HHS Public Access

Author manuscript

Trends Cogn Sci. Author manuscript; available in PMC 2024 September 01.

Published in final edited form as:

Trends Cogn Sci. 2023 September ; 27(9): 833–851. doi:10.1016/j.tics.2023.03.011.

Childhood socioeconomic status and the pace of structural neurodevelopment: Accelerated, delayed, or simply different?

Divyangana Rakesh, PhD[a], **Sarah Whittle, PhD**[b], **Margaret A. Sheridan, PhD**[c], **Katie A. McLaughlin, PhD**[a]

[a]Department of Psychology, Harvard University, USA

[b]Melbourne Neuropsychiatry Centre, Department of Psychiatry, The University of Melbourne and Melbourne Health, Victoria, Australia

[c]Department of Psychology and Neuroscience, University of North Carolina at Chapel Hill

Abstract

Socioeconomic status (SES) is associated with children's brain and behavioral development Several theories propose that early experiences of adversity or low-SES can alter the pace of neurodevelopment during childhood and adolescence. These theories make contrasting predictions about whether adverse experiences and/or low-SES are associated with accelerated or delayed neurodevelopment We contextualize these predictions within the context of normative development of cortical and subcortical structure and review existing evidence on SES and structural brain development to adjudicate between competing hypotheses. Although none of these theories are fully consistent with observed SES-related differences in brain development, existing evidence suggests that low-SES is associated with brain structure trajectories more consistent with a delayed or simply different developmental pattern than an acceleration in neurodevelopment.

Keywords

socioeconomic status; poverty; adversity; structural brain development; acceleration; delay

SES, adversity, and the pace of neurodevelopment

Adverse childhood experiences and access to resources in childhood, as measured by socioeconomic status (SES), have been consistently linked to children's neurodevelopment[1–5]. Recent theories have proposed that experiencing adversity or low-SES early in life may alter the pace of neurodevelopment[6–9]. While most of these models focus on adversity broadly[6–8], they have been expanded to include SES[9], given that SES likely impacts neurodevelopment via similar pathways[5,9,10] (although note that adversity

[##]Corresponding author: Dr. Divyangana Rakesh: divyangana_rakesh@g.harvard.edu, 33 Kirkland Street, Rm 1002, Cambridge, MA 02138. USA.

and low-SES are related but not interchangeable constructs, see Box 1). Importantly, these theories make contrasting predictions about whether adverse environmental experiences are associated with an acceleration or delay in the pace of neurodevelopment While some models propose that adversity and/or low-SES may lead to an acceleration in the pace of brain maturation, another recent model argues that delayed development is also a possibility. Empirically testing these predictions has been challenging as a clear articulation of specific evidence that would align with either acceleration or delay has not been provided. Further, most studies have relied on cross-sectional designs that cannot be leveraged to investigate these questions.

To adjudicate between these competing hypotheses, we first contextualize theoretical predictions within the context of normative structural neurodevelopment during infancy, childhood, and adolescence. We then review extant evidence from longitudinal studies to ascertain whether low-SES is associated with an accelerated, delayed, or a simply different trajectory of neurodevelopment We find that while none of these theories completely explain observed SES-related differences in structural neurodevelopment, current evidence indicates that low-SES is linked to brain structure trajectories that are more in line with a delayed or simply distinct developmental pattern rather than an acceleration in neurodevelopment. We suggest that low-SES may be associated with a distinct pattern of brain maturation that is less about the timing of the attainment of milestones (i.e., acceleration or delay) but the milestones themselves.

Evolutionary Development Theories

Theoretical models of how early experience might alter the pace of development are rooted in evolutionary developmental frameworks, which suggest that alterations in the pace of development may help children adapt to harsh and unpredictable environments[11–16]. These frameworks posit that evolution selected for enhanced plasticity during development such that early experiences could shape the pace of development to allow an individual to adapt to the demands of their current and future environment[15–17]. Resource-allocation trade-offs between growth, reproduction, and survival determine the pattern and timing of *life history traits*, including age of sexual maturation and reproduction, number of children, and investment in parenting. For example, in a harsh or threatening environment, faster development that results in earlier pubertal onset may be advantageous to maximize chances of reproduction prior to potential mortality[16,17]. In contrast, a slower and protracted developmental strategy may be adaptive in a safe and enriching environment with high parental investment[14]. The idea that early-life experiences may alter the pace of development has influenced developmental cognitive neuroscience, where it has been theorized that adversity is associated not only with the pace of pubertal development, but also with the pace of brain development

Neurodevelopmental frameworks

Numerous theoretical models make predictions about how early-life adversity and low-SES may influence the pace of neurodevelopment The Stress Acceleration Hypothesis (SAH) posits that adverse early-life experiences accelerate neurodevelopmental processes to reach

'adult-like' functioning earlier, specifically in brain circuits involved in emotion processing and regulation[6]. This model stipulates that early environments characterized by high levels of stress activate neural circuits underlying emotional learning and reactivity prematurely, accelerating the development of amygdala and medial prefrontal cortex (mPFC) functional connectivity. This acceleration of amygdala-mPFC circuit development is thought to be adaptive to allow for a faster transition from reliance on parents for emotion regulation to self-regulation[6]. The SAH focuses specifically on experiences of stress in caregiver-child relationships. Children from low-SES backgrounds are more likely to experience many forms of caregiving stress than their higher-SES peers, including parental separation, harsh parenting, family conflict, and low parental warmth and support[10,18–20]. Further, while the SAH refers specifically to caregiver-adversity and the acceleration of amygdala-mPFC circuit development, numerous studies have evaluated whether brain development is accelerated among children experiencing other types of adversity as well as low-SES[21–24].

A recent model extends the ideas of the SAH and applies them directly to SES, describing the types of experiences that may lead to accelerated neurodevelopment based on the valence and frequency of early experiences[9]. This model hypothesizes that negative and chronic childhood experiences, such as low-SES, are associated with faster brain development and reduced plasticity, while negative but uncommon experiences such as acute trauma are not[9]. This model posits that higher-SES is linked to a prolonged trajectory of neurodevelopment and enhanced plasticity that facilitates a longer trajectory of functional network segregation, ultimately resulting in more effective and refined neural circuits. Empirical studies testing these predictions are currently lacking.

Other theoretical models rooted in the Dimensional Model of Adversity[17,25–27], which distills adverse experiences into core underlying dimensions such as threat, deprivation, and unpredictability (see Box 1), make predictions about whether adversity is associated with accelerated or delayed development based on the dimension of adversity experienced. In the original dimensional model, reductions in social, cognitive, sensory, and linguistic stimulation associated with deprivation are argued to lead to excessive and exaggerated synaptic pruning, which leads to greater cortical thinning[7], a pattern typically interpreted to reflect accelerated cortical development More recent elaborations of these models, however, note that it is unclear whether a thinner cortex reflects acceleration or delay in neurodevelopment[15]. Rooted in the same conceptual framework, Colich et al.[24] hypothesized that threat, but not deprivation, would be associated with an acceleration in the pace of neurodevelopment specifically in cortical regions involved in social and emotional processing[24] that feature prominently in the SAH. It is therefore unclear whether SES should be associated with an acceleration or delay of neurodevelopment per these dimensional models.

Although most models predict acceleration of brain development as a function of adverse experiences, the recent "change of pace" model[8] considers both acceleration and delay in development This model suggests that the type of adversity encountered determines whether biological maturation is accelerated or delayed and that changes in the rate of development occur to eliminate gaps in parental caregiving. While the 'change of pace'

model focuses on the parent-child dyad, we have extended it to apply to low-SES in our review given the strong links between SES and parenting behaviors[28]. The model purports that delaying maturation lowers children's physiological requirements when there are unmet physiological needs in situations of deprivation, such as inadequate nutrition or parental care. In the event of threat or abuse, children may have unmet safety needs, and accelerated development may boost children's ability to provide for their own safety. The model also predicts that the aforementioned acceleration is time-limited and may switch to slower or delayed development after puberty. Low-SES is characterized by higher levels of material deprivation, such as food insecurity and reduced access to other basic necessities[29,30]. Given this, the change of pace model is consistent with the idea that SES might be associated with slower neurodevelopment Indeed, some longitudinal studies report that low-SES is associated slower neurodevelopmental trajectories[e.g., 31]. However, low-SES is also associated with greater exposure to community violence and other forms of threat[10,20], which are argued to accelerate neurodevelopment in this model. It is therefore unclear whether SES should be associated with an acceleration or delay of neurodevelopment per this model. Empirical studies directly testing the predictions of the change of pace model are currently lacking.

Evaluating the validity of these frameworks has been challenging as concrete predictions about what evidence would be aligned with acceleration or delay have not been articulated clearly. More problematic, most studies use data from cross-sectional designs to make inferences about accelerated versus delayed patterns of brain development[9,23,24]. Longitudinal research is needed to test how adversity and SES are associated with deviations from typical developmental trajectories, yet such studies remain rare.

Theoretical predictions within the context of normative development

In the following sections, we ground the predictions of each theoretical model in the context of normative patterns of gray matter development, highlight the types of evidence needed to adjudicate among competing hypotheses, and review empirical studies on SES and brain structure to ascertain which framework is best aligned with the evidence. We focus on low-SES and gray matter structure as longitudinal studies of other forms of adversity and other metrics of brain structure are limited[2] and typical patterns of cortical and subcortical development have been relatively well characterized[32–38].

Normative development

To investigate whether empirical evidence is aligned with theoretical predictions, we must contextualize these predictions within normative developmental trajectories. The brain undergoes protracted gray matter development throughout childhood and adolescence characterized by changes in cortical thickness, surface area, volume, and subcortical volume[37]. Cortical thickness increases during the first two years of life, with more rapid increases in the first relative to the second year peaking somewhere between 12-24 months[37]. Thickness then decreases rapidly in early childhood and is followed by monotonic thinning from childhood to adolescence[32–36]. Cortical volume increases in the first two years of life[39] followed by a more gradual increase in volume during childhood

peaking around 10 years of age[40], and non-linear decreases throughout adolescence, with varying rates of decreases across regions[36,41]. In contrast, cortical surface area greatly expands in the first years of life[42], continues to expand throughout childhood, peaking in late childhood or early adolescence, and then undergoes subtle decreases thereafter[32,34–36,43]. Finally, the volume of subcortical regions such as the amygdala and hippocampus increases throughout childhood and early adolescence, plateaus in middle to late adolescence, and decreases thereafter[40,44–47]. It is important to acknowledge however that these are *average* trajectories and that there is substantial individual variability in the magnitude and timing of the peak as well as in rates of change[48].

Theoretical predictions

Overall, models largely predict accelerated development as a function of adversity and low-SES, with the exception of one model that also considers the idea of delayed development. If the pace of brain development was accelerated[6,7,9] or delayed[8], we would expect to see a temporally *shifted* pattern of brain development That is, individuals with accelerated or delayed brain development would hit the same normative developmental milestones, but earlier or later, respectively. Below we briefly outline the expected patterns for different measures of cortical development as a function of low-SES. Since cortical thickness increases in the first two years of life, accelerated neurodevelopment in low-SES youth would be associated with more rapid growth trajectories resulting in an earlier peak and *increased* cortical thickness prior to age two years. Thereafter, accelerated development would manifest as more rapid cortical thinning, resulting in lower cortical thickness in low-SES relative to high-SES children beginning in early childhood and continuing through adolescence. If development were delayed, we would observe the opposite pattern—slower growth resulting in lower thickness during infancy, a later peak in cortical thickness, and slower thinning resulting a thicker cortex during childhood and adolescence in low relative to high-SES youth. Similarly, for volume and surface area, accelerated development would involve faster expansion and growth in early childhood, an earlier peak, and more rapid decreases during late childhood and adolescence. If development were delayed, the opposite pattern would be expected—slower growth in early childhood, a later peak, and slower decreases during adolescence. Finally, subcortical volume would exhibit more rapid growth resulting in higher volume if development were accelerated and slower growth resulting in lower volume if development were delayed. Figure 1 depicts these predictions using cortical thickness and subcortical volume as examples.

Most studies have examined individual differences in the pace of neurodevelopment using cross-sectional data in adolescents, which has hindered our ability to truly test these theories. For example, cross-sectional data makes it impossible to disentangle whether lower thickness, surface area, or volume in low-SES adolescents[1] reflects a difference in the amount of cortical gray matter or in the rate of change over time, highlighting the need for longitudinal studies. Therefore, in order to assess which of these frameworks is best aligned with existing evidence, several pieces of information are needed in conjunction. First, information on SES-related differences in the rate of change in cortical grey matter and subcortical volume across development is required, as models differ in predictions about whether the rate of change in brain structure is faster versus slower during infancy,

childhood, and adolescence. Second, models also differ in their predictions of whether cortical thickness and volume should be higher or lower in low-SES youth during infancy as well as childhood and adolescence (see Figure 1). Finally, knowledge about the timing of peak thickness and volume would help evaluate the predictions. Each of these pieces of information can be used to evaluate whether developmental trajectories are accelerated or delayed. We now review existing studies that provide the first and second pieces of information on SES and brain structure during infancy, childhood, and adolescence. Studies on differences in age at peak are currently lacking.

Empirical observations

Infancy

SES-related differences in cortical structure.—We identified six studies examining associations of SES with cortical structure in infants (Table 2). Four studies found that lower-SES was associated with *lower* cortical and subcortical volume[31,49–51] in neonates, infants, and toddlers. In contrast, one study reported both higher and lower cortical volume related to low-SES; infants aged 1-6 weeks from low-SES households had larger volumes in the occipital lobe, temporal pole, left inferior frontal regions, and anterior cingulate and lower volumes in the frontoparietal region and inferior temporal lobe relative to infants from high-SES households[52]. Partially in line with this, a study on a relatively large sample found low-SES to be associated with *higher* average cortical thickness and thickness of some frontal and temporal regions[53]. However, their findings could have been influenced by their adjustment for intracranial volume, which does not scale with thickness[54]. Although the literature is somewhat mixed, most findings, including those from well-powered samples of 756 infants aged 8-12 months and 280 neonates[51], suggest that low-SES is associated with *lower* cortical and subcortical volume early in life.

SES-related differences in rate of change.—To our knowledge, only one study has examined SES-related changes in cortical structure in infants or toddlers longitudinally. Low-SES infants had lower total, frontal, and parietal volume, and these differences became more pronounced with age[31], consistent with a slower pace of neurodevelopment.

Childhood and Adolescence

SES-related differences in cortical structure.—Numerous cross-sectional studies observe lower cortical thickness, surface area, volume, and subcortical volume among low-SES relative to high-SES children and adolescents[55–69]. For greater details see a recent systematic review[1]. Although studies vary in terms of specific regions where differences were observed, the evidence is remarkably consistent in the direction of the association between SES and brain structure.

SES-related differences in rate of change.—Longitudinal studies find low-SES to be associated with a *lower* rate of change (Table 3). For example, low-SES has been associated with reduced and slower growth in hippocampus[59,70,71] and overall subcortical[72] volume during childhood and adolescence. Three studies reported lower rate of change in cortical thickness and volume reported as a maturational lag in total gray matter, frontal,

and temporal volume in low-compared to high-SES children[73], lower rate of cortical volume growth in parts of the insula and superior temporal gyrus[72], and less cortical thinning over time in low-SES adolescents[74], suggesting slower cortical development[75]. Finally, using a brain-predicted age framework based on both cortical and subcortical data, one study showed that low-SES children had higher brain age gap values at age 12 followed by a negative trajectory, reflecting slower brain development[22]. Finally, a recent paper shows higher SES to be associated with more rapid cortical thinning and area reduction[76]. Not all findings are aligned with lower rate of change in youth from lower SES backgrounds, however. Mixed sex-dependent findings of slower and faster change[77], more rapid amygdala growth in males[74], and greater decreases in surface area[78] in low-SES adolescents have also been reported.

Brain developmental trajectories associated with low-SES may be simply different

Collectively, the evidence suggests that low-SES is associated with lower thickness, surface area, and volume and slower rate of change throughout infancy, childhood, and adolescence (Table 1, Figure 1). In addition, the pattern of findings does not appear to vary based on the specific SES indicator used, although the number of studies of each specific SES indicator is small. There have been a limited number of studies examining SES and brain structure in infants, and even fewer longitudinal studies, which makes it challenging to make definitive conclusions about this time period. However, the available evidence is more consistent with delayed than accelerated brain development in low-SES infants. While lower thickness, area, and volume in childhood and adolescence is consistent with accelerated brain development, patterns of change over time are consistent with delay rather than acceleration during this period. Although most findings were consistent with delayed brain development, the lack of evidence for low-SES children exhibiting *higher* thickness or volume in childhood or adolescence than high-SES children is inconsistent with a delayed maturational trajectory.

Based on this review, we stipulate that none of the models fully captures the existing pattern of evidence of SES-related differences in structural brain maturation. Instead, it may be that low-SES is associated with a simply *different* developmental trajectory characterized by lower cortical thickness and volume at all ages from infancy through adolescence as well as slower growth and slower thinning over time (Figure 1). This trajectory is most consistent with the evidence, which shows lower thickness, volume, and surface area and slower rates of change in individuals from low-SES backgrounds at all ages. Of note, this proposed trajectory may be more applicable to cortical and subcortical volume given the limited number of longitudinal studies that have examined cortical thickness and surface area trajectories, as well as the presence of null and mixed findings. Clearly, more longitudinal studies examining changes in different brain structural metrics over time are needed, particularly in the first years of life.

Mechanisms contributing to SES-related differences in brain structure

Several factors that vary as a function of SES—including prenatal factors, exposure to stress, and reduced cognitive stimulation—likely influence changes in underlying

neurobiological processes such as synaptic pruning and myelination and contribute to SES-related differences in large-scale brain morphology. These ideas have been discussed extensively[7,9,17,27,79]. We highlight some mechanisms that may explain SES-associated differences in the pace of brain development in each development period briefly.

Infancy

Local cellular events—such as rapid gains in dendritic complexity, myelination, synaptogenesis, glial proliferation, and axonal elongation—have been suggested to contribute to increases in cortical thickness and surface area in the first years of life[80–84]. Higher levels of enriching and stimulating experiences in high-SES households may alter cellular processes and contribute to SES-associated differences in brain structure. Evidence from animal models suggests that the expression of cellular signals involved in activity-dependent synaptic development is upregulated by enrichment including neurotrophins, brain-derived neurotrophic factor, synaptic proteins involved in synaptic proliferation and function, and factors implicated in glutamatergic signaling[79]. Low-SES is also associated with higher levels of family conflict and harsh parenting[85,86], meaningful sources of chronic stress in early life. Chronic stress also influences glial cell proliferation, which could contribute to differences in gray matter structure[87]. However, the mechanisms driving the associations between enrichment and stimulation, stress, and increases in cortical thickness and volume during the early years remain relatively unexplored.

Childhood and adolescence

Differences in brain structure are also evident in childhood and adolescence. It is possible that differences in proliferation during the first years of life simply carry forward into later developmental periods. Alternatively, differences in synaptic pruning could give rise to low-SES being associated with lower cortical thickness and volume in childhood and adolescence. For example, reduced dendritic spine density, branching and length of dendrites, and the number of synapses per neuron are all observed in animals raised in deprived environments[88–90]. In addition, greater chronic stress can cause spine loss[88], atrophy of apical dendrites[89], and suppress neurogenesis in the dentate gyrus[90], which could contribute to lower cortical thickness, volume, and subcortical volume. The slower rate of change reported in longitudinal studies suggests that greater pruning may not be a plausible explanation for SES-related differences in brain structure. Importantly, the biological mechanisms underlying reduced cortical thickness and surface area cannot solely be attributed to small-scale changes at the synapse level[37]. For example, changes in myelination and reduction in the number of glial cells can contribute to these developmental changes[37]. Understanding of how SES influences these processes remains limited.

Rate of change

To our knowledge, animal studies linking enrichment and stress with small-scale developmental changes at the level of synaptic pruning, myelination, and dendritic arborization have not been examined using longitudinal designs. The lack of such knowledge makes it challenging to comment on the mechanisms underlying slower *rates of change*. However, studies using the minimal bedding paradigm to mimic low-SES in rodents demonstrate impaired microglia-mediated synaptic pruning after this

manipulation[91]. Less pruning could reflect slower circuit refinement Enrichment also contributes to newly produced neurons being integrated into functional circuits[79], and computational neuroscience models show that network abilities benefit from early synaptic overgrowth followed by pruning of weak synapses[92]. Accordingly, lower overall synaptic proliferation could partially explain the differences in brain functional integration and segregation observed as a function of low-SES both early in life[93], and during childhood and adolescence[1,94–101]. For example, measures of network efficiency, such as within-network connectivity and global efficiency, which typically increase with age during development[102–107] are lower in children from low-SES backgrounds[95,108,109]. However, given limited longitudinal research on functional and structural connectivity, caution is warranted in interpreting these patterns.

Importantly, we have focused on postnatal differences in this review. However, given differences in brain structure observed in the first weeks of life[51], it is possible and even likely that SES influences brain structure before birth, which may create a persistent offset that is observed as cross-sectional differences in brain morphology at all ages. Differences at birth could be due to a host of prenatal factors including maternal stress, nutrition, prenatal complications, drug and toxin exposure, and pre-term birth[79]. Higher levels of stress, higher infection rates, and poor nutrition can increase the levels of corticotropin-releasing factor and glucocorticoids in the mother and fetus[110–113]. These factors can lead to restricted fetal growth and premature birth[110,111,113]. More neuroimaging studies that examine associations between prenatal factors and fetal brain development are needed. Further, genetics may also play a confounding role. That is, genetics may in part determine both the parent's SES as well as children's brain structure. Past work has shown that both SES and genetic factors contribute to educational attainment and impact cognitive and brain development in adolescents[78]. It is also possible that the initial offset present at birth may influence rates of change in brain structure, however, this is speculative and longitudinal research is needed to test this hypothesis. Finally, even postnatally, low-SES is associated with numerous factors other than chronic stress and cognitive stimulation that can influence brain development, including nutrition, school environments, and exposure to toxins and pollutants[50,114–116]. Research examining how these factors might independently and jointly shape neurodevelopment is sorely needed.

Concluding remarks and future directions

We examined the predictions of influential conceptual models on adversity and the pace of brain development Across models, the predictions differ in how adversity and low-SES should be associated with brain structure during infancy as well as childhood and adolescence and whether changes in brain structure should occur at a slower or faster pace. The empirical data suggests that none of these models fully captures the observed differences in structural development between low and high-SES youth, and that low-SES may be associated with a simply different neurodevelopmental trajectory. However, in the absence of longitudinal data that spans infancy, childhood, and adolescence, it is challenging to make definitive conclusions about accelerated, delayed, or different trajectories. Despite the first years of life being marked by rapid and dynamic brain development, there has been very little research on SES- and adversity-related differences during this period of life.

This is understandable given the challenges associated with infant neuroimaging. However, more longitudinal research that maps normative development as well as differences related to early experience from infancy to adolescence are needed to test these ideas thoroughly. Eventually, researchers will be able to combine data from studies such as Healthy Brain and Child Development and Adolescent Brain Cognitive Development to test associations between SES and changes in brain morphology from infancy to late adolescence.

Further, most of the conceptual models we evaluate focus on experiences of adversity broadly rather than SES specifically, with some exceptions[9]. We focus here on SES due to lack of longitudinal imaging studies and infant research on other forms of early-life adversity. However, it is important to acknowledge low-SES is not synonymous with adversity(Box 1) and that neurodevelopmental mechanisms beyond accelerated or delayed development may contribute to observed SES-related differences in brain structure. Many children raised in low-SES families receive enriching cognitive and social stimulation and are not exposed to harsh parenting or violence. Further, whether the patterns of structural brain maturation observed here apply to other forms of adversity is unknown and is a critical topic for future research, although similar patterns as those described here have been reported in relation to other forms of adversity in several studies. For example, numerous cross-sectional studies observe lower cortical thickness in children who have experienced maltreatment, exposure to violence, and severe deprivation related to institutional rearing[2], which is often interpreted to be consistent with accelerated development. Longitudinal work shows reduced growth in amygdala volume over time in adolescents exposed to maltreatment[117], which reflects a slower rate of development. More longitudinal research in this area is sorely needed. Further, SES is a broad and complex construct that can be operationalized in multiple ways—for example household income, parental education, and neighborhood SES as well as in the form of composite SES indices like Hollingshead Index. These indices tend to be moderately correlated[118] and may influence brain development through both distinct and similar pathways[see Box 1; 5]. More studies are needed to examine independent associations of different SES indicators with brain maturation.

Brain development is a profoundly complicated process. SES can influence brain development in numerous ways that vary meaningfully as a function of the presence of other risk and protective factors. Critically, the lack of longitudinal studies using other imaging modalities precluded us from examining associations between SES and the pace of maturation of white matter structure, structural and functional connectivity, and task-based activation(see Outstanding Questions), which also play an important role in behavioral outcomes. For example, although low-SES may be associated with lower cortical thickness and brain volumes on average, which has been shown to mediate links between SES and cognitive performance in young people[67,119,120], other neurodevelopmental changes associated with low-SES are likely to confer important advantages that help children adapt to the environment in which they are developing[121]. For example, the ability to switch between tasks or mental sets quickly and easily, and the capacity to track novel environmental information tend to be enhanced in children and adults who grew up in more unpredictable family environments[122,123]. Low-SES is likely associated with numerous brain adaptations that help children develop such skills and thrive in their environment[124]. Further, it is also important to consider the complex relationships of SES

and adversity with systemic and interpersonal racism when examining associations with neurodevelopment[125,126]. In addition to variability in covariates included, some studies have covaried for race and ethnicity while others have not (see Supplementary Materials), which makes it somewhat challenging to compare findings. Further, results from studies that covary for race/ethnicity but do not have an even distribution of SES across racial and ethnic groups in the study need to be interpreted cautiously[127]. In addition, research in this area has relied heavily on data from Western, Educated, Industrialized, Rich, and Democratic(WEIRD) countries, limiting our ability to generalize findings to other countries and cultures. Finally, while parental SES does not change substantially during childhood for most individuals[128], given our limited knowledge about timing effects and when brain maturation may be most sensitive to SES or changes in SES, we are unable to comment on how these brain maturation curves(Figure 1) may change if SES were to increase or decrease. This is an important direction for future work, particularly in the context of interventions (see Box 2).

In sum, existing evidence is more, but not entirely consistent with low-SES predicting delayed rather than accelerated brain development. No existing model *fully captures* observed differences between low- and high-SES youth. Low-SES and other adverse environments are likely associated with brain developmental trajectories that differ in multiple ways considering the available evidence. Our understanding of how SES may influence the pace of neurodevelopment is limited and more longitudinal work, particularly during infancy and early childhood, is needed to establish normative developmental trajectories and to test the predictions of neurodevelopmental pace models more rigorously. Based on the available evidence, we suggest that low-SES may be associated with a distinct pattern of brain maturation that is less about the timing of the attainment of milestones (i.e., acceleration or delay) but the milestones themselves.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Outstanding Questions

What are the normative brain developmental patterns in utero and during infancy, and how is SES associated with these trajectories?

How is SES associated with developmental changes in cortical thickness and surface area during childhood and adolescence?

How does SES relate to maturation of white matter structure, structural and functional connectivity, and task-based activation?

What are the neurobiological processes underlying change in cortical and subcortical brain structure across infancy, childhood, and adolescence, and what is the role of SES in shaping these mechanisms?

What are the proximal environmental factors that mediate the association between SES and changes in brain structure over time?

How do SES-associated differences in structural brain structure development impact functional network development and circuit refinement in the brain?

How can we disentangle the role of prenatal factors, genetics, and SES in shaping brain development?

Can this model be extended to other types of adversity including childhood abuse and traumatic experiences?

Highlights

Theories make contrasting predictions about whether adverse experiences and low SES are associated with accelerated or delayed neurodevelopment.

Existing evidence is more consistent with low-SES predicting delayed rather than accelerated brain development. However, no existing model fully captures observed differences between low- and high-SES youth.

Low-SES and other adverse environments are likely associated with brain developmental trajectories that differ in multiple ways considering the available evidence.

We suggest that low SES is associated with brain maturation patterns characterized by lower volume and slower rates of change throughout development.

Longitudinal research, especially in the early years, is needed to rigorously test how adversity and SES are associated with deviations from typical developmental trajectories.

Box 1:

Defining adversity and socioeconomic status.

Many conceptual models on the associations between early experience and the pace of neurodevelopment focus on experiences of adversity broadly rather than SES specifically. It is important to acknowledge that while low-SES is a risk factor for adverse experiences, low-SES is not synonymous with adversity. Childhood adversity is defined as early-life stressors that are either chronic and/or severe and likely to require meaningful adaptation by an average child[129]. Adversity can be conceptualized in different ways, including cumulative risk and dimensions of adversity such as threat, deprivation, and unpredictability[15] (see Table I). Children from low-SES backgrounds are more likely to experience these forms of adversity than their peers from higher-SES backgrounds [10,130], although it is important to note that many children raised in low-SES environments do not encounter adversity. In addition, it is important to note that low-SES is associated with other exposures and experiences that do not neatly fit into any of these adversity definitions but may influence neurodevelopment such as crowding, pollution and toxicant exposure, high levels of noise, and lack of access to green spaces.

Table I.

Definitions of different conceptualizations of adversity.

Cumulative risk	Dimensions		
	Threat	Deprivation	Unpredictability
The cumulative risk approach focuses on the number of adverse experiences a child has encountered and assumes that these experiences have additive influences on developmental outcomes. Cumulative risk assumes that different types of adverse experiences influence behavioral and neural development through mechanisms that are largely universal or shared.	Threat refers to experiences that involve harm or the possibility of harm to one's physical integrity. This includes experiences where the child is directly victimized, such as physical abuse, as well as situations where the child witnesses harm occurring to others, such as violence between caregivers.	Deprivation refers to reductions in social and cognitive inputs from the environment during development, leading to limited opportunities for learning.	Unpredictability can be described as a state of environmental instability, where there is a lack of routine and frequent, rapid, and/or unanticipated changes in the environment. It can also be defined as stochastic variation in extrinsic morbidity-mortality.

SES is a broad and complex construct that represents access to or possession of both material resources, which is often indexed by income, and non-material resources such as educational attainment and neighborhood quality (see Table II). Subjective social status and parent occupational prestige have also been used as measures of SES, although these methods of measuring SES have rarely been studied in relation to neural outcomes in developmental studies outside of composite SES indices[1]. Generally, these different metrics of SES tend to be moderately correlated [118], which suggests that they capture unique aspects of the environment and may influence brain and behavioral development through pathways that are both shared and unique[68,94]. Importantly, each of these

aspects of SES are associated with differences in exposure and experiences—like stress, adversity, stimulation, and support—to different extents.

Table II.

Definitions of commonly used indicators of SES.

Educational attainment	Income	Neighborhood disadvantage	Composite SES
Educational attainment reflects human capital. Education is usually operationalized either as the highest or average level of completion (e.g., high school, college, professional degree) or as the total number of years of education completed.	Income reflects financial or economic resources. Income is typically measured as total monthly or annual household income, typically adjusted for household size by computing an income-to-needs ratio.	Neighborhood disadvantage reflects the socioeconomic characteristics of the neighborhood. It is typically computed by aggregating across multiple neighborhood-level measures of employment, education, and income. In addition, neighborhood measures can also capture opportunity levels in the neighborhood like access to early childcare centers and school quality.	Composite measures of SES are aggregate measures of the child's socioeconomic environment. They can be operationalized by aggregating income, education, occupation (and other indicators) in measures such as the Hollingshead index, normalizing and averaging data across indicators, or constructing a composite measure using factor loadings of those indicators from a latent SES model.

Box 2:

Leveraging intervention studies to establish causal inferences

It is crucial to acknowledge that the findings presented in this review are derived from observational studies, and as such, cannot establish a causal relationship between SES and brain development Intervention studies that involve changing income through cash transfers and quasi- experimental approaches can provide more definitive causal evidence for these associations. Numerous such studies support a causal relationship between increased income and improved academic outcomes for low-income students. For example, in the United States and Canada, quasi-experimental research has utilized income boosts to demonstrate that increases in income produce higher levels of school achievement in children[131–133]. Similar intervention studies that examine impacts on neurodevelopment have rarely been conducted. One key exception is the Baby's First Years Study[134], which provides cash assistance to low-SES mothers during the first years of their child's life and is collecting metrics of brain structure and function in the children across development. These types of studies can determine whether changes in income are causally associated with corresponding changes in brain structure as well as the pace of neurodevelopment Further, studies that intervene on specific environmental pathways that may mediate associations between SES and outcomes, such as by providing higher quality early education and child care, have also shown promise in improving a wide range of developmental outcomes[135–138]. Determining whether these interventions improve outcomes by contributing to changes in brain development is a critical question for future research. Such research can also help to identify periods when brain development is most responsive to intervention and inform optimal windows for intervention.

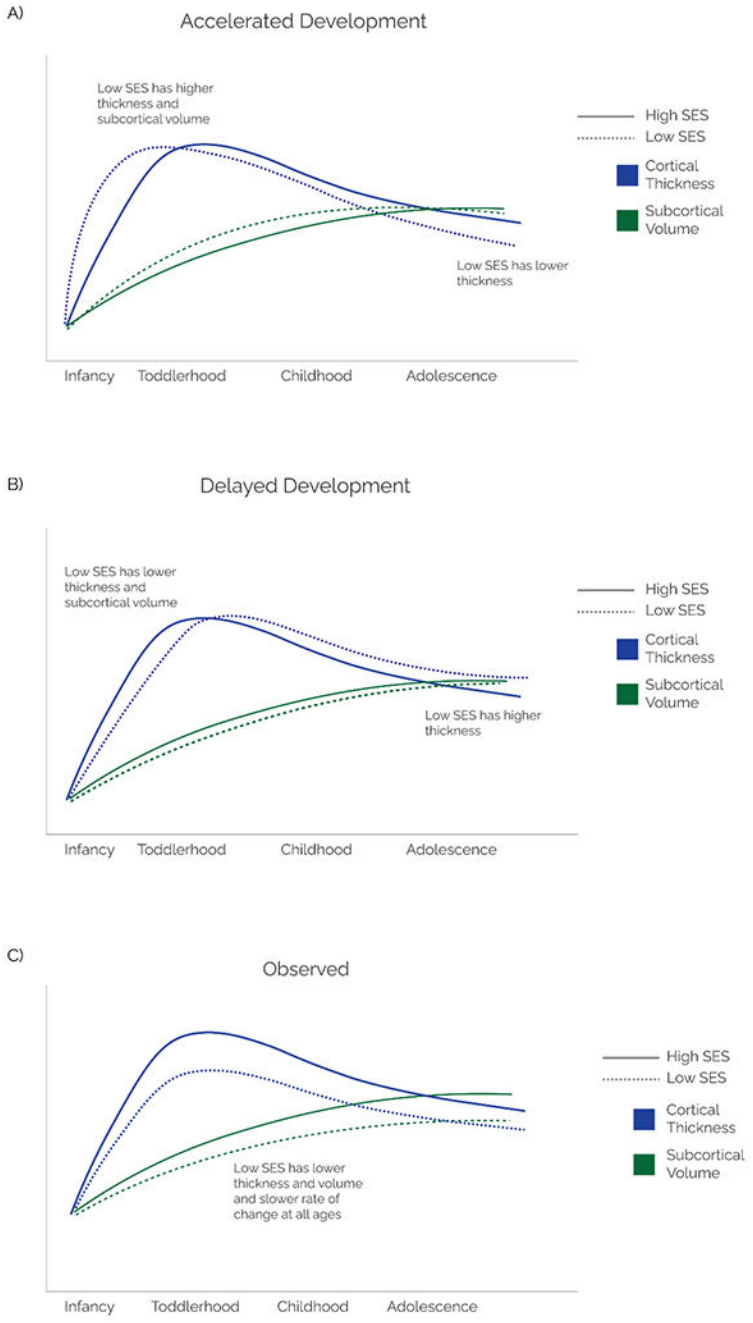

Figure 1: SES and the pace of neurodevelopment: theoretical predictions and empirical observations.

Expected trajectories of cortical thickness from infancy to late adolescence based on models of accelerated (A) and delayed (B) brain development Solid and dashed lines represent trajectories for high and low-SES youth, respectively. Panel C depicts the patterns observed in existing longitudinal studies. These patterns suggest that low-SES children have consistently lower cortical thickness, volume, surface area, and subcortical volume as well as slower rates of change during both growth and decline. Figures depict the starting point for low- and high-SES infants to coincide as evidence on SES-related differences in brain

volume at birth is limited. Blue and green lines represent average trajectories for cortical thickness and subcortical volume—specifically amygdala and hippocampus, respectively.

Table 1.

Predicted versus observed data.

	Accelerated	Delayed	Observed
	Cortical Measures		
Infancy, toddlerhood	Higher[1,2,3]	**Lower**[1,2,3]	Lower[3]
	Earlier peak[1]	Later peak[1]	Slower change[3]
	Faster change[1,2,3]	**Slower change**[1,2,3]	
Childhood, adolescence	**Lower**[1,2,3]	Higher[1,2,3]	Lower[1,2,3]
	Earlier peak[2,3]	Later peak[2,3]	Slower change[1,2,3]
	Faster change[1,2,3]	**Slower change**[1,2,3]	
	Hippocampus and Amygdala Volume		
Infancy, toddlerhood	Higher	**Lower**	Lower
	Faster change	**Slower change**	Slower change
Childhood, adolescence	Higher	**Lower**	Lower
	Earlier peak	Later peak	Slower change
	Faster change	**Slower change**	

The table depicts the predictions of accelerated versus delayed development and compares them to the patterns observed in empirical studies, with the top panel referring to cortical measures and the bottom panel subcortical measures. Bolded text indicates when a prediction matches an empirical observation, listed in the observed column. Of note, for measures that peak during childhood and adolescence such as surface area, cortical volume, and subcortical volume, if development were accelerated, values would be higher before the peak and lower after the peak. On the other hand, if development was delayed, values during childhood and adolescence would be lower before the peak and higher after the peak.

[1] = Cortical Thickness,

[2] = Cortical Surface Area,

[3] = Cortical Volume

Table 2.

Infancy/childhood studies

Study	Total N (Females)	Mean age (age range/SD)	SES measure	Imaging measure	Key Findings	Lower/ higher in low-SES
Betancourt et al. (2016) [49]	44 (25 low-SES, 19 high-SES), African American, 100%F	5 ± 0.9 weeks	Composite SES (based on maternal education and income-to-needs)	Cortical gray, deep gray, and white matter volumes	Low SES infants had lower cortical gray matter and deep gray matter volume than high SES infants	Lower
Hanson et al. (2013) [31]	77 (40%F)	13.7 months (5 months to 4 years)	Household income	Total GMV and lobal volumes, white matter volume, total cerebral volume	Low SES toddlers had lower total, frontal, and parietal volume than high SES toddlers	Lower
Jha et al. (2019) [53]	805 (47%F)	30.6 days (6- to 144-days post birth)	Paternal and maternal education, income	Cortical thickness and surface area (average and regional)	Low paternal education was associated with higher average cortical thickness and thickness of some frontal and temporal regions (while adjusting for global brain size and ethnicity)	Higher
Knickmeyer et al. (2017) [50]	756 (47%F)	8-12 months	Maternal and paternal education and income	Total GM and WM	High paternal education was associated with higher gray matter and white matter volume (partially mediated through birth weight)	Lower
Spann et al. (2020) [52]	37 (35%F)	1-6 weeks	Hollingshead (education and occupation)	Volume	Infants born to mothers with lower SES had larger local volumes in the bilateral superior and middle occipital gyri, right middle frontal, and temporal pole, left inferior frontal and anterior cingulate regions. Low SES was associated with lower volumes in the frontoparietal region and the inferior temporal region.	Mixed
Triplett et al. (2022) [51]	280 (47%F)	First weeks of life	Socioeconomic disadvantage: factor analysis of health insurance status, highest educational level, income-to-needs ratio, national Area Deprivation Index percentile at birth, and Healthy Eating Index.	Cortical and subcortical gray matter, white matter, and cerebellum volume, hippocampus and amygdala volume	Neonates born to mothers from disadvantaged backgrounds had lower cortical and subcortical gray matter volume and lower white matter volume	Lower
Qiu et al. (2017) [139]	168 (44%F)	Mean post-conception age at scan 40 weeks	Household income	Subcortical volume and cortical thickness (whole brain)	Neonates with genetic profiles associated with heightened risk for developing depression showed a negative relationship between their family income and right amygdala and hippocampal volumes, whereas those with low genetic risk profiles showed no such association	Higher (only in those with high genetic risk for MDD)

Abbreviations: GM = gray matter, GMV = gray matter volume, SES = socioeconomic status, WM = white matter

Table 3.

Studies measuring rate of change

Study	Total N (Females)	Mean age ± SD (range)	SES measure	Number of imaging time points	Imaging measure	Key Findings	Faster/ Slower/ Different
Barch et al. (2020) [70]	167 (48.5%F)	15.83 ± 1.11 (13-19)	Income-to-needs ratio (family income/federal poverty level based on family size)	3	Volume	Greater T1 poverty was associated with a shallower hippocampal slope	Slower
Barch et al. (2022) [72]	210 (50%F)	3-6 years at T1 and 15-21 at T5	Income-to-needs	5	Volume	High INR was associated with faster subcortical volume growth and faster cortical volume growth (of a few regions)	Slower
Ellwood-Lowe et al. (2018) [71]	116 (100% F)	14.82 ± 3.09 (10-23). Time between first and last scans - 4.61 ± 2.15	Family income and parental education	4	Volume	Hippocampal volume in girls from lower-income homes exhibited a modest decline during the teenage years, then an increase through to early adulthood. In contrast, hippocampal volume of high-income females peaked around the age of 19, and then started to decline.	Different (non-linear), high-income girls appear to reach a higher and earlier peak
Hair et al. (2015) [73]	389 (52.5% F)	11.1 (4-20) (first scan; follow-up period 24 months)	Household income	3	Volume	Low-income children exhibited maturational lag in total gray matter and the frontal lobe, temporal lobe, and hippocampus.	Slower
Hanson et al. (2013) [31]	77 (40%F)	13.5 ± 7.9 months at first scan, average time between scans = 6.5 ± 4.1 months	Average household income across time-points	3	Volume	Low SES was associated with a slower growth trajectory of development for total gray matter volume and volume of the frontal and parietal lobe	Slower
Judd et al. (2020) [78]	551 (58%F)	T1: 14.44 ± 0.38 T2: 19.01 ± 0.7	Composite SES: sum of mother's education score, father's education score, family stress unemployment score, financial difficulties score, home inadequacy score, neighborhood score, financial crisis score, mother employed score, and father employed score	2	Surface area, thickness, subcortical volume	Greater decreases in surface area in low-SES individuals (NS for thickness and subcortical volume)	Faster
Kalantar-Hormozi et al. (2023) [76]	183 (42%F)	Three repeated scans per subject, approximate interval 2.8 years; mean age: 11.2 ± 2.7, range 5-24.2	Hollingshead two-factor index	3	Covariation across multiple cortical features (cortical thickness, surface area, local gyrification index, and mean curvature)	Higher SES was associated with accelerated cortical thinning and area reduction	Slower

Study	Total N (Females)	Mean age ± SD (range)	SES measure	Number of imaging time points	Imaging measure	Key Findings	Faster/ Slower/ Different
King et al. (2020) [77]	147 (57%F)	T1: 11 ± 1 (9-13) T2: 13 ± 1 (12-17)	Income-to-needs	2	Volume	Higher INR was associated with less expansion, or greater contraction, in the right hippocampal cingulum in boys, but was weakly associated with greater volume expansion in girls Higher INR was associated with greater volume expansion in the left inferior temporal gyrus, bilateral lingual gyrus and in the right superior parietal lobule and less volume expansion (or more contraction) in an area of the bilateral superior frontal gyrus in boys	Mixed and sex-dependent
McDermott et al. (2019) [59]	623 (48%F)	12 ±4 (5-25)	Hollingshead two-factor index	Long (344 individuals had > 1 scan)	Volume	High SES was associated with greater increases in hippocampal volume	Slower
Rakesh et al. (2021) [22]	166 (52%F)	T1: 12.79 ± 0.43 T2: 16.7 ± 0.52 T3: 19.08 ± 0.46	Neighborhood disadvantage	3	Brain-predicted-age (based on surface area, thickness, and volume)	Low SES was associated with a negative brain-predicted-age trajectory (i.e., greater brain-predicted-age at earlier ages but not at later ages)	Slower trajectory
Whittle et al. (2017) [74]	166 (52%F)	T1: 12.79 ± 0.43 T2: 16.7 ± 0.52 T3: 19.08 ± 0.46	Neighborhood disadvantage	3	Thickness and volume	Low SES was associated with less thinning (more thickening), greater increases in amygdala volume	Slower for cortical thickness, faster for amygdala in boys

Abbreviations: INR = income-to-needs ratio, SES = socioeconomic status