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Socioeconomic status (SES) is associated with health (physical and mental) and cognitive ability. Understanding and ameliorating the problems of low SES have long been goals of economics and sociology; in recent years, these have also become goals of neuroscience. However, opinion varies widely on the relevance of neuroscience to SES-related policy. The present article addresses the question of whether and how neuroscience can contribute to the development of social policy concerning poverty and the social and ethical risks inherent in trying. I argue that the neuroscience approach to SES-related policy has been both prematurely celebrated and peremptorily dismissed and that some of its possible social impacts have been viewed with excessive alarm. Neuroscience has already made modest contributions to SES-related policy, and its potential to have a more effective and beneficial influence can be expected to grow over the coming years.

**Disciplines**
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# Socioeconomic status and the brain: prospects for neuroscience-informed policy

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Abstract| Socioeconomic status (SES) is associated with both health (physical and mental) and cognitive ability. Understanding and ameliorating the problems of low SES have long been goals of economics and sociology; in recent years, these have also become goals of neuroscience. However, opinion varies widely on the relevance of neuroscience to SES-related policy. The present article addresses the question of whether and how neuroscience can contribute to the development of social policy concerning poverty, and the social and ethical risks inherent in trying. I argue that the neuroscience approach to SES-related policy has been both prematurely celebrated and peremptorily dismissed, and that some of its possible social impacts have been viewed with excessive alarm. Neuroscience has already made modest contributions to SES-related policy and its potential to have a more effective and beneficial influence can be expected to grow over the coming years.

## [H1] **Introduction**

Poverty exists throughout the world and its prevalence in even affluent societies may be surprising to those unfamiliar with the statistics. In the US, for example, measured relative to the federal poverty line (an income corresponding to $25,100 to support a family of four[1]), 12.7% of the public live below the poverty line and 29.8% live on less than twice that amount[2]. These percentages are higher for children, with 18.0% and 39.1% living in poor and low-income families, respectively. Different countries use different definitions of poverty and direct comparisons are further complicated by many other social and economic differences between countries; however, recent surveys show that poverty by any reasonable definition is common across the globe[3,4,5]. Poverty reduction ranks among the top goals of many governmental and multilateral organizations, for compelling reasons: in addition to the obvious deprivations related to food, shelter and other basic needs, social science research shows that poverty is associated with shorter and less healthy lives, higher rates of mental illness and lower cognitive ability (Fig. 1a-c).

It is not only the poor who are afflicted by these physical and mental ills. Although risk for these outcomes is most concentrated among this group, it is present to a lesser degree for the near-poor and declines gradually as income rises[6]**.** Indeed, the recognition of these graded effects was a pivotal development in health disparities research, and in 1994 Adler and colleagues called on the field to confront "the challenge of the gradient"[6]. With the emphasis on understanding the gradient came a broadened conception of the factors that distinguish the poor from the affluent. In addition to income and other economic factors, social factors such as educational attainment and occupational status were found to cluster together with income and wealth, forming a construct known as socioeconomic status (SES).

There is obvious mutual relevance between research on poverty and research on SES and the distinction between these concepts is not always prominently marked in discussions of such research. Furthermore, much of the literature considered in this article lumps together economic and noneconomic measures of SES and the range of deprivation varies across studies. In this Perspective, I will therefore adopt a 'lumping' rather than 'splitting' approach to SES and poverty but note that more precisely characterizing these distinctions would be very worthwhile, especially with respect to policy[7,8].

Only recently have neuroscientists sought to understand SES. As shown in Fig. 1d, the neuroscience of SES emerged in the 21$^{st}$ century and has grown rapidly in recent years. The existing literature is primarily focused on elucidating the neural correlates of SES, their causes and their consequences for people's lives. Many of the consequences of SES noted earlier and illustrated in Fig. 1 have links to the brain. Mental health and cognitive ability are obviously related to neural processes. Less obvious, but empirically well-established, is the role of the brain in vulnerability to physical illnesses such as heart disease, stroke, diabetes, arthritis and cancer, through its role in transducing and regulating stress and the endocrine and immune responses that ensue[9,10].

Large organizations with poverty reduction agendas, such as the World Bank[11] and UNICEF[12] have taken note of the neuroscience of SES. The most recent installments in a series on child development in low- and middle-income countries published by The Lancet make extensive reference to neuroscience and brain development[13,14,15]. In the UK and US, neuroscience has been brought to bear on child policy: in the UK, in two influential reports to the British government[16,17] and, in the US, in the writings of

researchers affiliated with Harvard's Center on the Developing Child[18] (see Box 1). Neuroscience has even been embraced as a source of actionable poverty policy guidance concerning effective teaching[19] and economic betterment[20]. The hope that neuroscience will be of immediate, practical use against social problems is widely expressed in the popular press: for example, in 2016 Newsweek published an article on poverty[21] that proclaimed "neuroscience has now linked the environment, behavior and brain activity—and that could lead to a stunning overhaul of both educational and social policies." However, not all commentators have been so enthusiastic. The potential to use neuroscience in poverty policy has been criticized by some as unrealistic (e.g.[22,23,24]) and by others as laden with value judgements (e.g. [25,26]) or as a dangerous diversion of attention away from social and economic injustice (e.g. [27]).

The goal of this Perspective is to assess the promise of neuroscience as a source of guidance on poverty policy. Before considering how and whether the neuroscience of SES holds practical promise, a short overview of the science will be provided. I will then consider whether it can help us understand how SES comes to be associated with so many important life outcomes and whether it can now, or in the foreseeable future, provide specific, actionable policy guidance. Finally, I will ask whether framing socioeconomic disparities in terms of brain science increases our willingness to help the poor or imposes particular social values on them.

[H1] **The neuroscience of SES**

Less than a decade ago it was possible to provide a complete accounting of the literature on SES and the brain in 7 pages[28]. With the field's rapid growth, the state of the science is now challenging to present concisely; however, several recent reviews have aimed to do so (e.g. [29,30,31]).

Socioeconomic disparities in cognitive and emotional functions, from infancy through to old age, are being investigated using the tools of neuroscience. The literature includes studies using electroencephalography (EEG), event-related potentials (ERP) and structural and functional MRI[29,30,31]. These studies have aimed to characterize differences across levels of SES and to relate these differences to their potential causes as well as to differences in behavior, ability and wellbeing. Animal research has also been brought to bear on questions of causation, by testing hypotheses about the effects of factors that are correlated with SES in humans and the pathways through which SES differences become associated with brain differences[9,32].

It has become increasingly clear that different life adversities affect brain development and function through at least partly different mechanisms. The effects of institutionalization are probably not the same as those of neglect[33]. Similarly, socioeconomic disadvantage (which itself is multifactorial and somewhat variable from context to context) is distinct from other risk factors, such as abuse and neglect[34]. Many adversities tend to co-occur but do not necessarily affect the brain in the same ways. Sheridan and McLaughlin[34] have distinguished between the impacts of deprivation and threat, which presumably operate in different proportions in poverty and maltreatment, on different neural correlates[35]. Thus, findings on the effects of 'adverse childhood experiences'[36] or 'cumulative stress'[37], which are the subjects of larger and more established literatures, should not be assumed to generalize to poverty or low SES.

Rather than attempting to summarize our accumulating knowledge about SES and the brain, I will here offer selected examples of the kinds of questions that have been asked, along with studies aimed at answering them (see [29] for a recent review). These examples were selected to illustrate the wide range of methods and samples used and to have relevance to different aspects of cognition and emotion.

[H2]*The neural correlates of socioeconomic status*. One kind of question is a simple, descriptive one: does SES have measurable neural correlates and, if so, what features of the brain are correlated with SES? This question has been asked in studies of brain structure and brain function in children and adults, most often focusing on regional differences but increasingly also on networks. For example, by analyzing the structural MRIs of children and youth, a study by Noble and colleagues identified regionally-specific differences in cortical surface area as a function of both family income and parental education, with covariates including genetic ancestry[38]. When controlling for education and other covariates, significant effects of income on surface area remained in bilateral inferior frontal, cingulate, insula and inferior temporal regions and in the right superior frontal cortex and precuneus. Furthermore, the relationship between surface area and SES was shown to be strongest at the lowest SES levels; SES had a positive relationship with surface area at all levels of income and education, but the difference between poverty and near-poverty mattered most.

Questions about qualitative differences in neural structure and processing have also begun to be addressed. On the one hand, SES might simply increase or decrease some aspect of the brain in tandem

with increasing and decreasing measures of ability or health. On the other, it might moderate the relationship between brain and behavior such that individuals with higher and lower SES use their brains in different ways to perform the same tasks. As an example of findings supporting the latter possibility, a study that examined the neural correlates of children's arithmetic processing found that SES moderated the relationship between behavior and brain activation: in children from higher SES homes, the activity of regions associated with verbal performance (including the left middle temporal gyrus) tracked mathematical ability, whereas in children from lower SES homes ability was more closely related to regions associated with spatial processing (including the right intraparietal sulcus)[39].

Another type of question addressed by neuroscientists studying SES concerns the psychological significance of SES-brain relationships: that is, whether the neural correlates are epiphenomenal or whether they account for at least some of the socioeconomic disparities in cognitive or emotional psychological measures. In many cases the relationship of SES to brain structure or activity either partly or fully accounts for the relationship of SES to psychological measures of interest. For example, one study performed structural imaging in a large sample of healthy young adults and assessed a set of personality traits linked to depression[40]. This revealed that the relationship between family SES and depression-related traits was partially accounted for by the volume of the medial prefrontal and the anterior cingulate cortex (ACC). Other studies have produced similar findings; for example, the study of SES differences in cortical surface area, mentioned above, also found that these differences could account for socioeconomic differences in cognitive outcomes[38].

[H2] *Mechanisms linking socioeconomic status to brain function*. Ultimately, the most important questions about SES and the brain concern mechanisms, and these are questions we are only beginning to ask, let alone answer. How does SES become associated with brain structure and function? Decades of debate in the social sciences regarding the psychological correlates of SES have shown that even the direction of causality cannot be taken for granted (Box 2).

Mechanistic questions about SES are difficult to answer in part because SES is a distal factor. Income, education and other dimensions of SES are indices of risk but do not themselves directly impinge on the child or adult brain[41]. Rather, they are related to other, more proximal, factors, which have causal roles. These proximal factors include nutrition, toxin exposure, prenatal health, cognitive stimulation

(including linguistic interaction), stress, parenting behavior (particularly attentiveness and warmth) and the possible genetic differences associated with SES[30,42,43,44].

A growing number of studies in SES neuroscience have measured one or more candidate mediating factors and tested whether they can account, statistically, for some or all of the relationship between SES and brain structure or activity. For example, it was shown that mother-reported life stress and quality of parenting behavior (based on videotaped interactions between parent and child) together fully mediated the relationship between SES and hippocampal volume in children[45]. This is consistent with differences in stress and parenting practices being the proximal causes of this particular neural correlate of SES. Other human neuroscience studies have tested hypotheses concerning the causal factors linking SES and brain structure and function, and animal studies of specific proximal causes such as stress and other SES-correlated environmental factors can also be brought to bear (Box 3).

Of course, identifying proximal physical and psychosocial factors gives us only a partial mechanistic understanding of SES and the brain. These factors are transduced by cellular and molecular processes, of which we have some general knowledge but not a well worked-out understanding in relation to SES. An exemplary step toward filling in such mechanisms was taken in a longitudinal study of stress-related methylation of the serotonin transporter gene in adolescents[46]. Across two time-points, teenagers with lower SES showed a greater increase in methylation than their higher SES peers and this was associated with greater amygdala reactivity and more depression symptoms.

As the field learns more, questions about mechanisms are becoming more nuanced. For example, by what mechanisms of brain development does SES correlate with brain structure and function during different phases of childhood and adulthood and by what additional mechanisms of brain aging does SES manifest itself in the brains of older adults? Studying the relationship between SES and cortical thickness in children, an earlier and steeper decline among low SES participants was found[47]. The authors of this study suggested that this may be due to interactions between SES-linked differences in cognitive and linguistic stimulation and processes of synaptic pruning and myelination, which are responsive to experience and contribute to the thinning of the cortex. Additionally, they point to evidence that early life stress can accelerate brain development, potentially resulting in precocious thinning and, ultimately, the closing of sensitive periods for environmental influence[48]. In older individuals, who normally show cortical thinning, declines in white matter integrity and hippocampal volume loss, these effects are

magnified by low SES[49,50,51]. Given the multifactorial nature of SES and the manifold nature of brain development and function, it seems likely that the neural correlates of SES emerge through many different mechanisms, operating at different ages and being responsible for different aspects of brain structure and function.

[H1] **Understanding SES disparities**

Can neuroscience contribute in any substantive way to our understanding of poverty and its accompanying disadvantages? Opinion is divided. Early efforts to integrate neuroscience with larger social issues surrounding childhood poverty evoked great enthusiasm at the time, but were also criticized for failing to connect the neuroscience to the social issues (Box 4)

One advantage of the neuroscience approach to understanding socioeconomic disparities is evident even at this early stage: neural measures can reveal differences between higher and lower SES individuals that are not apparent in more traditional, behavioral measures. This is true even though the disparities we seek to understand are psychological and therefore more typically measured by behaviors such as task performance or survey responses. For example, in several studies ERPs have revealed SES disparities in the degree to which children filter out irrelevant sounds when paying attention in a dichotic listening task[51,52,53]. None of these studies found a significant SES-related difference in performance, even when performance was below ceiling, indicating that ERPs show greater sensitivity in these studies to disparities in attention compared to the concurrently collected behavior. The value of such measures is that a behavioral effect that is too small to be observed in a single laboratory testing session might nevertheless matter. It could have cumulative effects over time or larger effects in real world contexts. In other words, a larger sample of behavior or a sample from different tasks might detect disparities with as much or more sensitivity than the ERP methods used in these studies. No in-principle superiority of neural measures over behavioral measures is being claimed here, merely the empirical observation that for some purposes, neural activity predicts outcomes better than traditionally used measures of behavior[55].

Another important advantage of neural measures in the study of SES disparities is their ability to reveal qualitative differences in brain as a function of SES, not just more or less brain activity, volume or cortical thickness, but different patterns of the brain measure in question. Behaviorally measured SES

disparities generally take the form of a positive correlation between SES and task performance, which is a simple quantitative relationship. The neural disparities follow the same trend in some cases, but in others the neural differences appear to be qualitative. In the study of children's arithmetic ability mentioned above, for example, SES moderated the brain-behavior relationship, indicating that higher and lower SES children used different neurocognitive systems in performing the task, independent of level of performance[39]. As with the greater sensitivity of neural measures, the ability to reveal qualitative differences is not an intrinsic superiority of neural over behavioral approaches. In the example above, one can imagine behavioral research designs, such as a selective interference paradigm with verbal and visuospatial interfering tasks, that could lead to the same conclusion. Nevertheless, the insight about qualitative differences in arithmetical processing arose directly from the multivariate nature of brain imaging, which can characterize processes in terms of different brain regions as well as degrees of activation.

The final, and most distinctive, advantage of using the concepts and methods of neuroscience to understand SES is that some of its relationships to psychological or behavioral outcomes may be, at root, neurobiological. This point can be made most clearly by beginning with an example of a psychological phenomenon related to SES that can be understood without recourse to neuroscience. The SES gradient in performance on standardized school achievement tests is such an example, in that we can at least aspire to explain it in terms of school quality, available role models for academic achievement and many other factors whose relations to SES and test scores can be couched in the language of 'belief-desire' psychology[56]. Although there are also proposed neural mediators of the SES school achievement relationship[57,58], at present we do not know whether they add insight or predictive power beyond the psychological explanations**.**

By contrast, some psychological phenomena may result from aspects of brain development and function that can be explained only in terms of biological facts. If described in brain-free terms, such phenomena will seem inexplicable, whereas when neural implementation is considered they will make sense in terms of a wider fabric of explanation. One category of examples concerns obviously biological factors with neural impact that are likely to have roles in explaining SES disparities in behavior and psychology. On the environmental side, these include prenatal and postnatal nutritional deficiencies and SES-linked exposures to environmental toxins[59]. They also include the synergisms of such factors with one another and with ostensibly nonphysical socioeconomic factors such as parental education[60]. Finally, those

expecting a role for genes in explaining SES disparities need not dig very deep to find fundamentally neurobiological mechanisms[61].

Even when SES disparities can be described in purely psychological terms, the mechanisms underlying them may be irreducibly neural. For example, the psychological stresses of low SES have long been known to be related to the higher incidence of depression symptoms in individuals at low levels of SES[62]. This is particularly true of stress experienced early in life, which raises the risk of depression throughout the lifespan[63]. Why is this? Reasoning on the basis of psychology alone, it is not apparent why the stress of low SES early in life would render someone more susceptible to feelings of depression later (as opposed to less susceptible or equally susceptible). Current research favors an explanation involving stress mediated disruptions to the development of prefrontal cortex, the hippocampus, amygdala and reward system structures that are needed for the regulation of mood and stress response throughout life[64]. The pathways through which early-life stress (not SES per se) affects the development and function of these areas are now understood with greater cellular and molecular detail based on studies in animals and humans[9,65]. Such studies have also highlighted the moderating role of parental care in buffering the developing brain from the effects of stress[66] and such care has also been found to impact the effects of SES on hippocampal volume in humans[45,67]. The study methylation of the serotonin transporter gene in adolescents mentioned above is another example of the explanatory advantage that comes from neurobiological accounts of psychological phenomena[46].

Thus, although neuroscience research on the mechanisms of SES disparities is nascent, early findings demonstrate the 'explanatory value added' by neuroscience even for SES disparities in purely psychological traits.

[H1] **From knowledge to intervention**

Beyond its benefits to the scientific understanding of SES and the association of SES with myriad life outcomes, does the neuroscience of SES have real-world policy implications? Is it ready to guide policy, in the sense of counting in favor or against specific programs or practices? Here I use the term 'policy' to include not only governmental programs, but also official practice recommendations by professional groups (such as health professionals, educators, law enforcement, legal and human resource

professionals) and unofficial but nevertheless institutionalized practices involved in professional education and norms of professional practice.

[H2] *Current contributions of neuroscience to policy*. One could argue that neuroscience can already guide policy, insofar as it provides additional converging evidence in favor of approaches already indicated by behavioral research. Examples include programs aimed at reducing child and parent stress and at increasing parenting skills[68]. This is certainly not the "stunning overhaul" heralded by *Newsweek*, but it is nevertheless a contribution. In policy-making, it is not only new and unprecedented ideas that matter; additional support for a familiar idea can tip the balance in decision-making and therefore be consequential.

This approach may seem odd to those whose understanding of science is based on critical experiments designed to pit hypotheses against one another to see which one survives the attempt to falsify it. If we already know something with a high degree of certainty, what is the value of showing it in a new way? In answer, one can point first to the many ways in which null hypothesis significance testing (NHST) can mislead[69]. Second, there are broader issues of validity, including construct validity and external validity, which are particular challenges in the study of complex social and biological systems and which limit the generality of conclusions that can be drawn from any single method. There are generally irreducible gaps in the applicability of knowledge from any one study or method to the real world. As different methodologies tend to have different strengths and weaknesses, converging evidence across methods has value.

The approach typically used for policy decision-making focuses on the 'weight of evidence' (WOE). In comparison to NHST, WOE is a less formalized approach to answering empirical questions related to policy and typically involves an intuitive synthesis of diverse evidence, including qualitative and quantitative data, the results of observational and experimental studies with humans or animals and even the results of in vitro studies[70]. As an example, we can consider a hypothetical new policy concerning pediatricians' interactions with parents that suggests that pediatricians routinely ask parents about their stress levels and the methods they use to manage stress, and direct parents to therapy and support when stress is high or inadequately managed. It seems obvious that parental stress is not good for children and psychology research has already provided behavioral evidence that stressed parents have more troubled children[71]. However, given the costs of adding new procedures and responsibilities

to pediatric practice, a singular source of evidence for the benefits might not suffice to support such an intervention. The addition of new types of evidence from human and animal neuroscience studies could therefore add to the WOE supporting the value of addressing parental stress in pediatric primary care practice.

Do the policy implications of neuroscience go beyond merely adding confirmation to our existing understanding of policy costs and benefits? At present, I believe the answer is no; however, given the short history of SES neuroscience and its rapid growth, this may soon change. Although innovation is, by nature, hard to predict, I have below tried to imagine some actionable neuroscience advances that illustrate the variety of ways in which neuroscience might eventually inform policy.

[H2] *Prospects for the future: actionable insights from neuroscience* As Pavlakis and colleagues have noted, a likely near-term application of neuroscience to policy is the use of measures of brain structure and function as biomarkers[72]. Projecting modestly from current scientific knowledge, it can be imagined that such measures could indicate risk for future cognitive and educational problems of the kind faced by children of low SES. This approach capitalizes on the properties of neural measures discussed earlier: these measures (which include relatively inexpensive and portable EEG-based measures) hold promise as more sensitive predictors than behavioral data. Such biomarkers would be particularly valuable where preverbal infants and young children are concerned. To the extent that interventions may be most effective for young children, this advantage of predictive biomarkers is all the more importantant[72].

In addition to their use in clinical or educational practice, such biomarkers would facilitate research on the efficacy of interventions. Analogous to the use of biomarkers as selection criteria or endpoint measures in clinical trials targeting preclinical Alzheimer's disease[73], appropriately validated biomarkers of SES disparities might serve as proxies and harbingers of later behavioral outcomes. Along these lines, interventions designed to help disadvantaged individuals have been studied with the help of biomarkers[74,75]. Proposed future intervention studies[76] will incorporate EEG outcome measures and, in one ambitious plan[77] to study and enhance child development among the poor of Bangladesh, will also incorporate functional near-infrared spectroscopy (fNIRS) measures of brain activity.

A related policy benefit of neural measures comes from their ability to reveal qualitative differences in psychological processes as a function of SES. As noted above, studies have demonstrated different ways of performing arithmetic in children of higher and lower SES[39]. An accumulation of such findings would have implications for education policy. Specifically, in academic domains showing SES differences such findings would indicate that we should not assume that teaching methods that have been validated with one SES group will necessarily be effective for a different group.

Research in developmental neurobiology may also deliver actionable insights in the foreseeable future. Consider prenatal brain development, which lays the groundwork for lifelong cognitive ability and emotional wellbeing. Although reducing stress, toxin exposure and nutritional deficiencies are good goals for any stage of life, the brevity of the prenatal period and our growing knowledge of the specific windows of prenatal time when particular interventions could help makes policies promoting prenatal neurodevelopment seem especially feasible and impactful[78]. For example, maternal SES has been shown to be related to prenatal stress-immune system interactions, which were in turn related to infant brain development in the first year of life[79]. Furthermore, early, but not late, pregnancy cortisol levels have been demonstrated to affect amygdala volume and child behavior at age 7[80].

Concrete recommendations based on cellular mechanisms of brain development during childhood can also be envisioned. For example, the possibility (described above) that low SES might lead to a premature reduction of plasticity and consequent reduction of opportunities to learn[47], if supported by additional research, would recommend that we seek potentially modifiable causes of this environmentally-driven precocity. These would plausibly include diet, endocrine disrupters and aspects of the psychosocial environment[48,81]. A surprising array of other influences on the timing of brain development, from neuromodulators governing the opening and closing of critical periods[82] to the gut microbiome[83,84], offer additional potential avenues of intervention.

Finally, specific epigenetic changes associated with SES have been identified during prenatal and postnatal life[46,85,86,87]. An animal model of early life stress and parenting has been developed[88], which specifies the pathways linking environmental stimuli to gene expression in the brain through a detailed molecular pathway. When and if we understand SES-linked epigenetic changes and their consequences for brain and behavior in comparable detail, we will be in possession of a wealth of potentially

actionable knowledge. This includes intervention targets in the environment or in combination with pharmacological therapies, as has been proposed for other indications[89,90].

By considering examples of policy actions that could conceivably follow from neuroscience in the future, we have entered the realm of science fiction. None of the possibilities sketched out above are feasible at present and some may await decades of scientific progress before they can finally be judged as viable, laughable, or something in between. However, this exercise in imagination serves a purpose: to illustrate the variety of ways in which advances in the neuroscience of SES could, in principle, lead to specific policy recommendations and the range of forms that such recommendations could take.

[H1] **Morality and politics**

Scientific theories can be controversial, and applications of science to real world human problems even more so. In the case of neuroscience and policy related to SES, the disagreements are not just about what's true and what works; they are also about ways of viewing morally significant problems[91]. For this reason, contemporary policy discussions invoking neuroscience, such as those lead by the World Bank[11] or the Center on the Developing Child, are viewed as dangerous by some. To the extent that neuroscience discourse emphasizes certain aspects of social problems and their solutions, it can exert a powerful influence on public opinion and policy design.

The potential dangers of neuroscience discourse in this context have been discussed by sociologists and historians of science working within a discipline known as 'critical neuroscience'[92]. The term critical is used in this field in a non-derogatory sense to mean reflective, analytic and interpretative. These scholars have not, by and large, found fault with the science being cited in policy discussions. Rather, they have noted that certain values, allocations of responsibility and policy goals seem implicit in the framing of socioeconomic issues in terms of neuroscience. Below I summarize four of these critiques. In each case I attempt to convey what I consider the valid concerns raised by the critiques as well as the ways in which they may overstate the problems.

[H2*] Responsibility and blame*. It has been suggested that neuroscience presents us with a description of a malfunctioning biological system rather than a morally wrong social arrangement. According to one critic, instead of confronting "issues of equity, power, and justice [we instead focus on] the impact of the

'environment' on brain function"[93]. Other critics have likened the neuroscience approach to "saying slavery is ... morally wrong because it impacts brains" and suggested that the focus on brains has implications for policy in that it eclipses social and economic policy targets, "effectively work[ing] to conceal the social forces—both the actual poverty suffered by people and the systemic effects and politics of inequality—from view"[27].

I would agree that the neuroscience approach does draw more attention to the brain than to social structures and justice, and that this could lead to the neglect of promising policy targets in society. For example, the neuroscience-inspired policy literature has much to say about parenting practices in low SES communities and parent training is a frequent policy recommendation[94]. However, it is important to consider the context in which parents are caring for their children. Food insecurity, the threat of homelessness and other stresses of poverty may well lead mothers to be preoccupied or depressively withdrawn and, as a consequence, they may behave toward their children in less than beneficial ways. Neuroscience-based arguments on the importance of early maternal responsiveness could divert attention from interventions targeting nutrition, housing and other needs, in favor of parent training programs. Such arguments could even be misunderstood as pointing the finger of blame at mothers of low SES.

[H2] *Essentializing low SES.* Another concern is that neuroscience leads us to view low SES as a property of person or brain, rather than a situation, "re-inscribing social and economic differences into differences in brain architecture"[95]. Attributing the life failures of poor adults to epigenetic and other environmental effects on their brains may seem kinder than blaming them for laziness or labeling them hopeless because of their genes; however, as has been pointed out, it nevertheless categorizes them as biologically inferior[96]. It has been suggested that there is a small step from this to stigmatizing or (given the biomedical framing of neuroscience) pathologizing the poor[27].

It is my belief that we should neither essentialize poverty, viewing it as a property of certain people's brains, nor stipulate that poverty can only be understood in terms of the extra-personal properties of society. In trying to understand people in their contexts, ignoring the person is as unenlightening as ignoring the context. Yes, it feels bad to talk about the causal contributions that suffering people make to their own suffering, even if those contributions are understandable and inadvertent. But if we cut short our analyses of the chains of influence at the point that those influences cross the boundary between world and person, we are imposing a large blind spot on our view of a complex causal network.

[H2] *Values*. A third criticism concerns the values embodied in neuroscience-based policy. One critic notes that "the discourse of brain science affirms middle-class values/lifestyles by invoking cultural practices and preferences specific to this group in the discourse's explication of the 'ideal' environment for 'stimulating' neurological development"[25]. One education leader points out that the ways in which low SES students differ from other students are automatically labelled deficits and states "I am deeply concerned today that work in what I will refer to as applied neuroscience is making deficit claims with regard to language learning and executive control among populations of young people living in poverty"[97]. She suggests that there is more than one kind of good development and that developmental goals may vary across cultures and communities.

Others perceive, in neuroscience-based policy discussions, the neoliberal economic values of human capital theory[98]. According this theory, economic development requires more than raw materials and money; it also requires people who are productive members of society — the 'human capital' of healthy, skilled and disciplined workers. Self-control and future-mindedness are traditional middle-class values that are also important forms of human capital, and they figure prominently in research on executive function development and SES[99]. Policies arising from this work may seem to place more value on raising productive workers than on human wellbeing. One critic writes of the "entrepreneurial forms of self-governance" prioritized by neuroscience, and suggests that the goal is "babies emotionally primed to navigate an economic system that prioritizes flexible, mobile, and adaptable workers"[26]. These concerns shade into worries about social control, with another critic seeing "the discourse and practices of brain science extend[ing] and legitimiz[ing] the extension of Foucauldian governmentality over lower income populations, which are perceived as threatening social and state security"[25].

I would agree that the case for increased government support for low-income families is often framed in terms of economic development and social order. For example, the neuroscience-inspired Allen report in the UK summarized the benefits of early childhood intervention as "improvements in behaviour, reduction in violent crime, higher educational attainment, better employment opportunities and more responsible parenting of the next generation"[16]. And these were the noneconomic benefits, presented before the report's economic case for investment in early childhood! Creating more productive and law-abiding individuals is certainly in the state's interest; however, it does not follow that these efforts deserve the negative connotations of social control. Furthermore, executive function can facilitate achievement of one's personal goals, as well as, or even in opposition to, the goals of the state. There is

no necessary affinity between neuroscience and neoliberal ideals of humanity; neuroscience can in principle illuminate any aspect of human psychology and behavior.

[H2] *Fatalism*. The brain's critical or sensitive periods are frequently mentioned in the policy literature, and have been used to emphasize the importance of supporting early childhood development. However, the emphasis on prenatal and early postnatal development has led critics to warn of fatalism, which could lead us to write off the needs of older children and adults. In the words of one author, "If young brains subjected to deprived conditions, and to the inadequate parenting that often goes along with them, are irrevocably damaged – pickled in stress hormones, stripped of synapses – there is no time to waste, that is true. Yet such alarm, though it conveys urgency, can all too easily fuel defeatism... the case for subsequent help is bound to seem weaker."[100].

In response, I would agree that a strong emphasis on critical or sensitive periods does indeed discourage efforts to help older children, teens and adults, given fixed resources. Of course, neuroscience is not alone in suggesting that earlier interventions will generally be more effective than later interventions; for example, economists have reached the same policy conclusion[101]. We may well feel uncomfortable with unequal distribution of help, such as the prioritizing of 1 year-olds over equally needy 10 year-olds or 60 year-olds. However, even if we recognize a moral obligation to help all needy people, the expected effectiveness of help at different ages is surely relevant to our decisions about resource distribution. Finally, it must be clarified that contemporary neuroscience does not support sharply defined critical periods for psychological development, and has only begun to understand age-related changes in plasticity[102]. Indeed, one of the most important insights of recent decades in neuroscience is the degree of plasticity that remains present in the adult brain[103].

[H1] **Conclusions**

The neuroscience of SES is a young field (Fig. 1d). Many of the questions and controversies discussed in this Perspective can be traced to its fledgling status. This is certainly true of the scientific criticism that neuroscience knows too little about SES to be of use. It may even be true of the social, political and moral criticisms reviewed above. My own experience working on the neuroscience of SES has taught me that ideological criticisms are more likely to fasten onto schematic proposals and 'in principle' ideas than onto real empirical work in progress. Nevertheless, the social criticisms reviewed here raise worthwhile

considerations. The models and metaphors of science can implicitly influence policy choices in good or bad ways and, for that matter, existing policy preferences may bias the inferences we make from the science. These are reasons to de-silo and seek closer collaboration between neuroscientists and social scientists engaged in critical neuroscience[104].

Scientifically, we have only scratched the surface of the SES–brain function relationship, and many questions remain open. Which findings will replicate and generalize, and which will not? What can we say with confidence about the mechanisms linking SES and the brain? To what extent do the answers to these questions depend on specific dimensions of SES, such as income or neighborhood characteristics, or on poverty per se as opposed to gradations between higher levels of SES? Are individuals' ages, genders and genotypes part of the answer? Do the same mechanisms underlie SES disparities globally; that is, do they apply in middle- and low-income countries as well as in the high-income countries in which most of the research has been conducted? Do they vary across cultures or ethnicities, or between urban and rural communities? There is little that we can now say with confidence. This is particularly true when we remember that findings on adverse experience more generally (including trauma, maltreatment and institutionalization) cannot be applied automatically to the understanding of socioeconomic adversity. Thus, our knowledge of SES and the brain remains quite limited. If we fail to appreciate the preliminary state of our knowledge, we risk the field's credibility by promising too much and disappointing policy-makers and funders. We also risk premature translation of research findings into policy, which could harm the very people in need of help.

Will neuroscience research on SES advance to a state of practical usefulness given time? In one critic's view, "Neuroscience has little or nothing to contribute to addressing these problems [of low SES] and is unlikely to add anything of significance in the future"[24]. I believe that one reason to doubt this prediction is the multitude of ways that basic and clinical neuroscience have already found to shape brain development and function, with no obvious barriers to their eventual application to the problems of poverty. Another reason to reject pessimism at this point is the very short time that neuroscientists have been studying SES. Depth of understanding and applicability take decades to emerge in any area of neuroscience and, in the case of SES, the work is all quite recent and being pursued in relatively few laboratories. Nevertheless, our current knowledge is sufficient to frame new scientific hypotheses to be tested, boding well for the coming decade of basic research on SES and the brain.

In sum, at this early stage of development, neuroscience for poverty policy is positioned between enthusiasts and critics. In my view, enthusiasts often fail to recognize the scientific challenges ahead. whereas critics either dismiss the whole enterprise as unrealistic, based on early results from a nascent field, or see it as socially dangerous, based on the possibility that it could be used to justify harmful policies. Thus surrounded, neuroscientists studying SES will need to manage expectations in the coming years and, at the same time, assert the potential of this science for a positive impact on society.

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Box 1 Simplifying models, metaphors and messages

Harvard University's Center on the Developing Child has been a leader in the integration of ideas and insights from neuroscience with child policy (e.g [18]) and has introduced a number of influential

neuroscience-based concepts into public discourse on early childhood development. The director of the Center and his collaborators noted that public resistance to support for some programs might stem in part from preconceptions about human development and poverty more specifically[105]**.** To address this issue, they partnered with the FrameWorks Institute, a communications research firm that "develops simple and concrete metaphors that help people to organize information on issues in new ways, to fill in understanding currently missing from the public's repertoire, and to shift attention away from the unproductive patterns they default to in understanding those issues." To do so, the FrameWorks Institute "identifies, empirically tests, and refines explanatory metaphors for complex social problems".

Together the Center on the Developing Child and the Frameworks Institute developed a 'core story' of early childhood development. The story features neuroscience prominently in the form of what they term 'simplifying models' or 'metaphors': these include 'brain architecture' and 'toxic stress'[105]. This framing highlights the psychological needs of young children, even preverbal infants who might seem to need little more than food and shelter, by emphasizing the foundational role of early experience in building brain architecture. It also counteracts common assumptions about personal responsibility and the causes of poverty by emphasizing the "toxic stress" that accompanies adversities such as poverty and the effects of such stress on parents and children.

The idea that socioeconomic disadvantage has physical consequences may impress upon laypeople and policymakers the seriousness of poverty's effects and the benefits of prevention or intervention. Indeed, the effectiveness of this approach is illustrated by the comments of a focus group participant who, when presented with the physiological consequences of severe stress that might accompany deep poverty stated "...what really gets me from the study is that it could actually have a chemical or biological or some sort of impact on the child's brain... Behavior is one thing, and attitude and personality is one thing, but if it can really negatively impact... the chemistry and the makeup of the brain—you can damage that that early—that's really serious"[105].

Those sensitive to neuroscience over-reach might view the Center's messages as implying a more detailed scientific understanding of human brain development in real world contexts than we currently have[23]. However, although the Center has made an unusual use of science for messaging, in my opinion it is not a misuse. Indeed, a number of eminent neuroscientists have worked with the Center on the Developing Child to ensure that the message, while simple and metaphorical, is not oversimplified or misleading.

Box 2 Correlation and causation

Socioeconomic status (SES) is correlated with intellectual achievement and mental health[106,107], but what pattern of causality underlies this correlation? Social scientists have long debated the direction of causality between SES and various behavioral traits[108] and their answers fall into two broad categories.

According to the theory of social causation, SES causes differences in cognition and emotion though environmental influences on the person[108]. In other words, SES causes its psychological correlates. Given the importance of cognitive and emotional development for academic and occupational success and emotional wellbeing, this would create a vicious cycle: a family's poverty would causally impact the capacities needed for socioeconomic success in the next generation.

According to the theory of social selection, however, psychological differences that are under genetic control cause SES differences through lowered educational and occupational performance[108]. In other words, psychological differences cause SES differences. Genetic transmission of psychological traits within the family cause children to develop the behavioral phenotypes associated with their family's SES, which would explain the intergenerational stability of SES. Social scientists who hold a belief in social selection tend also to be skeptical about the effectiveness of interventions[109]: indeed a recent critique of the neuroscience of poverty questioned its practical utility, given the possibility that social selection is responsible for the neural correlates of SES[24]. Whereas harmful environmental effects can in principle be prevented by policies that eliminate the specific harms from the environment, it is less clear how to correct detrimental effects of genetic origin (although critics do note that some genetic diseases can be treated).

In the relationship between SES and the brain, it seems likely that selection and causation both operate. It is hard to imagine how innately higher or lower abilities or greater or lesser emotional resilience would not encourage upward or downward drift in SES over a lifetime. Indeed, this

has been referred to as 'Herrnstein's syllogism' for its seeming inevitability[110]. On the other hand, there is evidence from social science that SES causes at least some of its psychological correlates.

The strongest evidence for social causation comes from 'natural experiments', in which SES is raised or lowered for reasons external to the subjects themselves[111]. For example, in the course of a longitudinal psychiatric epidemiological study of children in primarily low-income rural communities, a subset of the subjects began receiving unearned income supplements from the opening of profitable casinos in their communities[112]. This provided the equivalent of an experimental manipulation introduced part-way through the study. Fewer psychiatric symptoms were found in subjects from families that had received the income supplement, and this was particularly notable in those who were youngest when the supplements began. A recent follow-up showed the same pattern in adulthood, with lower rates of psychopathology among those who received the supplements at younger ages[113]. In another study, adoption provided a quasi-experimental test of environmental causation in the realm of intelligence[114]. This study examined the effect of adoption of one sibling to a higher or lower SES home on IQ in adolescence, with the nonadopted sibling of each adoptee as a comparison subject. The findings showed that the greater the SES increase of the adoptive family over the biological, the greater the IQ advantage of adopted child over sibling.

Box 3 Establishing causality in the neuroscience of socioeconomic status

In the effort to distinguish 'mere' correlation from causation, neuroscience has an advantage that is not shared by social science: the ability to experimentally manipulate the life conditions of animal subjects. Of course, animals do not have socioeconomic status (SES). However, many of the environmental factors that have been proposed to be proximal causes of SES disparities by social scientists can be manipulated in animals and have been shown to exert a causal impact on the brain[32]. For example, environmental stimulation has pervasive effects on brain structure and function[115]. Stress causes numerous molecular, cellular and anatomical changes in the brains of rodents and nonhuman primates[9]. Parenting behaviors are affected by environmental factors such as stress[116,117] and play a causal role in buffering the effects of stresses experienced by the

offspring[118]. Although we do not yet know how detailed the parallels are between the neural effects of stimulation, stress and parenting in animals and the correlates of SES in humans, there is a broad-brushstroke similarity. If this similarity is not a reflection of some shared causal mechanisms, then it is a remarkable coincidence.

Human neuroscience can also be brought to bear on the relationships between experience and the brain, albeit without the power of experimental methods to test causality. Some have examined the factors that statistically mediate the relationship of SES to brain function (e.g. [46]). For example, studies have shown that measures of stress or related measures of inflammation can account for the effects of SES on brain structure or function[45,119,120,121,122] (but see [123] for an exception).

A few human neuroscience studies have assessed the effects of interventions to improve the environment of poor children. Although these studies do not alter SES per se, they do manipulate some of the proximal factors by which SES is proposed to affect brain structure and function. Parenting interventions have been found to cause changes in attention and language processes reflected in event related potentials[74] and to be associated with less hippocampal volume loss at long-term follow-up[67]. Comprehensive programs including early childhood cognitive enrichment have resulted in changed neuroendocrine function[75] and later brain structure[124]. Ultimately, however, only a randomized controlled manipulation of SES can offer a definitive test of the causal effect of SES on the brain. It has recently been announced that such a study is being launched and will include an income intervention and neural as well as behavioral outcome measures[76].

Box 4 Neuroscience over-reach

Many individuals and organizations have embraced neuroscience as a source of poverty policy. Others have been decidedly less enthusiastic. For example, in 1997 the education theorist John Bruer attended a White House conference on the topic of ‘Early Childhood Development and Learning: What New Research on the Brain Tells Us About Our Youngest Children’. According to Bruer, the program displayed the sparseness of the connections between neuroscience and

childhood disadvantage; only one neuroscientist presented at the conference, and she cited work on visual development in cats. He reflected, “I heard numerous wide-ranging policy recommendations based on the new brain science. Yet, I had heard relatively little brain research ... and none that provided a clear link between blind kittens and welfare reform”[22].

A roughly contemporaneous project was the landmark book, ‘From Neurons to Neighborhoods’[125], whose title also seemed to promise a neuroscience–policy integration. Based on a National Academy of Sciences collaboration among scientists, child development and policy experts, the book contained an excellent chapter on brain development, alongside many other authoritative chapters on the psychology of children, families and communities. However, reflecting the state of the science of the time, there was little cross-referencing between the brain chapter and the others.

It is tempting to view these early attempts at neuroscience–social policy integration as indicative of neuroscience ‘over-reach’. Indeed, a number of prominent voices have argued that neuroscience is being oversold as an approach to diverse societal problems[23,126,127,128]. In addition, the ‘seductive allure of neuroscience’, a term coined by Weisberg and colleagues[129], describes the tendency of laypersons to be persuaded by neuroscience, even when it is nonsensical. Although these critics describe many valid examples of neuroscience over-reach, I believe that it would be a mistake to paint all attempts to develop nonmedical applications of neuroscience with the same broad brush. And, although the seductive allure of neuroscience is a real phenomenon, so is the ‘seductive allure of seductive allure’[130], which has created its own legacy of uncritical denigration of neuroscience, especially neuroimaging.

Furthermore, although there is no guarantee that progress in neuroscience will help us understand socioeconomic status (SES) and its life-course correlates, it seems highly likely that it will. Why would a fuller understanding of any problem, couched at any applicable level of explanation – physical, biological, psychological, economic, social or political – not be an asset when trying to solve the problem? In this very minimal way, at least, the neuroscience of SES is relevant to the design of poverty policy. Poverty has measurable neural correlates, for which neuroscience offers potential causal accounts, and this establishes the potential for policy relevance. As

modest a claim as this is, I believe that it bears stating explicitly as counterpoint to recent criticisms of neuroscience over-reach.

Figure 1 Trends in health and cognitive outcomes across levels of SES, and in SES neuroscience publications across time. a-c| Socioeconomic status is related to many important life outcomes, including physical health and longevity, mental health and wellbeing, and cognitive ability. Three illustrative findings are shown here. a| Estimated remaining life expectancy in 2005, for 25 year old Americans with different levels of educational attainment. b| Relationship between household income and the percentage of individuals that report symptoms similar to those of a mood disorder in Canada in 2014. c| Relationship between family income and average scores on the US Scholastic Aptitude Test in 2013. d| The neuroscience of SES has recently become an active area of research. This is reflected in the number of publications on this topic since the beginning of the century. The chart shows publications between 2000 and 2017, identified by searching titles and abstracts in PubMed for the five terms ‘socioeconomic’, ‘socioeconomic status’, ‘SES’, ‘income’ and ‘poverty’, combined with eighteen neuroscience terms including ‘MRI’, ‘DTI’, ‘EEG’, ‘grey matter’, ‘cortical’, ‘prefrontal’ and ‘default mode’, and eliminating articles with the terms ‘schizophrenia’, ‘breast’, ‘injury’ and ‘disease’. Part a, adapted from Rostron, Boies & Arias[131]. Panel b adapted from Canadian Community Health Survey, 2014. Ottawa, ON: Statistics Canada. Panel c adapted from College Board, 2013.

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Fig 1