeed, neuroscientists have much to bring to the conversation regarding social inequalities and their effects on cognitive, affective, social, and health outcomes.⁷,⁸

Most work that has investigated socioeconomic status (SES) influences on brain function in the past two decades has focused on the development of neural systems important for cognitive function (e.g., working memory, long-term memory, language, and executive functioning).⁷,⁸,¹⁰ This important work by developmental cognitive neuroscientists has shed light on how SES environments in early life influence brain development that may play a role in educational outcomes, which could perpetuate socioeconomic disadvantage across generations. More recently, a growing literature in health neuroscience has focused on how SES influences neural activity important for health, focusing on the functioning of brain systems that...
are relevant for physiology and health behavior.\textsuperscript{5,11} While this nascent literature demonstrates that socioeconomic factors do indeed influence brain systems that are linked with poor health outcomes, much more work is needed in this area.

With this backdrop in mind, the purpose of the present review is to synthesize prior health neuroscience research that has examined how SES affects functional neural activity in brain circuitry relevant for health, and offer recommendations for future research in this area. I begin with a brief discussion of issues related to how we define and measure socioeconomic factors in neuroscience research. Then, I provide a general overview of the potential pathways by which SES may influence brain function, focusing on psychological, cultural, and neighborhood factors. I also review the neural systems that may play a role in linking SES and health. Next, I dive in to the crux of the paper: a review and synthesis of the health neuroscience literature that has empirically interrogated the links between SES and health-related brain functioning, focusing on associations between SES and neural activation in response to threat and stress, during reward processing and during emotion regulation (Table 1). I provide a novel conceptual model (Fig. 1) that attempts to integrate the current health neuroscience literature together with research in social psychology, health psychology, and public health to provide a framework for thinking about the pathways by which SES may influence brain function and, subsequently, health outcomes. Recommendations for future work that could fill gaps in our current knowledge are made throughout. I conclude with a broader picture of ways in which neuroscientists can contribute to our understanding of the pathways linking SES and health, and additional ideas for future work in this area. Overall, the goal is to encourage cognitive, affective, developmental, and social neuroscientists to think more about ways in which they can incorporate SES into future studies, and to provide a picture of the growing literature exploring SES influences on brain function for non-neuroscientists who are interested in health disparities. I hope this overview of the literature will provide a common knowledge base to facilitate increased collaboration between neuroimagers and disparities researchers to further our understanding of how SES affects brain function in ways that may impact health.

Defining and measuring SES

What is the best way to define and measure SES in health neuroscience research? While finding an answer to this question may seem simple at first blush, it is in fact quite a complex issue that deserves careful attention during study design and analysis. Many prior papers have offered insight into and perspective on this important issue,\textsuperscript{12,13} so for the purposes of the present paper, a brief overview of different measures of SES and their potential utility in examining health disparities is provided (for more in-depth discussion, see Refs. 12 and 13).

Educational attainment and income are very commonly used as indicators of SES in the existing literature in health neuroscience and in health disparities research more broadly. These measures have the advantages of being relatively straightforward for participants to report on, as well as allowing researchers to split individuals into meaningful categories or extreme groups based on reported income and occupation (e.g., poverty versus not, less than high school education versus college degree). However, it should be noted that this approach may obscure meaningful gradients across the entire SES spectrum,\textsuperscript{14,15} and as such, treating education and income as a continuous scale may be more useful. It is also important to point out that education and income are often only modestly correlated,\textsuperscript{12} and they may confer different advantages/disadvantages when examined in combination with race/ethnicity (e.g., a Black American with the same level of education as a White American is likely to have lower income) and other demographic factors,\textsuperscript{12} thus demonstrating that the two should not be used interchangeably or as proxies for one another.\textsuperscript{12} Finally, it is important to appreciate the ways in which education and income may influence neural function and health through similar or different pathways. For example, while both may influence availability of material resources, education may provide other noneconomic benefits (e.g., health literacy, problem-solving abilities, and prestige)\textsuperscript{12,16} that could also impact brain function. In sum, it is important to consider educational attainment, income, and their interacting effects with other demographic factors (e.g., sex and race/ethnicity) as potential indicators of SES that could influence brain function and health.
Beyond education and income, numerous other indicators of SES are used in the literature and may also be useful to measure in the context of neuroimaging studies. Occupation is often employed as an SES measure in studies of health outcomes in European countries (e.g., the seminal Whitehall studies in the UK\textsuperscript{15,17}), and occasionally in the United States as well.\textsuperscript{18} However, difficulty in categorizing occupations based on skills, prestige, and power makes this a challenging SES measure to use. Recently, more attention is being paid to the importance of neighborhood-level SES (e.g., neighborhood income and education) in addition to individual SES in predicting health outcomes.\textsuperscript{19–21} Relatedly, relative deprivation and economic inequality (at the local and macro level) are also increasingly being appreciated as predictors of health outcomes.\textsuperscript{22–24} In other words, it is not just one's education, income, and occupation that matter for health, but also features of the broader economic environment at the neighborhood and societal level that contribute to disparities. Unfortunately, both neighborhood SES and relative deprivation/inequality may be challenging to measure in the context of health neuroscience studies; collaborations with researchers in psychology, public health, and sociology who focus on these factors in their work will be useful in moving forward. Finally, subjective perceptions of one's social status and SES are also important for health and may influence neural responses. For example, the widely used MacArthur Subjective Social Status Scale (often referred to as the “ladder”),\textsuperscript{25,26} which asks individuals to rank their standing in society based on their education, income, and occupation, has been shown to predict health outcomes, often over-and-above the effects of more “objective” indicators of SES, such as years of education or household income.\textsuperscript{27–29} It has been proposed that this single-item measure of subjective social status may reflect some amount of “cognitive averaging” of different SES indicators,\textsuperscript{27} and may also better reflect the level of prestige and “social capital” inherent to SES measures, which may be lost in looking at objective SES measures (i.e., two people with the same degree may have attended very different universities that afford differing levels of status despite offering the same degree). In sum, SES is a multifactorial construct that can be measured and operationalized in different ways, each of which presents both challenges and opportunities for data collection and interpretation. Careful consideration should be paid to which SES measure(s) are selected for inclusion in a study, as distinct mechanisms may link different facets of SES (e.g., income versus subjective status) to brain functioning, and some neural systems may be more affected by one SES indicator over another.

Any discussion of SES conceptualization and measurement would be remiss without considering the developmental timing of SES influences on brain function and health. Indeed, a large body of literature has now accumulated to suggest that SES in early life (during gestation, in early childhood, and during adolescence)\textsuperscript{30–32} is associated with health outcomes in adulthood, often even when controlling for concurrent SES. While there is certainly a relationship between SES in early life and SES in adulthood, there is a reason to suspect that there may be “sensitive periods” during development in which SES may exert a stronger influence on brain function and health outcomes than during other periods.\textsuperscript{30} As such, assessment of SES both in early life (i.e., via retrospective reports of parental education, occupation, income, and subjective status) and in adulthood (i.e., current) is warranted and may provide valuable information regarding how SES affects the brain and the body.\textsuperscript{12} Further, we need future neuroimaging studies in both adults and children/adolescents to develop a clearer picture for how SES influences brain function at different developmental stages.\textsuperscript{33}

Finally, it is also important to note that though low SES is a major risk factor for disease morbidity and early mortality, not all individuals from lower SES backgrounds suffer from poor health outcomes. Indeed, recent research is starting to uncover the social, psychological, and biological processes that may confer resilience to low SES circumstances, or the processes that allow some individuals to thrive and live long, healthy lives despite socioeconomic disadvantage. One prominent model of resilience to low SES circumstances, the “shift-and-persist” model, suggests that, with the help of positive role models, children from lower SES backgrounds can develop an adaptive approach to coping with disadvantage that involves shifting perspectives (i.e., accepting stressors for what they are, reappraising negative contexts and experiences), and persisting in the face of challenges (i.e., finding meaning, maintaining optimism about the future), which may be...
health protective.\textsuperscript{34} Another influential model, the reserve-capacity model, suggests that interpersonal resources including greater social support and social integration, as well as intrapersonal resources such as high levels of mastery, optimism, and self-esteem, may also support resilience among individuals from lower SES backgrounds and be associated with better health outcomes.\textsuperscript{35} Given the increasing emphasis on resilience in the broader health disparities literature, it will be important for health neuroscience research investigating SES influences on brain function to incorporate the idea of resilience in future studies, some ideas for which are suggested in subsequent sections of this paper.

**Pathways linking SES, brain function, and health: psychological and physical contexts**

With this consideration of issues related to SES definition and measurement in mind, I now turn to the question of how a “macro level” factor like SES may come to influence the functioning of individual-level processes, such as neural activity. Certainly, the pathways connecting SES to brain function and ultimately to health are multiple, varied, and interact with one another and with other factors (e.g., race/ethnicity and gender) in complex ways. Numerous important models exist that provide tremendous insight into the interacting environmental, psychological, and biological mechanisms linking SES and health,\textsuperscript{5,36–39} and reviewing them all is beyond the scope of the present paper. Instead, I will highlight three possible pathways linking SES, brain function, and health here: psychological stress, cultural orientation, and neighborhood contexts. I focus on these three pathways based in part on their well-established links with brain functioning in the health/social neuroscience literature, and also on their links with health more broadly.

**Psychological stress**

Unsurprisingly, one commonly posited mechanism that likely contributes to SES disparities in health that may also influence the functioning of health-relevant neural systems is psychological stress.\textsuperscript{5} Prior empirical investigations have demonstrated that lower SES is associated with increased exposure to stressful life events\textsuperscript{40} and greater vulnerability to the deleterious effects of stressful life events when they occur,\textsuperscript{41} higher levels of job strain\textsuperscript{42} and lower job control,\textsuperscript{43} and more severe daily hassles.\textsuperscript{44} Studies of SES influences on physiological reactivity to stress have also shown that individuals from lower SES backgrounds or those who report lower subjective social standing show greater physiological reactivity to psychological stress across a variety of biological systems, including the autonomic nervous system (ANS),\textsuperscript{45} hypothalamic-pituitary-adrenal (HPA) axis,\textsuperscript{25} and the innate immune system (i.e., inflammation)\textsuperscript{46–48} which, over time, could lead to increases in allostatic load and poor health.\textsuperscript{49,50} Further, a large body of work utilizing animal models and a burgeoning literature in human neuroimaging shows that psychological stress influences neural functioning in a variety of different brain systems (see below for more detail).\textsuperscript{4,51–53} Thus, psychological stress, both acute and chronic, likely plays an important role in linking SES, neural function, and health.

**Cultural orientation**

While historically SES influences on health and behavior have been studied by researchers in health psychology, sociology, and public health, more recently, social psychologists have also begun to investigate how SES influences social cognition and social behavior in ways that may be relevant for health. This work has yielded some fascinating theoretical and empirical insights into the fact that different levels of SES may be associated with a distinct set of norms and values related to how one should interact with others in their environment and how one should think about the self in relation to others.\textsuperscript{39,54,55} The idea of “social class as culture” posits that individuals from lower SES backgrounds may have a more interdependent self-construal or communal orientation and greater appreciation for how contextual factors influence behavior, while individuals from higher SES backgrounds may have a more independent self-construal and emphasize personal agency and dispositional explanations for behavior.\textsuperscript{54,55} These distinct cultural lenses may lead to differences in emotion and social behavior that have implications for health. For example, research has shown that lower SES individuals are more engaged in social interactions\textsuperscript{56} and are better at understanding others’ emotions\textsuperscript{57} compared to higher SES individuals. While social engagement, perspective-taking, and empathy are typically viewed as positive
characteristics, other recent research shows that they may also come with costs. Along these lines, individuals who are more empathic have been shown to have higher levels of inflammation, a marker of innate immune system activation that is linked with a variety of negative health outcomes. This may be particularly true when enhanced perspective taking occurs in negative contexts (i.e., having a child with high levels of depressive symptoms). In sum, recent work in social psychology suggests that lower SES individuals may have a more other-focused cultural orientation, which could result in enhanced physiological activation linked with poor health, particularly in negative contexts.

Neighborhood contexts
A third pathway that may link SES to neural activation and subsequently to health relates to conditions in the environments (e.g., neighborhoods and communities) that individuals from different SES groups inhabit. For example, lower SES individuals may have fewer opportunities for engaging in health-protective behaviors given a lack of neighborhood and community resources. Along these lines, low- and medium-SES neighborhoods have fewer free-for-use facilities for physical activity (i.e., public parks, sport facilities, and walking/biking trails) than higher SES neighborhoods, suggesting that affordable opportunities for physical activity and outdoor play may be limited. Further, lower SES neighborhoods have been characterized as “food deserts,” with low physical proximity to supermarkets and grocery stores and greater density of convenience stores and fast food outlets. This lack of availability of high-quality, nutrient dense foods combined with the higher prices of such foods when they are available may lead low SES individuals to consume more unhealthy foods. Research has also shown that lower SES neighborhoods have greater prevalence of tobacco marketing as well as greater availability of tobacco products in comparison to higher SES neighborhoods. Finally, individuals living in lower SES neighborhoods report greater perceptions of neighborhood crime and violence, higher untrustworthiness of neighbors, and greater prevalence of drug use, all of which contribute to a backdrop of greater stressful circumstances in low SES communities. Taken together, this body of previous research suggests that the neighborhoods in which low SES individuals live their lives are characterized by few opportunities for positive health behaviors and greater stress at the community level, all of which may affect neural function and health.

To sum up, while there are undoubtedly many pathways by which SES influences brain functioning relevant for health, and here I highlight three: psychological stress, cultural orientation, and neighborhood contexts. Together, these psychological and physical contexts provide a backdrop for understanding the ways in which macro-level demographic factors like SES may get “under the skull” and affect neural functioning. In turn, this differential neural responsivity may influence physiological reactivity and health behaviors, a topic reviewed in more depth in the following section.

Overview of health-relevant neural circuitry
As mentioned previously, a large body of work in developmental cognitive neuroscience has focused on how SES affects brain development in regions important for educational outcomes. Here, I instead focus on how SES influences neural responses to threat and stress, to reward, and during emotion regulation. These domains are focused on for two primary reasons: (1) most health neuroscience research that has examined neural processes affected by SES focuses on these areas and (2) there is evidence suggesting that neural responses to these tasks are linked with health. While a comprehensive review of each of these systems and their role in health-related processes is beyond the scope of this paper (and is covered expertly by other articles in this volume), the following section provides a brief background on the neural underpinnings of these processes, and their links with health, before turning to how SES modulates neural responsivity in these domains.

Neural responses to threat and stress
A large body of neuroimaging research has investigated the neural systems engaged during the processing of threatening social information (e.g., negative emotion expressions), as well as during acute stressors. Regions whose activity is often increased during such tasks and is associated with downstream increases in physiological activation include the amygdala, both pregenual (pACC) and dorsal anterior cingulate cortex (dACC), the anterior insula, more dorsal aspects of medial
prefrontal cortex (DMPFC), and the posterior cingulate cortex (PCC), in addition to others observed less consistently (e.g., periaqueductal gray and hippocampus). Activation of these neural regions during threat and stress is linked with health outcomes primarily via their associations with physiological responses in the ANS, HPA axis, and inflammatory processes. For example, numerous studies have linked amygdala activity to heightened cardiovascular, cortisol, and inflammatory responses to stress. The engagement of the DMPFC, MPFC, and PCC during stress and threat may reflect greater mentalizing, or thoughts directed at understanding others, during these experiences. Given that social evaluation is a potent activator of both the HPA axis and inflammatory processes, greater engagement of regions involved in thinking about others may also lead to physiological activation that could affect health. In sum, activation of the amygdala, anterior insula, DMPFC, MPFC, and PCC during tasks involving threat and stress is important for initiating physiological activation that may be adaptive for dealing with stress in the short term, but, over time, can lead to increases in allostatic load that put an individual at-risk for poor health outcomes.

**Neural responses to reward**

Neural responses to rewarding or valued stimuli are another mechanism by which SES may influence health. A large literature now exists showing that the ventral striatum, the ventromedial prefrontal cortex (VMPFC), and sometimes, the amygdala are active during the processing of a variety of rewarding stimuli, including monetary rewards, palatable food cues, images of cigarette smoking among smokers, and social rewards. In the short term, enhanced neural activity to cues of reward may have stress-reducing properties, as past animal research has demonstrated that pleasurable behaviors (e.g., palatable food intake and sexual activity) dampen behavioral and physiologic responses to stress by decreasing activity in the amygdala and human neuroimaging research suggests that greater activity in reward-related regions may also dampen amygdala activity and result in lower levels of physiological activation. However, greater activity in the ventral striatum during the processing of rewards has also been associated with a difficulty with “curbing craving” for palatable food, as well as for tobacco products and other substances. Further, the relationship between neural reactivity to reward and reward-seeking behavior is a dynamic process that may change over time as addiction and dependency develop. For example, obese individuals often show reduced striatal responses to rewarding stimuli, which made lead to overconsumption to reach the same “rewarding threshold.” Thus, neural responses to rewarding stimuli (both enhanced and diminished reactivity) may be linked with negative health behaviors, such as overconsumption of unhealthy foods and drug and tobacco use, which pose long-term risks to health.

**Neural underpinnings of emotion regulation**

A third neurocognitive domain that may be affected by SES and that is important for health is related to emotion regulation. Emotion regulation can be defined as “... how we try to influence what emotions we have, when we have them, and how we experience and express these emotions.” One commonly studied method of emotion regulation (particularly in the neuroimaging literature) is cognitive reappraisal, or thinking about a stimulus differently so as to alter its emotional impact. A recent meta-analysis of functional magnetic resonance imaging (fMRI) studies that have examined neural responses during cognitive reappraisal revealed that a set of regions is consistently active during this process, including posterior DMPFC/ACC, dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), as well as posterior parietal cortex. It appears that emotion regulation strategies lead to reductions in negative affect via downregulation of amygdala reactivity by these prefrontal regions. Beyond reappraisal, neuroimaging studies of other emotion regulation tasks, including affect labeling and self-distancing, show a similar pattern of enhanced activation of lateral prefrontal regions and corresponding reduced activation in the amygdala during emotion regulation.

How are neural responses during emotion regulation linked with health? In an important early study in this area, Urry and colleagues found that older adults who successfully modulated amygdala activity during a reappraisal task showed a steeper decline in cortisol levels throughout the day, suggesting that successful emotion regulation may be related...
to healthier HPA axis functioning.90 Since then, other work has shown that greater activity in the dACC during a cognitive reappraisal task is linked with greater evidence of preclinical atherosclerosis, as mediated by greater levels of inflammation (i.e., interleukin-6).91 Given evidence reviewed above suggesting that amygdala activity may drive physiological reactivity to stress and threat, the ability to successfully downregulate amygdala responses during negative situations may be critical for controlling physiological responses that are important for health.

**Evidence linking SES and activation of health-relevant neural systems**

With this backdrop regarding the key neural systems that are relevant for health in mind, I now turn to the crux of this review: the literature linking SES and neural activation during threat/stress processing, reward processing, and emotion regulation. While relatively small, this growing health neuroscience literature provides important insight into the neural mechanisms by which SES may affect health. At the end of each section, ideas for future research are provided, with the hopes that new studies will be conducted to fill gaps in our current knowledge and expand our understanding of the ways in which SES gets into the brain to affect health.

**SES and neural responses to threat and stress: existing evidence**

In one of the earliest papers to explore the relationship between SES and neural activity, Gianaros and colleagues92 found that an individual’s perceptions of their parents’ social standing was associated with amygdala reactivity to angry faces (versus neutral faces and shapes), such that individuals who perceived their parents as having lower standing showed greater amygdala reactivity to angry faces. These effects held even after controlling for a variety of other variables, including objective measures of parent education and participants’ own social standing, suggesting that perceived social standing of one’s family during childhood is an important correlate of amygdala activity later in life. Since these important initial findings, a number of studies have replicated the observed negative association between SES and amygdala reactivity to negative social cues. For example, Muscatell and colleagues93 reported a negative association between family SES (i.e., maternal education and household income) and amygdala reactivity to angry faces in a sample of adolescents, and, Javanbakht and colleagues94 found that young adults who had spent their childhoods in poverty (as measured by family income-to-needs ratio) showed greater amygdala activity to fearful faces (versus happy faces and shapes) compared to those who had grown up in middle-income households.

With links between SES and amygdala reactivity to negative social information relatively well established, recent work has moved toward exploring the proximal biological mechanisms by which low SES may lead to greater amygdala activity. In an exciting recent study, Swartz and colleagues95 examined the role that epigenetic modification of gene expression may play in linking SES and amygdala reactivity. Using a prospective, longitudinal design, results showed that lower family SES (i.e., parent education and income) at ages 11–15 was associated with greater increases in DNA methylation (a marker of epigenetic modification) of the proximal promoter region of the serotonin transporter gene (SLC6A4) at ages 13–18. Greater change in serotonin transporter gene methylation was in turn associated with greater change in amygdala reactivity to fearful facial expressions (versus shapes) from age 11–15 to 13–18, suggesting an epigenetic mechanism by which low SES in early life may be translated into greater amygdala activity to negative social information. These exciting findings point to the value of utilizing multiple levels of analysis (e.g., environmental, genetic, and neural) in exploring SES influences on brain function relevant for health.

To sum up, a handful of studies have now found that lower SES is associated with greater amygdala reactivity to negative social cues,92–94 and more recent work shows that methylation in the serotonin transporter gene is one possible mechanism underlying this association.95 Interestingly, all of these studies examined SES in early life (either through retrospective reports or by utilizing adolescents/young adults as participants and examining concurrent SES), suggesting the possibility that associations between SES and activity in threat-related neural systems may be established early in development and persist into adulthood. This is consistent with research showing that other types of early adversity (i.e., institutional care, abuse, and neglect) also affect amygdala functionality both concurrently in early life and
later into adulthood. Together, this body of work suggests the possibility that multiple types of early adverse experiences, including growing up in a low SES environment, shape amygdala reactivity into adulthood. Given that the amygdala has strong links to hypothalamic and brainstem regions that initiate physiological activation (e.g., ANS reactivity, HPA axis activity, and inflammation), it is possible that this enhanced amygdala sensitivity to social threat among low SES individuals may play a role in contributing to health disparities.

Another region that has emerged consistently in studies of SES and neural responses to threat and stress is the DMPFC. For example, in the same study mentioned above that found an association between family SES and neural responses in the amygdala to angry faces in adolescents, there was a similar negative association between SES and neural responses in the DMPFC. Indeed, adolescents from lower SES families showed greater activity in this mentalizing-related region when processing angry faces, relative to adolescents from higher SES families. A
more recent investigation explored if activation in the DMPFC during negative social feedback mediates the association between SES and inflammatory responses to stress.\textsuperscript{47} To address this, participants completed a scanner-compatible social evaluative stress task, in which they received negative, positive, and neutral feedback from an evaluator. Markers of inflammation (i.e., levels of the inflammatory cytokine interleukin-6) were measured before and after the stressor, and participants reported on their subjective SES using the ladder measure of social status. Results revealed that individuals reporting lower subjective social status showed a greater increase in inflammation in response to the stressor, and that neural activity in the DMPFC in response to the negative feedback (versus neutral) mediated this association. In other words, the tendency of lower status individuals to activate the DMPFC in response to negative feedback was associated with greater inflammatory reactivity. This multi-system study incorporating measures of SES, neural responses to stress, and inflammation is one of the first to bring neuroimaging research together with psychoneuroimmunology work to examine neural mechanisms that may link SES and health. Together with the literature linking SES and amygdala reactivity reviewed above, it appears that activity in regions involved in processing motivational-salience (i.e., amygdala)\textsuperscript{98} and in mentalizing (i.e., DMPFC) is modulated by SES.

Given the dense connections between the amygdala and the medial prefrontal cortex, it is worth noting here some of the research that has explored functional connectivity between the amygdala and distinct subregions of the MPFC. Only one known study has examined how SES influences amygdala–MPFC functional connectivity, and the findings suggest that individuals from lower SES backgrounds show decreased functional connectivity between more ventral portions of MPFC and the amygdala, relative to higher SES individuals.\textsuperscript{94} Coupled with a large body of literature demonstrating that other types of early life stress (e.g., neglect, institutional rearing, etc.) are associated with decreased amygdala–VMPFC connectivity,\textsuperscript{96} and research suggesting that VMPFC (extending into sACC) plays an important role in downregulating amygdala activation in fear extinction,\textsuperscript{99,100} these data suggest the interesting possibility that there may be SES differences in VMPFC (sACC)–amygdala connectivity, with lower SES individuals showing less positive coupling between these regions. Interestingly, while no known research has examined SES influences on connectivity between more dorsal MPFC subregions and the amygdala, other work has shown that enhanced DMPFC–amygdala connectivity is associated with enhanced fear learning,\textsuperscript{100} anxiety,\textsuperscript{101} and inflammatory responses to acute stress.\textsuperscript{70} Thus, while more ventral aspects of MPFC (extending into sACC) may inhibit amygdala responding, more dorsal aspects of MPFC (extending into dACC) may amplify amygdala reactivity.\textsuperscript{100} More research is needed to clarify how SES may influence these patterns of amygdala–MPFC connectivity in ways that are relevant for health.

\textit{SES and neural responses to threat and stress: ideas for future research}

While arguably the most research to date on SES influences on neural functioning has examined neural reactivity in response to stress and threat, there is still much to be learned in this domain. For example, following the lead of recent work by Swartz and colleagues\textsuperscript{95} and Muscatell and colleagues,\textsuperscript{47} future work could examine the physiological pathways linking SES and amygdala/DMPFC reactivity to threat, as well as the potential physiological correlates and consequences of enhanced neural reactivity to threat. Along these lines, other health neuroscience research has demonstrated that increases in inflammation are associated with enhanced amygdala reactivity to threat,\textsuperscript{102,103} and that amygdala reactivity during a stressful task is associated with cardiovascular, HPA, and inflammatory reactivity (for a summary, see Ref. 4). However, no known work has examined a full mediation model linking SES, amygdala reactivity, and physiological activation (or, physiological activation and amygdala reactivity), which is important for establishing the health relevance of this enhanced amygdala reactivity. In addition to looking for potential physiological correlates and consequences of SES differences in neural activation during threatening experiences, it will also be interesting for future research to examine the psychological pathways that link SES and enhanced threat-related neural activity. For example, are experiences of psychological stress and negative affect, enhanced attention to others conferred by an interdependent self-construal, or facets of the...
neighborhood context (among many other possibilities) more proximal mechanisms linking low SES to greater threat reactivity? Answering questions such as these is vitally important for identifying modifiable risk factors that could be intervened upon to disrupt links between SES and neural reactivity to threat. Finally, future studies could examine the potential specificity (or lack thereof) between SES and amygdala/DMPFC reactivity to negative social versus nonsocial information. To date, all work in this area has focused on neural responses to facial expressions of negative emotion (i.e., fear and anxiety) or negative social feedback, and as such, it is unclear if low SES individuals show enhanced threat-related neural reactivity to other types of negative stimuli (e.g., prepared fear stimuli, such as snakes and spiders). It may be the case that the greater interdependent self-constructual demonstrated by lower SES individuals is associated with enhanced neural reactivity to social threats in particular (given that they could signal disruptions to intergroup cohesion), but this idea has not been tested explicitly. In sum, given a solid foundation of existing literature in this domain, there are exciting opportunities for future work on how SES influences neural reactivity in threat-related circuitry.

**SES and neural responses to reward: existing evidence**

In contrast to the relatively large literature on SES and neural responses to threat and stress, only two known studies have investigated how SES influences neural activity during reward processing. An early study in this area showed that African American individuals from lower SES backgrounds (i.e., lower education and income; both current and during childhood) demonstrated less activity in the caudate, a region often associated with reward, during the processing of positive images (compared to neutral), relative to individuals from higher SES backgrounds. While intriguing, little description of the content of the positive images was provided in this paper, and a number of participants were lost due to motion (i.e., total N = 15 for fMRI analyses), thus limiting conclusions that can be drawn from this particular study.

Following this initial paper, Gianaros and colleagues examined how parental education influences neural responses to a class of stimuli very often studied in the context of the reward-system: monetary rewards. Results revealed that lower parent education was associated with less activity in medial and lateral PFC regions, as well as pACC, to stimuli signaling monetary rewards. Lower parent education was also related to less functional connectivity between pACC and orbitofrontal cortex and less connectivity between DMPFC and ventral striatum. The authors suggest that early life SES (as measured by parental education) may influence the development of corticostriatal circuitry that is important for reward-related responding, such that less activity in these regions involved in top-down control over limbic and forebrain regions involved in reward responding could lead to maladaptive health behaviors (e.g., smoking, impulsivity, and palatable food intake) among lower SES individuals. It is interesting to note that there were no differences in reactivity of canonical reward-related structures (e.g., ventral striatum and nucleus accumbens) as a function of parent education in this study, which raises the possibility that SES has less of an impact on “bottom up” reward processing but rather more of an influence on “top down” reward-regulation.

**SES and neural responses to reward: ideas for future research**

Given the quite small current literature on how SES modulates neural responses to reward, there are very exciting possibilities for future research in this area. First and foremost, studies examining how SES influences neural responses to varied types of rewards, including social rewards (e.g., images of support figures and positive social messages), food (e.g., images of palatable foods versus healthy foods), and cigarette/drug/alcohol cues, are vitally needed. Numerous studies in social and health neuroscience have established clever paradigms for assessing neural responses to these various types of reward, and the time is ripe to explore how SES may influence reward-related activity to these tasks, in an effort to understand how corticostriatal functioning may confer both risk and resilience across the SES spectrum. Along these lines, it would be exciting for future work to use a “brain-as-predictor” approach to examine how neural responses to rewarding stimuli in the laboratory are associated with behavior in daily life. For example, are potential SES differences in neural response to food, smoking, or alcohol cues associated with greater craving for these rewards, and/or greater likelihood of
overconsumption of unhealthy food, or more cigarette smoking and alcohol use? As with threat reactivity, it will also be important to examine how neighborhood contextual factors (e.g., cigarette and alcohol advertising and availability, food deserts/availability of high quality, and affordable food options) as well as psychological factors (e.g., food insecurity and psychological stress) may mediate the associations between SES and activity in reward-related regions to a variety of positive stimuli. Finally, examining activity in reward-related circuitry in response to positive social cues may shed light on resilience factors that could buffer against negative outcomes for low SES individuals. It is increasingly appreciated that not all individuals from low SES backgrounds suffer from negative health outcomes, and the presence of a supportive individual who can serve as a mentor and provide both emotional and practical assistance during stressful periods may be particularly helpful in buffering the negative effects of low SES. Examining how reward-related responses to positive social cues and/or reminders of support figures are modulated by SES can provide additional evidence for the pathways by which mentors and other supportive individuals may decrease risk for negative health outcomes among low SES individuals. This approach could also shed light on additional ways in which a greater interdependent cultural orientation among lower SES individuals may modulate neural responses in reward circuitry in response to social rewards.

**SES and neural responses during emotion regulation: existing evidence**

To date, only two known papers have reported on the association between SES and neural responses during emotion regulation, both utilizing the same sample of participants from a longitudinal study who completed two different emotion regulation tasks. The first paper examined how SES (i.e., family income-to-needs ratio) when participants were children (i.e., age 9) influenced neural activity during a standard cognitive reappraisal task completion when participants were young adults (i.e., age 24), controlling for concurrent adult income. Results showed that lower SES during childhood was associated with diminished activity in DLPFC and VLPFC during cognitive reappraisal (versus look), as well as greater amygdala activity during reappraisal. Interestingly, mediation analyses suggested that chronic stress during childhood (measured by indicators of family turmoil and violence, as well as neighborhood noise, crowding, and housing quality) mediated the association between SES and reductions in lateral prefrontal activity in early adulthood. Thus, it appears that low SES in childhood is associated with lower prefrontal control over emotion and greater difficulty suppressing amygdala activity during cognitive reappraisal during young adulthood, perhaps via chronic stress pathways.

The second paper from this study asked a related question regarding how SES in childhood is linked with neural responses to emotion regulation in young adulthood, but focused on an “implicit emotion regulation task” that involves cognitive appraisal processes similar to affect labeling. Results from this task showed that lower SES (again measured as income-to-needs ratio) during childhood was again associated with reduced DLPFC activity during implicit emotion regulation in young adulthood. This paper also reports how exposure to an acute stressor changed neural responses during implicit emotion regulation as a function of childhood SES, showing that individuals from lower SES backgrounds showed less activity in the hippocampus during implicit emotion regulation following stress. Consistent with the prior paper, these data suggest that SES influences lateral prefrontal responses to emotion regulation across both explicit and implicit emotion regulation tasks, and that acute stress may also influence neural responses to emotion regulation differently as a function of SES.

**SES and neural responses during emotion regulation: ideas for future research**

With only two known studies examining how SES affects neural responses during emotion regulation to-date, there is much knowledge to be gained in future research in this domain. For example, it would be useful to know if SES modulates neural reactivity during emotion regulation to all types of stimuli, or if there are particular domains in which low SES may be associated with lower LPFC/greater amygdala activity during regulation tasks. Both studies conducted to-date utilized negative images to induce affect, but the content of those images, particularly if they involved social versus nonsocial scenarios, is unknown. Therefore, a future study could focus on how SES influences neural responses
Table 1. Summary of health neuroscience studies linking SES to neural activity

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>SES measure</th>
<th>Task</th>
<th>MRI analysis approach</th>
<th>Covariates</th>
<th>Main findings</th>
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</thead>
</table>
| Gianaros et al.98 | 33 undergraduate students (M age = 20, SD = 1.3; 21 females; 7 non-White | Perceived parental social standing: rankings from 9-rung “social ladders” (average of maternal and paternal) | Emotional face matching task: matching angry, surprised, and neutral facial expressions versus matching simple geometric shapes | Whole-brain, ROI (bilateral amygdala) | Sex, ethnicity, self-mastery, optimism, extraversion, agreeableness, depressive symptoms, parental education, and self-perceived social standing | Lower perceived parental social standing associated with greater amygdala reactivity to angry faces (versus neutral faces and shapes)  
Effects held after controlling for all covariates |
| Muscatell et al.89 | 22 adolescents (M age = 13.02, SD = .29; 14 females | Family SES: Composite index of household income + mother’s highest level of education | Viewing threatening (angry) emotional expressions versus fixation crosshair | Whole-brain, ROI (bilateral amygdala, DMPFC, MPFC, and precuneus/PCC) | None | Lower family SES associated with greater left amygdala reactivity + DMPFC activity to angry faces (versus fixation crosshair) |
| Javanbakht et al.90 | 52 adults (M age = 23.7; 24 females; all White | Childhood income-to-needs ratio: per capita index, adjusted annually for costs of living (US Census Bureau defines “poverty” as ratio ≤ 1.0) | Emotional face matching task: matching angry, fearful, happy, and neutral facial expressions versus matching simple geometric shapes | ROI (bilateral amygdala and mPFC), PPI (amygdala as seed) | Age, gender, and current income-to-needs | Childhood poverty associated with higher left amygdala and mPFC activity to fearful faces (versus happy faces and shapes)  
Childhood poverty associated with decreased functional connectivity between left amygdala and mPFC |
| Swartz et al.91 | 132 adolescents (longitudinal study, followed from age 11 to 19; 62 females; all White, non-Hispanic (final fMRI sample size: 87) | Household SES: parent education + income | Emotional face matching task: matching angry and fearful facial expressions versus matching geometric shapes | ROI (left amygdala) | Age at wave 1, time between waves, gender, anxiety diagnosis, family history, genotype for 5-HTTLPR, YSR affective problems scores at wave 2 | Lower family SES at wave 1 (age 11–15) associated with greater increases in DNA methylation of the proximal promoter region of the serotonin transporter gene (SLC6A4) at wave 2 (age 13–18).  
Greater change in serotonin transporter gene methylation associated with greater change in amygdala reactivity to fearful expressions (versus shapes) from wave 1 to wave 2 |
| Muscatell et al.94 | 31 young adults; (M age = 19, range = 18–22); all females; 32% Asian/Asian American, 23% Hispanic/Latina, 22% mixed/other, 13% African American, and 10% White non-Hispanic/Latina | Subjective SES: MacArthur Scale of Subjective Social Status (0–6 rung “social ladders”) | Social evaluative stress task—receiving negative, positive, and neutral feedback from an evaluator | Whole-brain, ROI (amygdala and DMPFC) | None | Lower subjective social status associated with a greater increase in inflammation (IL-6) in response to the stressor  
Lower social status associated with greater DMPFC activity to negative feedback (versus neutral)  
Neural activity in the DMPFC in response to negative feedback (versus neutral) mediated the association between SES and IL-6 |

Continued
### Table 1. Continued

<table>
<thead>
<tr>
<th>Study</th>
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<tr>
<td><strong>Studies examining SES and neural responses to reward</strong></td>
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<tr>
<td>Silverman et al. ^48^</td>
<td>15 young adults (M age = 24, range = 19–29); 9 females; all African American</td>
<td>Childhood and current SES: low versus high SES (no further information provided)</td>
<td>Viewing positive, neutral, and scrambled images, and identifying whether images are scrambled</td>
<td>Whole-brain</td>
<td>None</td>
<td>▪ Individuals from lower SES backgrounds showed less activity in the right insula, left fusiform, subgenual ACC, posterior cingulate, bilateral caudate, left pons, right hippocampus during the processing of positive images (versus neutral), relative to individuals from higher SES backgrounds</td>
</tr>
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</table>
| Gianaros et al. ^98^         | 76 adults (M age = 44.5); 41 women; 70 White, 3 African American, 1 Asian | Parental education: no college degree versus college degree | Card-guessing game: guessing the value of a playing card and winning money (positive feedback) or losing money (negative feedback) | Whole-brain, ROI (VS, DS, and frontal lobe), effective connectivity (pACC, LPPFC, DMPFC, IPC, and OFC) | Participants’ annual household income and education, a composite indicator of community-level socioeconomic position, age, sex, self-reported frequency of alcohol use, depressive symptoms, and dispositional reward responsiveness | ▪ Lower parent education associated with less activity in medial and lateral PFC regions, as well as pACC, to stimuli signaling monetary rewards  
▪ Lower parent education also related to less effective connectivity between pACC and OFC and less connectivity between DMPFC and ventral striatum |
| **Studies examining SES and neural responses during emotion regulation**                                                                                                      |
| Kim et al. ^101^            | 49 young adults (M age = 23.61, SD = 1.30, range = 20–27); 22 females (note: same sample as 105) | Childhood family income-to-needs ratio: Divided total family income by the poverty threshold | Emotion regulation task: viewing negative images and either experiencing the natural emotional state or decreasing the intensity of negative affect by using cognitive appraisal | Whole-brain, ROI (amygdala), PPI (left amygdala and VLPFC/DLPFC) | Concurrent adult income | ▪ Lower childhood SES associated with decreased activity in DLPFC and VLPFC during cognitive reappraisal (versus look), as well as greater amygdala activity during reappraisal  
▪ Lower childhood SES associated with greater positive coupling of amygdala-VLPFC; higher childhood SES associated with negative coupling of amygdala-VLPFC  
▪ Chronic stress during childhood mediated the association between SES and reductions in lateral prefrontal activity in early adulthood |

Continued
during cognitive reappraisal or implicit emotion regulation depending upon if the affect to be regulated was induced by a social or a nonsocial stimulus. Along similar lines, it would be interesting to explore if SES affects neural responses to other types of emotion regulation strategies, particularly interpersonal emotion regulation.\textsuperscript{109} Given the literature on SES and cultural orientation suggesting that individuals from lower SES backgrounds are more interdependent, an examination of neural responses during the utilization of interpersonal emotion regulation strategies seems especially warranted.

Another exciting direction for future research in this area involves exploring how SES influences neural responses during the regulation of positive affective states or in response to rewarding stimuli. A growing literature suggests that similar prefrontal circuitry seen in studies of regulation of negative emotions helps to down-regulate activity in reward-related regions like the ventral striatum during efforts to control responses to positive cues, such as palatable food\textsuperscript{83} and cigarette craving among smokers attempting to quit.\textsuperscript{110} How SES influences neural responses during more broad attempts at self-regulation beyond the affective domain is currently unknown, and future research should explore this issue. Along these same lines, it would be interesting to examine how SES-based modulation of corticostriatal circuitry during attempts at self-regulation might interact with facets of the neighborhood environment (i.e., availability of fast food, liquor stores, and tobacco advertising) to increase risk for negative health behaviors. This type of “brain by environment interaction” could contribute to SES disparities in obesity and smoking rates, and would be exciting to test empirically.

### Concluding comments

In the present review, I explored the growing literature in health neuroscience\textsuperscript{1} that has examined how SES influences neural responses to threat, to reward, and during emotion regulation. Generally, results from across the small number of studies that have been conducted in this area to date suggest that SES does affect neural function in regions that are important for health, including the amygdala and DMPFC during threat/stress, corticostriatal functioning during reward processing, and lateral prefrontal and amygdala activity during emotion regulation (Table 1). I suggest a novel conceptual model (Fig. 1) by which SES may influence brain function, and how SES modulation of neural responsivity...
may be translated into health outcomes. In particular, facets of the psychological and physical contexts associated with low SES, including greater psychological stress, an interdependent cultural orientation, and decreased opportunities for engaging in healthy behaviors, may shape the functioning of neural circuitry involved in threat, mentalizing, reward-responding, and emotion regulation. Alterations in the functionality of these neural regions, in turn, can influence physiological activation, including increasing ANS, HPA axis, and inflammatory cascades that have been linked to health, as well as engagement in negative health behaviors, including overconsumption of unhealthy, palatable food, alcohol and tobacco use, and low levels of physical activity. Over time, changes to physiological functioning and greater negative health behaviors may lead to disease morbidity and early mortality. While we are still in the fledgling stages of understanding the ways in which SES influences neural functionality, there are exciting opportunities to advance our knowledge in this area with future research, some ideas for which are suggested throughout.

It is worth noting a few additional thoughts regarding ways in which future research in this area may wish to progress. First, all of the research reviewed here is correlational (i.e., SES is examined as a correlate of brain activity during a task), making it impossible to draw conclusions about causality. To remedy this major limitation of our existing knowledge, it would be interesting for future research to conduct experimental work in which perceptions of SES or subjective social status are manipulated and subsequent changes in neural responses measured. Recent literature in social psychology has demonstrated effective ways for manipulating perceptions of SES, including asking individuals to focus on comparing themselves to those either at the top or the bottom of the SES ladder, and manipulating perceptions of economic inequality. While certainly experimental models of SES and status do not perfectly map on to the experience of being low SES outside of the laboratory, this approach may help isolate the psychological contributors to links between SES and health, and can also provide more causal evidence for the ways in which SES influences neural functioning.

A second important direction for future research concerns an issue discussed at the beginning of this review: how to measure and conceptualize SES, and the association between different SES indicators (e.g., education, income, inequality/relative deprivation, and neighborhood SES) with neural function. To date, most research in this area has used income, education, and/or subjective social status as measures of SES, leaving the relationship between other important facets of SES, including inequality and neighborhood factors, and neural responsivity largely unknown. It is also interesting to note that almost all of the studies conducted on SES and health-relevant neural function have focused on SES in childhood/adolescence. While certainly early life SES is an important predictor of later life health outcomes (and thus this focus of past research is warranted), it will be important for future research to examine how SES influences neural functioning at other developmental stages, perhaps especially during aging and later life, when health issues are likely to be especially important.

A final thought related to future work concerns the urgent need for collaborative, interdisciplinary science that brings together teams of researchers from neuroscience, social and health psychology, public health, sociology, anthropology, economics, nursing, and medicine to develop comprehensive study designs and build conceptual models that incorporate multiple levels of analysis. Without a doubt, the pathways linking SES and health are numerous, and only through “team science” can we begin to fully understand these mechanisms. The learning curve for conducting neuroimaging studies and analyzing neuroimaging data is steep, and therefore, cognitive, affective, and social neuroscientists who have expertise in these areas have a critical role to play in furthering our knowledge of how social disparities become health disparities. One idea along these lines is to partner with researchers who are conducting, large, representative, cohort-based studies of SES influences on health, and to acquire neuroimaging data from a subsample of participants from such studies. This would allow us to combine neural data together with the wealth of data collected through the larger study, thus providing rich information about SES, psychological and neighborhood factors, neural function, physiological activation, health behaviors, and health outcomes, all in the same participants. This approach will also move us closer to “population neuroscience” in which we have larger, more
representative samples in our fMRI studies, another limitation of most past research.

To conclude, there are challenges but also tremendous opportunities in studying how SES influences neural functioning relevant for health. To date, the small literature that exists in this area has been influential in informing how we think about the mechanisms linking socioeconomic factors to physical and mental health. Health neuroscience research has an important role to play in contributing to our understanding of the development of health disparities, and there are many, many exciting ideas for future research in this area. Ultimately, our knowledge of the neural mechanisms that may contribute to SES disparities in health outcomes may lead to more effective intervention and prevention efforts, so we can help to ameliorate disparities and ensure that all individuals, regardless of SES background, live healthy lives.

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Competing interests

The author declares no competing interests.

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