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Associations between Socioeconomic Status, Cognition, and Brain Structure: Evaluating Potential Causal Pathways Through Mechanistic Models of Development

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Abstract

Differences in socioeconomic status (SES) correlate both with differences in cognitive development and in brain structure. Associations between SES and brain measures such as cortical surface area and cortical thickness mediate differences in cognitive skills such as executive function and language. However, causal accounts that link SES, brain, and behavior are challenging because SES is a multidimensional construct: correlated environmental factors, such as family income and parental education, are only distal markers for proximal causal pathways. Moreover, the causal accounts themselves must span multiple levels of description, employ a developmental perspective, and integrate genetic effects on individual differences. Nevertheless, causal accounts have the potential to inform policy and guide interventions to reduce gaps in developmental outcomes. In this article, we review the range of empirical data to be integrated in causal accounts of developmental effects on the brain and cognition associated with variation in SES. We take the specific example of language development and evaluate the potential of a multiscale computational model of development, based on an artificial neural network, to support the construction of causal accounts. We show how, with bridging assumptions that link properties of network structure to magnetic resonance imaging (MRI) measures of brain structure, different sets of empirical data on SES effects can be connected. We use the model to contrast two possible causal pathways for environmental influences that are associated with SES: differences in prenatal brain development and differences in postnatal cognitive stimulation. We then use the model to explore the implications of each pathway for the potential to intervene to reduce gaps in developmental outcomes. The model points to the cumulative effects of social disadvantage on multiple pathways as the

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source of the poorest response to interventions. Overall, we highlight the importance of implemented models to test competing accounts of environmental influences on individual differences.

Keywords: Cognitive development; Socioeconomic status; Brain development; Structural MRI; Intelligence; Heritability; Artificial neural networks; Intervention; Cortical surface area; Cortical thickness

1. Introduction

In recent years, there has been an increasing application of developmental science to the effects of poverty on child development, such as the Lancet series *Advancing Early Childhood Development: from Science to Scale* (Black et al., 2017; Britto et al., 2017). Some of the basic science on which this work is founded includes emerging literature on the effects of differences in socioeconomic status (SES) on neurocognitive development (Farah, 2017; Noble & Giebler, 2020). For example, in one influential finding, Noble et al. (2015) demonstrated that in a U.S. sample of over 1000 children between 3 and 20 years of age, family income was associated with differences in cortical surface area in a nonlinear fashion, with effects found across the SES range but stronger associations at lowest levels of income. The association was particularly observed in temporal and frontal regions associated with language and executive skills, two domains where correlations are observed between family SES and behavior in children; indeed, the measure of cortical surface area was found to partially mediate the relationship between SES and behavior.

In principle, data of this type can be used to inform interventions that can narrow the gaps between children (Tomasi & Volkow, 2021). However, SES is a complex construct with multiple dimensions correlated with, among others, differences in prenatal maternal nutrition and stress; parenting behavior and parental relationships; physical, cognitive, and social resources; environmental factors impacting health; neighborhood and schooling; as well as community values and expectations. Influences on neurocognitive development may, therefore, operate via multiple causal pathways reflecting proximal factors correlated with SES, such as stress, linguistic and cognitive stimulation, parenting practices, prenatal care, toxins, and nutrition (Farah, 2017). Causal models are necessary to test hypotheses concerning the origin of brain structure differences and their relationship to function. Without such models, empirical data can be misinterpreted. They may, indeed, represent a double-edged sword: although brain structure differences send a strong message to policymakers regarding the impact of poverty on child brain development, they may also encourage the deterministic view that if brain structure has been affected, the cognitive gaps between children will be difficult to narrow.

Constructing a causal account of the brain structure differences associated with SES represents a formidable challenge because the account must simultaneously link between levels of description (brain structure and behavior); it must operationalize the proximal pathways through which distal measures of SES (such as family income and parental education) influence brain and cognition; and it must embed individual difference factors, such as SES, within a developmental framework. Macro changes in brain structure metrics derived from magnetic resonance imaging, such as cortical thickness and cortical surface area, must be linked to their

local functional consequences, which then contribute to variation in the high-level behavior of the whole organism. Specific behavioral measures must be linked to specific mechanisms. Moreover, any account of environmental influences on the development of brain and cognition must also capture the often larger effects of genetic variation on brain and behavioral development—thereby implicating the even lower level of description of genes (Deary, Cox, & Hill, 2022).

In this paper, we consider how to make progress in formulating a mechanistic account that links SES, cognition, and brain structure. There are four parts. In part 1, we give an overview of what sets of data need, minimally, to be integrated into putative causal accounts of SES effects on neurocognitive development. In many cases, these data are global in nature, identifying macro measures of brain structure and overall intelligence. We then argue that computational modeling offers a useful tool to help build such an account because it clarifies causal relationships and evaluates the implications of competing assumptions. However, while the empirical data often pick out global effects, models of mechanism must pertain to specific domains. To illustrate, in part 2, we present a simple model, drawn from the domain of language, to show how behavior, brain structure indices, and environmental influences associated with variations in SES can in principle be related within a developmental framework. The multiscale model is based on an artificial neural network (ANN) and builds top-down from behavior to interpret substrate properties that may link with function. The model accommodates individual differences in three neurodevelopmental processes: network growth, experience-dependent change, and connectivity pruning. The model is used to contrast two of the putative pathways by which environmental influences might impact behavioral and brain development: early network growth or postnatal cognitive stimulation (as well as testing the combination of both influences). In part 3, we then use the simple model as a foundation to test the effectiveness of a mid-childhood behavioral intervention targeting increases in cognitive stimulation. Within this limited framework, these simulations address the following question: do the different potential pathways of SES-associated environmental influence on brain and cognitive development have implications for the effectiveness of behavioral interventions to narrow the gaps in the cognitive outcome? In part 4, we discuss the strengths and limitations of using highly simplified, implemented models to address the association between SES, brain, and behavior—given that SES is, ultimately, a social construct.

2. Associations between SES, brain, and behavior, and the data with which they must be reconciled

2.1. SES effects

Differences in SES are correlated with variation in cognitive development (Farah et al., 2006). These associations are not uniform across all areas of cognition and are particularly marked in the development of language and cognitive control (executive functions). SES associations have been observed on intelligence (IQ): lower IQs are observed for children from lower SES families from an early age. In one sample of over 14,000 UK children followed

longitudinally from 2 to 16 years of age data, von Stumm and Plomin (2015) observed that SES gaps in IQ widened across development.

Objectively measured SES refers to a cluster of related measures concerning parental income, parental education, and the family and neighborhood environment in which a child is raised. It is a marker for potentially multiple causal pathways acting on cognitive development, among them effects on prenatal brain development, postnatal nurturing, and postnatal cognitive stimulation, together representing complex interacting social and biological influences across a prolonged developmental period (Farah, 2017; Hackman, Farah, & Meaney, 2010). For example: prenatal cortisol exposure predicts infant IQ at 17 months of age, suggesting that maternal stress during pregnancy affects brain development prenatally (Bergman, Sarkar, Glover, & O'Connor, 2010); children growing up in lower SES families may experience greater levels of chronic stress, which may negatively impact on postnatal brain development (Merz, He, Myers, & Noble, 2020); the contrasting environmental challenges experienced by children at different SES levels may produce different behavioral adaptations in decision-making, leading to exposure to different subjective environments (Sheehy-Skeffington, 2020); and different levels of language experience associated with different SES levels predict children's subsequent vocabulary growth (Hart & Risley, 1995), consistent with the view that differential cognitive stimulation is a contributing causal pathway (Brito, 2017). Despite the multiple potential causal pathways linking a distal measure of SES such as parental education with cognition—or perhaps because of the correlated nature of those influences—when Tucker-Drob (2013) applied structural equation modeling to data from a sample of 4800 U.S. children between kindergarten through 12th-grade who had been tested on six distinct cognitive abilities, he found that only a single dimension of variation linked parental education and cognition.

An increasing literature has addressed correlations between SES and variations in brain structure (e.g., Betancourt et al., 2016; Brito & Noble, 2014; Farah, 2017; Judd et al., 2020; McDermott et al., 2019; Noble et al., 2015; Noble & Giebler, 2020; Raffington et al., 2019; Tomasi & Volkow, 2021; Yaple & Yu, 2020). Compared to functional measures of brain activity, structural measures have the advantage of revealing the extended, accumulated effects of environmental influences, rather than reflecting details of task paradigms, baseline comparisons, and potential differences in the way that individuals from different SES levels react to experimental settings. The disadvantage of macro measures of brain structure is the remoteness of indices such as cortical thickness and cortical surface area from the micro-level neurophysiological processes that drive behavior. In turn, this forms a barrier to building mechanistic explanations of the observed differences.

Structural studies have considered associations between SES and global brain measures (such as volume of cortical or deep gray matter, cortical thickness, and cortical surface area), regional measures, the size of limbic structures, such as the hippocampus and the amygdala, and white matter connectivity. Findings have not always been consistent, in part due to differences in the regions and measures considered, perhaps also due to the heterogeneity of samples. There has been a particular focus on the prefrontal cortex, due to the correlation between SES and executive function skills. Reflecting the state of the field more generally,

Farah's (2017) review of the literature concluded that despite positive findings, no specific area of the frontal cortex has been reliably demonstrated to consistently vary with SES.

In the formal modeling section of this article, we focus on two exemplar studies of SES effects on brain structure. The first is a large and influential cross-sectional study from the United States. Researchers collected behavioral and brain structure data from 1099 individuals between the age of 3 and 20 years of age, considering a wide range of SES and controlling for differences in genetic ancestry (Noble et al., 2015). The authors considered relationships between two metrics associated with SES, parental education and family income, and global and regional measures of cortical thickness and cortical surface area, as well as the volumes of the left and right hippocampus and amygdala. The vast majority of the variance in brain structure was not explained by SES measures. This is because global metrics of brain structure tend to scale with intracranial volume and body size (Grasby et al., 2020) and appear less sensitive to the environmental variation indexed by SES. Nevertheless, small but reliable associations were found in both global and regional cortical measures, for parental education and family income. Effects were stronger for family income (see also Tomasi & Volkow, 2021) and as we have seen, the relationship was both graded across the SES range and nonlinear, manifesting more strongly at lower levels (e.g., for annual family incomes below $25,000 per annum). Lower family income was linked to smaller cortical surface area and thinner cortex, with stronger effects on surface area ($\beta = .185$, $p = .004$) than on thickness ($\beta = .088$, $p = .054$). Family income explained around 10% of the variance in behavior (four cognitive tests of vocabulary, reading, working memory, and selective attention); around 3% of the variance in cortical surface area (Fig. 1); and 1% in cortical thickness. The effects were uneven across cortical areas, showing the strongest effects in temporal and frontal regions, which the authors linked to the SES associations observed with executive function, language, reading, and spatial skills. Given the older age of these children, these effects are candidates for experience-dependent influences on brain structure.

Our second exemplar study looks at an earlier age group. In a cross-sectional sample spanning 3–20 years of age, there is extensive scope for postnatal factors to produce SES-linked differences, such as parental nurturing and cognitive stimulation. Betancourt et al. (2016) looked at a small sample of 44 1-month-old infants to reduce the potential for such factors to act. They focused on healthy-term female African-American infants to increase sample homogeneity, families who were poor or near-poor, and used a composite SES measure derived from income-to-needs ratio and maternal education. In analyses that controlled for birthweight, they observed linear relationships between SES and cortical gray matter (around 8% of the variance; shown in Fig. 1b), SES and deep gray matter (7% of the variance) but not white matter volume (1.5% of the variance, not significant). Given the young age of the participants, the authors pointed to prenatal effects of maternal health, toxin exposure, nutrition, sleep quality, and stress as possible causal pathways.

Given multiple pathways by which SES may influence brain structure, what systematic methodology might distinguish which are the most important? Farah (2017) identified three important properties within the data: the age at which differences are observed, the way that SES modulates developmental trajectories, and whether observed structural differences mediate the relationship between SES and behavior.

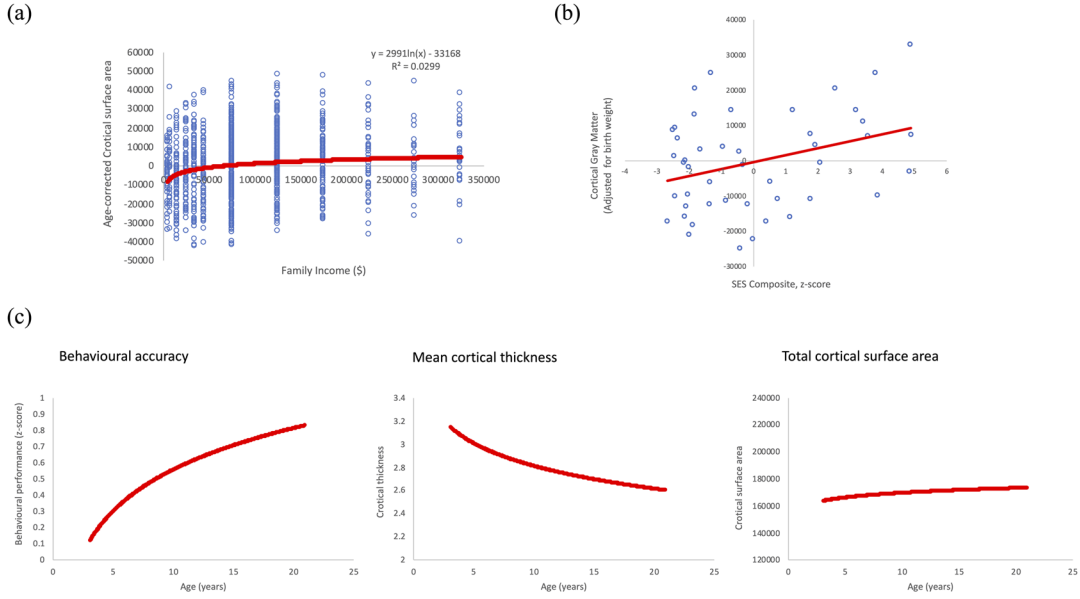

Fig. 1. Illustrative empirical data linking SES measures, brain structure, and behavior: (a) the relationship between an SES metric, family income, and total cortical surface area in a cross-sectional sample of 3- to 20-year-olds. Data show residuals from a function linking age and total cortical surface area in mm^2, plotted against family income ($) (data replotted from Noble et al., 2015); (b) the relationship between an SES composite (income-to-needs ratio and maternal education) and cortical gray matter in a sample of 1-month old infants (data replotted from Betancourt et al., 2016); (c) schematics showing developmental change, including the empirical relationships between age and behavior, age and mean cortical thickness, and age and total cortical surface area (curves estimated from Noble et al., 2015). Behavioral accuracy is in z-scores computed across the whole sample, averaged across cognitive tests of vocabulary, reading, working memory, and selective attention; cortical thickness units are mm; cortical surface area units are mm^2.

First, in terms of age, observations of very early differences in brain structure would support the view that SES impacts prenatal and immediately postnatal brain development, rather that solely arising from postnatal experience-dependent changes (Hurt & Betancourt, 2016). Prenatal effects might occur via environmental influences associated with economic hardship that impact the mother, including chronic stress, poor nutrition, and exposure to toxins, such as alcohol, drugs, and pollutants. Hanson et al. (2013) examined global and regional gray matter volume in 77 children from 5 months to 4 years of age from diverse SES backgrounds, in a dataset including repeated longitudinal scans. The authors reported lower global gray matter volumes in children from lower SES backgrounds. Regional differences were also found in frontal and parietal lobes but not for temporal or occipital lobes, nor for white matter. Lower SES was associated with slower trajectories of brain growth across this age range, such that SES gaps increased with age. There was insufficient statistical power to indicate the age at which SES effects were observed, but SES differences were not accounted for by infant birth weight, the infant's early health, or differences in head size at birth. These data implicate

factors influencing early postnatal brain development. But as we have seen, data from the Betancourt et al. (2016) study implicate even earlier influences.

Second, the way that SES alters developmental trajectories can also be informative about causes. If SES disparities in brain structure widen with age, this may suggest compounding effects of different causes across development. If SES disparities hold constant, it is suggestive of early-acting effects impacting the onset of development but not its subsequent course. If SES disparities narrow with age, it suggests a common developmental pathway that is followed at different rates, leading to eventual catch-up of lower SES groups. Above, we saw evidence that lower SES is associated with slower rates of growth of gray matter in infancy and early childhood such that trajectories diverge (Hanson et al., 2013). Using the same sample studied by Noble et al. (2015), the Pediatric Imaging, Neurocognition and Genetics data set (PING; Jernigan et al., 2016), Piccolo, Merz, He, Sowell, and Noble (2016) examined rates of change of cortical thickness and cortical surface area according to family income and parental education. While cortical thickness showed no main effect of SES factors, these factors moderated patterns of age-related change in cortical thickness but not surface area. Mean cortical thickness reduced more quickly in children from lower-income families, and in a more nonlinear fashion (see also Khundrakpam et al., 2019, and Parker et al., 2017, for similar findings; McDermott et al., 2019, and Judd et al., 2020, for two studies that did not observe this interaction; and Tooley, Bassett, & Mackey, 2021, for discussion).

In adolescents, Ziegler et al. (2020) reported that individuals who had experience early life disadvantage before age 12, based on neighborhood data, exhibited slower growth in myelination in cortical, subcortical, and core white matter regions, and noted that neither IQ at baseline, alcohol use, body mass index, parental occupation nor self-reported parenting quality explained the effects. Parental education did, however, partially predict the effect, thereby implicating differences in cognitive stimulation as a cause. In adults, Kim et al. (2015) found that education level was positively correlated to cortical thickness in a number of regions, an effect that was larger in older adults, and was interpreted as faster cortical thinning in individuals with lower SES.

For the third property, a stronger indication that differences in brain structure are causally related to the association between SES and behavior can be gained by demonstrating that differences in brain structure *mediate* the relationship between SES and behavior. A number of studies have reported such findings, across a range of behaviors. In their sample, Noble et al. (2015) reported that measures of cortical surface area mediated the relationship between two of their four behavioral measures, the flanker and working memory tasks (both tapping executive function skills) and family income, though not vocabulary nor reading; no mediation effects were observed for cortical thickness, nor in an analysis of the integrity of white matter tracts (Ursache & Noble, 2016). In a U.S. sample of 623 participants from 5 to 25 years combining cross-sectional and longitudinal magnetic resonance imaging (MRI) scans, McDermott et al. (2019) observed positive associations between SES (based on parental education and occupation) and total volumes of the brain, cortical sheet, and several subcortical structures. They reported that anatomical variation within a subset of cortical regions partially mediated the positive association observed between SES and intelligence scores. Mediation effects have also been reported in memory

(chronic-stress-related hair cortisol concentrations mediated the association of SES to hippocampal CA3 and dentate gyrus volumes in 5- to 9-year-old children; Merz et al., 2019), and SES to emotion processing and regulation (in uncinate fasciculus connecting orbitofrontal cortex and amygdala, lower fractional anisotropy mediated the relationship between material hardship and internalizing symptoms in 5- to 9-year-old girls).

Mediation is not the only possible causal relationship between SES, brain structure, and cognition. For example, SES might *moderate* the relationship between brain and cognition, such that brain–behavior associations are different at different levels of SES. This relationship has been considered more often in functional brain imaging studies, where it has been used to test the idea that children at different SES levels might employ different cognitive mechanisms or strategies to carry out the same task. For example, in a mathematics task, Demir et al. (2015) found that in higher-SES children, activation in regions associated with verbal cognition tracked performance (e.g., left middle temporal gyrus), while in lower-SES children, performance was tracked by activation in regions associated with spatial cognition (e.g., right intraparietal sulcus).

In the modeling section, we focus on mechanisms within a specific domain, that of language development. This is a useful domain to consider SES effects for several reasons. First, it is a domain where some of the largest SES effects are observed in children's behavior, particularly with respect to vocabulary and phonology (e.g., Hackman & Farah, 2009, 32% of the variance in a language composite for U.S. first graders was explained by SES differences, larger than the effects observed in cognitive conflict or working memory, both 6%). Second, notable differences have been observed in the linguistic environments to which children are exposed in families at different SES levels (e.g., Hart & Risely, 1995), and differences in language experience have been argued to be causal in producing differences in behavioral outcomes (e.g., Brito, 2017; Hoff, 2003). Third, language experience, language skill, and brain structure are linked: Romeo et al. (2018a) tested the hypothesis that SES-related differences in language development stem from variations in the level of language experience and that these in turn produce specific differences in brain structure. Independent of SES, greater levels of adult–child conversational interaction in 4- to 6-year-old children were related to stronger, more coherent white matter connectivity in the left arcuate and superior longitudinal fasciculi, with the structural measure mediating the relationship between conversational turns and children's language skills. Fourth, an emerging literature has reported functional neural correlates of SES-related differences in language and literacy, in domains such as reading, phonological skills, phoneme discrimination, and speech processing, and in brain areas linked to language processing, such as left frontal and left temporal regions (see Farah, 2017, for review). Fifth, despite environmental influences, variation in language skills is partly heritable, with genetic similarity predicting perhaps half the variance (Stromswold, 2001). The exact genetic basis is largely unknown, although it is likely highly polygenic (Newbury et al., 2019); candidate gene variants have been implicated in processes of early neurodevelopment, including neuronal migration and axon growth guidance (Galaburda, LoTurco, Ramus, Fitch, & Rosen, 2006). Sixth, there are proposed cognitive enrichment interventions that seek to narrow SES-related gaps in children's language development, which target parent–child language interactions (see Pace, Luo, Hirsh-Pasek, & Golinkoff,

2017; Romeo et al., 2018b, for discussion; Thirty Million Words initiative for an example: https://cri.uchicago.edu/portfolio/thirty-million-words/).

In sum, the emerging picture is that there may be multiple potential causal pathways for SES to influence brain structure, including prenatal effects on brain development, postnatal effects of nurture and different levels of cognitive stimulation, and postnatal effects of chronic stress linked to economic disadvantage. These have both global and regional effects on brain structure, and regional structural differences can be observed to mediate the relationship between SES and behavior.

Two complications cloud this picture: the possibility that pathways may be different for different children, perhaps dependent on the absolute level of hardship—this would mean empirical findings would be influenced by the particular participant sample included in each study; and the possibility that causal pathways may operate differently on different brain regions. The hippocampus, for example, is particularly vulnerable to the effects of chronic stress (Merz et al., 2020), while brain regions that take longer to mature may be more sensitive to differences in cognitive stimulation. As indexed by gray matter density, frontal and temporal regions have the longest developmental time course (Gogtay et al., 2004), and are also the areas observed to show the strongest effects of SES factors (Noble et al., 2015). Thus, accounts of SES on brain structure may be sample specific or brain-region specific.

2.2. Challenges for causal accounts

In building causal accounts that link SES-associated environmental influences with brain structure and cognition, there are several challenges. Below, we consider two of them: how to map macro brain structure measures to micro function; and how to embed accounts of SES-linked influences within a developmental framework that also incorporates other influences on individual differences, most notably, genetic variation.

2.2.1. Mapping brain structure measures to function

The correlational evidence reviewed in the preceding section requires grounding in a mechanistic account. However, it is not fully known how macro-level measures of brain structure, such as cortical thickness, cortical surface area, gray matter volume, or white matter volume, relate to the micro-level neurophysiological processes that produce function. This is both in terms of *structure affecting function*, where, for example, SES-linked environmental factors might influence early trajectories of brain growth which then impact on functional properties; and in terms of *function affecting structure*, where, for example, SES-related differences in cognitive stimulation might influence functional development, which is then reflected in structural changes. The same challenge arises more broadly in linking general differences in behavior, such as intelligence, to brain structure. Are differences in brain structure the cause of differences in intelligence, or does intelligence partly drive brain differences (Deary et al., 2022). For example, Grasby et al. (2020) exploited polygenic scores for cortical surface area, general cognitive function, and educational attainment in a Mendelian randomization design to investigate the causal relationships between structure and function. They found evidence both for a causal effect of total cortical surface area on general cognitive function and

education attainment (structure affecting function) and evidence for the reverse causal effects of general cognitive function and educational attainment influencing cortical surface area (function affecting structure).

What does developmental change in a given macro brain structure measure represent at the micro level? Johnson and de Haan (2015) argued that cortical thickness and cortical surface area may index the operation of different developmental mechanisms: cortical surface area may reflect the development of cortical columns, while cortical thickness may reflect the development of cells within the columns along with synapse formation, pruning, and myelination. Similarly, Grasby et al. (2020) interpreted genome-wide association data for variation in cortical thickness and cortical surface area as supporting the *radial unit hypothesis*, whereby the surface area is driven by the proliferation of neural progenitor cells during fetal development (evidenced by enrichment of genetic loci active during prenatal cortical development), while thickness is linked to morphological changes after mid-fetal development, such as myelination, branching, or pruning (evidenced by enrichment of active regulatory elements in adult brain samples). Grasby et al. (2020) reported only a small (negative) genetic correlation between cortical surface area and thickness suggesting different genetic influences.

In the same way, developmental changes in gray matter and white matter may be underpinned by a range of different cellular mechanisms. For gray matter, these include neurogenesis, synaptogenesis, synaptic pruning, and changes in neuronal morphology; for the white matter, they include changes in the number of axons, axon diameter, the packing density of fibers, axon branching, axon trajectories, and myelination; extra-neuronal changes include increases in glial cell size and number, and angiogenesis (changes in the vasculature) (Zatorre, Fields, & Johansen-Berg, 2012). Zatorre et al. argued that any of these cellular changes might influence magnetic resonance imaging signals. Meanwhile, there are ongoing methodological debates concerning the interpretation of MRI signals to derive structural measures. For example, cortical thinning has long been argued to be a result of pruning: neural processing is improved by the removal of inefficient synapses, dendrites, and neurons, serving to optimize brain circuits for specific functions (e.g., Huttenlocher, 1979; Petanjek, Judas, Kostović, & Uylings, 2008; Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986). However, Natu et al. (2019) argued that contrary to prevailing theories, cortical tissue does not thin during childhood but instead becomes more myelinated; the appearance of thinning is due to a shift of the gray-white matter boundary up into the cortex. Cortical myelination has been shown to have only a partial overlap with genetic influences on cortical surface area and thickness (Schmitt, Raznahan, Liu, & Neale, 2020).

Clearly, the link from measurements of neural tissue to underpinning neural processing is problematic. At the current time, to construct a model that makes commitments about the functional role of structural indices will require speculation, and bridging concepts that capture in the vocabulary of the functional model what the tissue measurements may represent.

2.2.2. Embedding SES effects in a developmental model which includes genetic influences on brain and behavior

A putative mechanistic explanation that links environmental influences associated with SES, behavior, and brain structure must also accommodate a wide range of other empirical

phenomena on cognitive development, on the origin of individual differences in cognition, on developmental changes in brain structure, and on the relationship between cognitive ability and measures of brain structure. To date, most of these relationships have been observed in global measures, and it remains a matter of conjecture the extent to which they will be reflected in specific neural mechanisms linked to particular behavioral tasks.

Foremost, proximal causal effects of environmental influences associated with SES must be embedded within a developmental framework that explains the origin of changes in behavior and brain structure over time. Moreover, although behavioral skills typically increase across development, this is not the case for all measures of brain structure: some measures increase (white matter volume and cortical surface area) but others decrease following a peak in early or mid-childhood (gray matter volume and cortical thickness) or show complex nonlinear trajectories (Giedd, Blumenthal, Jeffries, Castellanos, & Rapoport, 1999; Schnack et al., 2015; Sowell et al., 2004). For illustration, the broad developmental patterns for behavior, mean cortical thickness, and total cortical surface area are shown in Fig. 1. Some recent accounts of SES effects are couched primarily in developmental terms: Tooley et al. (2021) argued that SES modulates the rate of development, with the stresses of social and economic disadvantage inducing faster brain maturation. The cortex thins over development, and ex hypothesi, the cortex thins more quickly in children from low SES family backgrounds.

While environmental measures associated with SES reliably predict individual differences, a larger proportion of variance in cognitive ability, brain structure, and change in brain structure across development is predicted by the genetic similarity between people—these phenotypes are highly heritable (Brans et al., 2010; Jansen, Mous, White, Posthuma, & Polderman, 2015; Kruggel & Solodkin, 2020; Plomin, DeFries, Knopik, & Neiderhiser, 2013). Outside of genetic syndromes, the genetic influences appear to be *polygenic*, arising from many small contributions from a large number of common genetic variants (e.g., Biton et al., 2020; Cox, Ritchie, Fawns-Ritchie, Tucker-Drob, & Deary, 2019; Davies et al., 2018). The heritability of cognitive ability has been observed to increase across childhood (Davis, Haworth, & Plomin, 2009; Haworth et al., 2010). Although there is regional variability, heritability estimates for measures of brain structure may also increase slightly from childhood to adulthood (Douet, Chang, Cloak, & Ernst, 2014; Jansen et al., 2015). For example, Schmitt et al. (2014) reported an increase in the heritability of cortical thickness throughout late childhood and adolescence. Lastly, heritability levels may be modulated by SES: it has been observed that in individuals from low SES backgrounds, the heritability of IQ can be reduced, though this pattern is not found in all populations (Tucker-Drob & Bates, 2016; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

Larger brains are associated with higher intellectual ability. Meta-analyses suggest correlations of .1–.3 between brain volume and IQ (Deary et al., 2022; McDaniel, 2005). Relationships between individual differences in general cognition and global brain structure measures are observable both in gray matter volumes and white matter volumes, and brain–cognition relationships appear to be underpinned by common genetic influences (e.g., Posthuma et al., 2003). Ritchie et al. (2015) found that brain volume explained 11.3% of the variance in general cognitive ability, cortical thickness another 4.7%, white matter volume an additional

1.3%, and all structural measures together 18.4% of the variance. Thus, genetic influences on intelligence in part operate via influences on brain structure. For example, Lett et al. (2020) found that cortical surface area mediated the relationship between a polygenic score for intelligence and general intelligence levels. Nevertheless, genetic data suggest that influences on the brain act, by and large, on general properties of neural computation (neurogenesis, the synapse, postsynaptic density, neuron differentiation, and oligodendrocyte differentiation); their effects on behavior are then instantiated through the operation of specific structures in functional circuits (Deary et al., 2022; Grasby et al., 2020; Plomin, Kovas, & Haworth, 2007).

IQ is also related to the rate of change of the cortex with age (Schnack et al., 2015; Shaw et al., 2006), and white matter maturation profiles (Deoni et al., 2016). Shaw et al. (2006) found that higher IQ is associated with faster thickening of the cortex across early childhood, and then faster thinning of the cortex from mid-childhood onward. It is noteworthy that the latter finding is not obviously consistent with associations between SES and cortical thinning, where lower SES was associated with faster thinning of the cortex (Piccolo et al., 2016). Can faster thinning be a property both of higher IQ and lower SES, when higher IQ is typically associated with *higher* SES (von Stumm & Plomin, 2015)?

Lastly, SES-related and genetic influences on brain and behavior may have complex and sometimes nuanced relationships. Although there is evidence that SES-related and genetic influences (the latter, for instance, as indexed by a polygenic score for educational attainment) may have independent contributions to variation in brain structure (Judd et al., 2020; Paul et al., 2021; Raffington et al., 2019), these studies and others indicate that the two influences may be correlated. This may occur either through population stratification or other forms of gene–environment correlation whereby, for example, children inherit genes from their parents and also experience the nurturing environment of their parents (itself influenced by the parents' genetics). Allegrini et al. (2020) explored environmental and genetic predictors of educational achievement and found both independent contributions but also strong correlations between them, such that they estimated 40% of the polygenic scores' effects on achievement were mediated by environmental effects, and in turn that 18% of environmental effects were accounted for by the polygenic model. Similarly, Belsky et al. (2018) observed that children who achieved greater educational success than their parents, a marker of upward social mobility, had higher polygenic scores for education, indicating that genetic variation influences characteristics that foster success, yet they still observed that such children also tended to come from better-off families with potentially enriched environments.

We have, then, a range of empirical effects at different levels of description (behavior, brain, genes, and environment) which represent the minimal set of findings that ultimately need to be captured by a mechanistic account of SES-linked environmental influences. These are summarized in Table 1. Our goal in the rest of this paper is to explore the contribution of formal computational models for advancing causal accounts that could in principle link these data sets, as well as the initial simplifications and narrowing of scope necessary to construct such models.

Table 1
Preliminary list of empirical phenomena relating SES-related environmental influences, behavior, brain structure, and genetic variation (see text for references)

	Phenomenon
1.	Behavioral accuracy increases across development
2.	Some measures of brain structure increase across development (white matter and cortical surface area)
3.	Some measures of brain structure reduce across development (gray matter and cortical thickness)
4. and 5.	(a) Lower SES is associated with lower IQ and (b) SES gaps widen across development
6. and 7.	(a) Lower SES is associated with reduced cortical surface area in a graded but nonlinear fashion, with larger effects at lowest SES levels; (b) smaller SES effects are seen on cortical thickness
8.	Lower SES may be associated with faster thinning of the cortex over development
9.	Lower SES does not modulate the rate of development of cortical surface area
10. and 11.	(a) Cortical surface area partially mediates the relationship between SES and behavior; (b) smaller mediation effects observed for cortical thickness
12.	Individual differences in behavior and brain structure are highly heritable
13.	Genetic influences are highly polygenic
14.	Genetic influences on behavior and brain structure are partially overlapping
15.	Genetic influences on intelligence increase across childhood
16.	Low SES can reduce the heritability of IQ
17.	Brain volume correlates with IQ
18. and 19.	Across development, (a) higher IQ is associated with faster thickening and then (b) faster thinning of the cortex

Note: Abbreviations: IQ, intelligence quotient; SES, socioeconomic status.

3. A simple multiscale computational model of SES-associated environmental influences to explore links between levels of description

The effects of adversity on the development of the brain and cognitive system are complex and system-wide (Boyce et al., 2021). A great deal of simplification is necessary to construct a computational model. These simplifications are of two sorts. First, indices of SES, such as family income, parental education, and neighborhood deprivation, are distal measures, and a model of cognitive process must make a commitment to the proximal factors that produce effects on development. The model then becomes one of the environmental influences that are correlated with SES. Nevertheless, implemented models provide the advantage of forcing clarity through specification, testing the adequacy of putative causal pathways to capture empirical data, and generating novel predictions.

Second, most of the findings reviewed in the previous section are general or global in nature, such as SES-linked effects on intelligence, or correlations between SES and brain volume or total cortical surface area. An implemented model requires a specific mechanism delivering a target behavior. Although as we saw, the link between global relationships and specific associations usually proceeds via the assumption that global properties are reflected in the operation of specific structures in functional circuits (Kovas & Plomin, 2006), nevertheless, this assumption is speculative and ultimately a hypothesis to be tested empirically. The following simulation work focuses on a narrow domain within language development and environmental/genetic predictors of its development, before extrapolating to global associations.

We chose language development as the domain for the model because there is strong evidence of associations with SES measures, as well as suggestive evidence of the pathways through which influences operate. We employed an ANN model drawn from one area of language acquisition, English past tense formation. This is a well-characterized domain, where there is a simple but well-established cognitive-level model which has already been applied to simulating behavioral data on SES effects on children's language acquisition at a population level (Thomas, Ronald, & Forrester, 2013). Our interest is in evaluating assumptions that might link properties of the ANN to structural brain measures. Past tense formation involves temporal and frontal brain areas processing phonology that are known to be influenced by SES factors (Joanisse & Seidenberg, 2005). Although there have been theoretical debates concerned with how children learn the past tense of regularly versus irregularly inflected verbs (e.g., Pinker, 1994), functional brain imaging data point only to the importance of phonological factors driving differences in activation across verb types (Joanisse & Seidenberg, 2005), and here, we only consider the acquisition of regular past tense formation as our target behavior.

To build the simple multiscale model, we added both neurobiological assumptions and assumptions about how genetic influences impact neurocomputation. The additional neurobiological assumptions distinguished three developmental processes: (1) network growth; (2) activity-dependent change in a network of integrate-and-fire neurons; and (3) a phase of connectivity pruning/optimization. Within simulated populations, two sources of variation were distinguished: variation across a range of neurocomputational parameters, capturing

polygenic influences and environmental influences on network growth, activation, and plasticity; and variable levels of environmental stimulation.

The main goal for these simulations was to contrast two of the putative causal pathways of SES-related environmental influences on brain structure and cognitive development: prenatal effects on brain development, which we assumed would impact on the growth of the network; and postnatal effects of cognitive stimulation, which we assumed would impact experience-dependent change. We further considered the combined effects of both influences. Multiscale models require the stipulation of assumptions at each level of description, to which we now turn.

3.1. Multiscale assumptions

In ANN models of cognitive development, abstract principles of neurocomputation are embodied in systems whose activation states correspond to concepts and whose inputs and outputs can be linked to behavior (see, e.g., Thomas & McClelland, 2023). The accuracy of *behavior* is, therefore, measured by the activation states that represent the acquisition of input–output mappings.

Thomas (2016) argued that with a simple addition—the onset of pruning of unused connectivity after a certain point in training—these models could give plausible analogs to measures of *brain structure*. The total number of connections would serve as an analog to properties that decrease over development (gray matter and cortical thickness) under the view that unused connections are pruned away, causing a loss of volume. This measure can be captured in the idea of *resources for connectivity*. The combined magnitude of connection weights (excitatory and inhibitory) would serve as an analog of properties showing increases (white matter and cortical surface area) under the view that retained connections are optimized through myelination, causing an increase in volume. This measure can be captured by the idea of *operational connectivity*. We used the same scheme here.

An ANN trained with backpropagation has limited biological plausibility, a point to which we will return. Nevertheless, the model contained (and, therefore, tested) a key set of assumptions relating measures of network structure to measures of brain structure. They were as follows: (1) postnatally, neuron number is fixed so that changes in structure then reflect changes in connectivity; (2) structural measures that increase over development (cortical surface area and white matter) reflect increases in connection strength, while structural measures that decrease over development (cortical thickness and gray matter) reflect reductions in connection number; (3) connection strength increases can only be experience-dependent; (4) connection strength decreases can be experience-dependent (training reduces some connections), intrinsic (weight decay), or both (an intrinsic pruning process operates depending on connection strengths which in turn are influenced by experience); (5) connection number is intrinsic (growth) or an interaction with experience (pruning); and (6) we did not include an assumption that growth in connection number might be partly experience/environment dependent, nor that there might be intrinsic contributions to connection strengthening (e.g., myelination occurring through maturation). The adequacy of the model in capturing the patterns of empirical data serves as a test of these assumptions. (To foreshadow our results, we will conclude

that we do not think all of the assumptions are correct). In mapping the model to the empirical data, we initially work on the assumption that changes in the implemented phonological mechanism would scale to produce the kind of regional changes observed in language-related temporal cortical gray matter, of the sort reported by Noble et al. (2015)—correlating with family income and parental education—and language-related white matter pathways, of the sort reported by Romeo et al. (2018a)—correlating with language exposure.

To capture *genetic influences* on behavior and structure, each ANN must have a genome, and genomes must vary between individuals. To the extent that cognition is construed as information processing in the brain, genetic effects on cognition must unpack as influences on neurocomputational properties. Accordingly, Thomas, Forrester, and Ronald (2016) used a method to simulate individual differences where the neurocomputational parameters of the ANN (e.g., number of hidden units and learning rate) were encoded in an artificial genome (details below). Genetic variation produced parameter variation (a larger, unimplemented set of genes was assumed to underly the operation of invariant, species-universal processes involved in cellular and neural function). In behavior genetics, the heritability of a phenotype such as behavior or brain structure is often assessed using the twin design, where more heritable phenotypes show greater similarity between monozygotic (MZ) twins than dizygotic (DZ) twins. MZ twin ANNs can be simulated by creating networks that share the same genome (and, therefore, parameters), while DZ twins can be simulated by networks that share on average 50% of the gene variants in their genomes (see Thomas et al., 2016, for further details). Heritability of behavior and brain structure can then be simulated by comparing the respective correlations between MZ twin networks versus DZ twin networks. This scheme can be viewed as addressing the complexity of how genes effect function in the following ways: genetic effects are taken to act on neurocomputational properties; variations in neurocomputational properties are influenced by the action of many low-level modeled genes, reflecting polygenic coding; effects on behavior are the consequence of the interaction of small variations in multiple neurocomputational properties; modeled genomes allow the encoding of patterns of relatedness. It does not reflect biological reality in terms of cellular processes or DNA; nor in terms of indirect effects in a wider system, such as motivation or health; nor in terms of active or passive gene–environment correlations.

The *environment* with respect to the single mechanism corresponds to the sequences of learning events to which it is exposed that induce activation states in the system, each amounting to an input–output mapping drawn from the ANNs training set—in this case, the full set of phonologically encoded English verb stems and their past tense forms.

SES-linked environmental influences can plausibly be implemented in ANNs in several ways (Thomas et al., 2013). They might correspond to proximal environmental influences impacting initial network growth (equivalent to prenatal effects on brain development); or how the network is maintained (equivalent to postnatal effects such as stress); they might influence the information on which the network is trained (equivalent to differences in levels of cognitive stimulation during postnatal development, or in a reinforcement learning framework, schedule of rewards); or they might influence all these factors. In the following simulations, we contrasted three hypotheses: (1) the relevant environmental influence impacts initial network growth; (2) the relevant environmental influence impacts the level of network

training; and (3) influences impact both factors in a correlated manner. Note that although we implemented both genetic and SES-linked influences, in the current model these were implemented as operating independently.

3.2. Method

3.2.1. Network architecture

The base model was a three-layer backpropagation network, with 57 inputs and 62 outputs. Network growth, in terms of the number and size of initial connections, was modeled as the outcome of the interaction of four parameters which varied across individuals: the number of weight layers, the number of hidden units in the internal layer, the sparseness of connectivity, and range of initial random weight variation. Connection pruning occurred after a specified training epoch, and removed any connections below a specified threshold with a specified probability. Each of these three parameters was also free to vary between individuals. Pruning onset varied between 0 epochs and 1000 epochs, where 1000 epochs of training represented the full lifetime (median value 100 epochs); pruning threshold varied between a magnitude of 0.1 and 1.5 (median 0.5); pruning probability varied between 0 and 1 (median 0.05) per pattern presentation.

Overall, 14 neurocomputational parameters were free to vary between individuals. These were: the architecture (fully connected or three-layer), number of hidden units, sparseness of connectivity, sigmoid activation function temperature, activation noise added to unit net inputs, nearest neighbor output threshold, learning rate, backpropagation error measure (root mean square or cross-entropy), momentum, initial weight variance, weight decay, pruning onset epoch, pruning threshold, and pruning probability (see Thomas, 2016, for parameter specifications, and range of values, for the "GWEW" condition). The majority of these parameters have been varied in other ANN models of cognition as possible neurocomputational explanations of cognitive variability, including general intelligence and disorders, such as developmental language disorder, dyslexia, autism, and schizophrenia (see Supplementary Material).

3.2.2. Training set

The training set comprised 508 artificial monosyllabic verbs, constructed using consonant–vowel templates and the phoneme set of English. Phonemes were represented over 19 binary articulatory features (the format of the phonological representations is shown in Thomas, 2018, Fig. 3). The verbs conformed to the past-tense patterns observed in English, with 410 regular verbs (forming the past tense via the +ed rule) and 98 irregular verbs of three types, no-change, vowel-change, and arbitrary (see Thomas et al., 2016, for more details). Training used pattern presentation in random order without replacement. One epoch of training corresponded to a single presentation of each pattern in the network's training set.

3.2.3. Implementation of behavior

Behavior was assessed by measuring the proportion of regular verb past tense forms that were correctly output on each epoch. Regular verbs were the majority mapping type in the training set. Irregular verbs and generalization were not considered in these simulations.

3.2.4. Implementation of brain structure measures

On each epoch, the total number of connections was measured in each network, along with the total magnitude of connections (ignoring whether weights were positive or negative).

3.2.5. Implementation of SES-linked environmental differences

Each simulated child was raised in a family with a given assigned SES. In the *Stimulation* condition, a parameter called family quotient was sampled uniformly between the range 0 and 1. This proportion was applied as a one-time filter on the full training set. A network raised in a family with a family quotient of 0.75 would be exposed to a training set with around 75% of the training patterns. With a range between 0 and 1 (mean .50), networks could in principle be exposed to very few training patterns (see Thomas, 2016, for discussion). In the model's original domain of application, this corresponds to raising a child in a family with a certain level of language stimulation, observed to be correlated to the family's SES (Hart & Risley, 1995; Romeo et al., 2018b). In the Stimulation condition, the SES-associated influence was indexed by the family quotient parameter.

In the *Structure* condition, SES was implemented as a difference in the number of hidden units in the internal layer, under the view that prenatal and early postnatal effects might impact gray matter development and be influenced by factors associated with SES, such as differences in nutrition and stress (Betancourt et al., 2016; Hanson et al., 2013). Values were chosen at 10, 15, 20, 30, 40, 50, 75, 100, and 200 units and were approximately equally represented in the population. Both siblings in a family were assigned the same hidden unit value. The range of values, with greater frequency of lower values, was chosen because there is an asymmetric relationship between hidden unit number and performance: reductions below sufficient numbers produce a faster decline in performance compared to the increases achieved above sufficient numbers. The intention was, therefore, a uniform distribution of performance across the SES range rather than hidden unit value. Results from pilot studies used to calibrate the range of variation for hidden unit numbers and for family quotient are included in Supplementary Materials (Figs. S1 and S2). Calibration aimed to ensure the same mean performance of each population. The family quotient values were sampled over a similar range to the Stimulation condition but were assumed to be a form of environmental variation uncorrelated to SES. In the Structure condition, the SES-associated influence was indexed by a hidden unit number.

In the *Stimulation+structure* condition, the training sets from the Stimulation condition were placed in rank order of family quotient. The networks from the Structure condition were placed in rank order of the number of hidden units. The ranked training sets were then linked to corresponding ranked families, so that the level of stimulation and the amount of network structure correlated (the correlation was $r = .866$). In this condition, the SES-associated influence was measured by the average of the family quotient and the hidden unit value normed between 0 and 1.

3.2.6. Implementation of genetic differences

Our main assumption was that genetic influences on behavior would occur in part via variations in neurocomputational parameters, and the similarity of these parameters between

individuals should depend on their genetic relatedness. The 14 parameters were simultaneously varied across individual networks, with learning ability determined by their cumulative effect. Thomas et al. (2016) showed how these parameters could be encoded in an artificial genome under a polygenic coding scheme. The population distribution in learning ability was simulated by the accumulation of many small influences from different neurocomputational parameters, themselves influenced by multiple genes, ensuring a polygenic relationship between genes and behavior. Individual gene–behavior correlations were of very small effect size (Thomas et al., 2016). Artificial genes were binary, and set randomly to 0 or 1 to create a population. Sets of genes were allocated together to determine the value of each neurocomputational parameter, the number of genes depending on how many values each parameter could take in the population. The artificial genome had a total of 126 bits as follows—hidden units: 10; temperature: 10; noise: 8; learning rate: 12; momentum: 8; weight variance: 8; architecture: 6; learning algorithm: 4; nearest neighbor threshold: 10; pruning onset epoch: 10; pruning probability: 8; pruning threshold: 10; weight decay: 10; and sparseness: 12. A lookup table was used to derive the parameter value. Artificial genes independently influenced each parameter, implementing polygenicity but no pleiotropy. For the Structure and Stimulation+structure conditions, the hidden unit value was not specified according to genotype, but set separately according to environmental influence. This exception notwithstanding, all variation in these parameters was considered to be under genetic control. For Stimulation and Structure conditions, there was a random association of family quotient to genotype, that is, no gene–environment correlation.

A population of parents was created with random binary genomes. Pairs of parents were used to generate two siblings with the given level of relatedness, either 100% for MZ twins or 50% for DZ twins, using sexual reproduction (see Thomas, 2016, for further details). MZ and DZ network pairs then permitted the assessment of the heritability of individual differences in behavioral and brain measures.

3.2.7. Simulation design

For the Stimulation and Structure populations, 1000 networks were created in sets of pairs, either MZ (250 pairs) or DZ twins (250 pairs). The Stimulation+structure population was created by linking the training sets of the Stimulation condition with the parameter sets of the Structure condition. Twin pairs were trained using the same training set, to capture siblings being raised in the same family. Each network was trained for 1000 epochs. On each epoch of training, behavior (regular verb accuracy) and the two network measures, the total number of connections and magnitude of connections, were assessed. Spreadsheets with full parameter sets and the dependent variables for the three populations are available at https://doi.org/10.18743/DATA.00000215.

3.3. Results

We begin by considering developmental trajectories across age for simulated behavior and brain structure measures. We then consider how simulated SES-linked influences and ability modulate these measures, first cross-sectionally, and then developmentally. We address

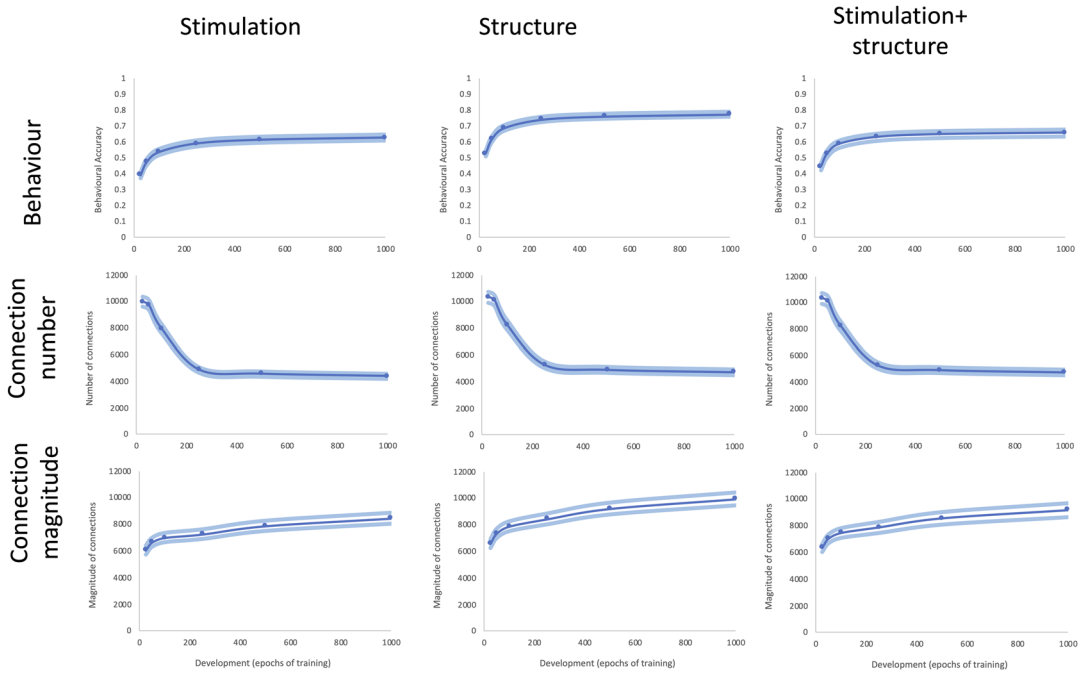

Fig. 2. Simulated relationship between development (epochs of training) and behavior for the three conditions, Stimulation, Structure, and Stimulation+structure (top row); and between development and the two metrics of network structure, total number of connections (the cortical thickness analog) and total magnitude of connection strength (cortical surface area analog) (middle and bottom rows). Shaded lines show 95% confidence intervals around the mean. (Data points reflect population performance at 25, 50, 100, 250, 500, and 1000 epochs of training, chosen as representative points of developmental change.)

whether simulated brain structure measures mediated the relationship between SES-linked influences and behavior, and also test for moderation effects. Finally, we consider the heritability of individual differences for simulated behavior and brain structure markers, and how these are altered by simulated SES level and by development. In each case, we initially summarize the target empirical data and judge the model's qualitative fit to the data.

3.3.1. Development

Fig. 1c depicts developmental trajectories for behavioral accuracy, cortical thickness, and cortical surface area data (estimated from Noble et al., 2015 cross-sectional sample). All trajectories are nonlinear; behavior and cortical surface area increase monotonically, while cortical thickness decreases. Fig. 2 shows equivalent trajectories for the three simulation conditions for the full lifespan of the networks. The first third of these trajectories would map approximately to the data in Fig. 1. On average, each simulated population received 50% of the training set, so behavioral accuracy levels were at approximately this level. All simulations exhibited a nonlinear increase in behavior. Connection magnitude demonstrated a slower, more linear increase with age, in line with cortical surface area.

Table 2
Linear correlations between simulated behavior, SES, connection number, and connection magnitude at a given point in development (100 epochs of training)

		SES	Connection number	Connection magnitude
Stimulation	Behavior	.579	.232	.235
	SES		−.008*	.153
	Connection number			.686
		SES	Connection number	Connection magnitude
Structure	Behavior	.293	.290	.272
	SES		.823	.604
	Connection number			.692
		SES	Connection number	Connection magnitude
Stimulation +structure	Behavior	.725	.528	.444
	SES		.837	.709
	Connection number			.746

* Not significant. All other correlations $p<.01$.

Connection total showed a nonlinear decline, with a decreasing gradient across age, in line with cortical thickness. Simulation data showed greater variation around the population mean trajectory than the human data, such that the log curve fits explained less of the variance (see Supplementary Material for regressions; Fig. S3 for frequency distributions). Qualitatively, all the simulation conditions captured the developmental trajectories of the three dependent variables.

3.3.2. Cross-sectional SES effects

Fig. 1a shows one example of the relationship between SES-linked influences (indexed by family income) and cortical surface area (replotted from Noble et al., 2015). In Noble et al.'s cross-sectional data, both age-corrected cortical thickness and cortical surface area showed a small logarithmic relationship with family income (3% of the variance), with stronger effects at the lowest levels of income, and a slightly weaker linear relationship with parental education. Higher parental education and family income were each associated with *higher behavioral scores, thicker cortex, and greater cortical surface area*. Fig. 1b shows another example relationship, this time between an SES composite and cortical gray matter in 1-month-old infants (replotted from Betancourt et al., 2016). Higher SES was associated with more gray matter. The effects were larger (around 8% of the variance) and linear in nature.

Turning to the model, Table 2 shows a linear correlation matrix between simulated SES-linked effects and behavior, connection total, and connection magnitude, respectively (in the following figures and tables, SES-linked environmental influences are abbreviated to "SES" as an independent variable). Relationships were derived at a single fixed point in training of 100 epochs. (Fig. S4 shows how the linear correlations from Table 2 altered across development.) The Stimulation condition showed larger SES-linked effects on

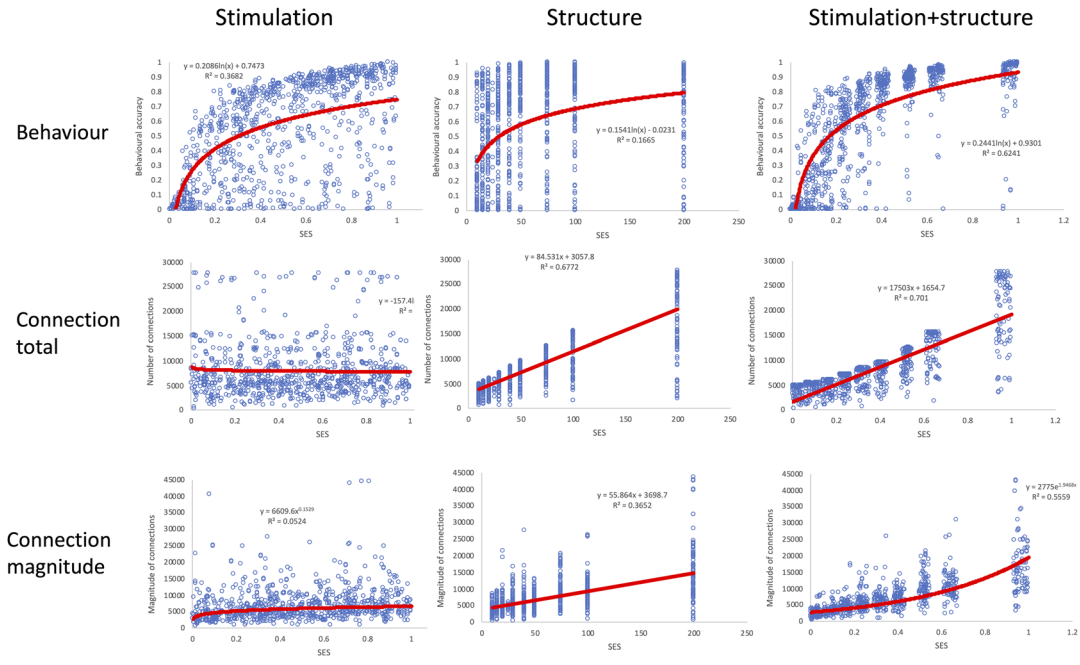

Fig. 3. The relationship between the SES measure and behavior, connection number, and connection magnitude, for the three simulation conditions implementing SES via Stimulation, Structure, and Stimulation+structure. These associations were derived at a single point in development at 100 epochs of training. Fitted functions show the best fit (highest R^2) from the following functions: linear, logarithmic, exponential, and power.

behavior than on connection measures, and larger effects on connection magnitude (the analog to surface area) than connection number (the analog to thickness), in line with the empirical data. For the Structure condition, correlations were larger for network structure measures than behavior, and larger for connection number than magnitude. The Stimulation+structure condition showed large and comparable correlations for both behavior and connection measures. Across development, SES-linked effects operating via stimulation tended to make correlations increase. For SES-linked effects operating via structure, the correlation between SES and behavior increased but those with structure decreased. Despite these differences, correlations all tended to be positive: higher SES was associated with higher behavioral scores, higher connection number, and greater connection magnitude, as with the empirical data. However, effects tended to be stronger than those observed in the empirical data.

Turning to the shape of the relationships, Fig. 3 shows simulation data depicting the best-fit functions linking SES and behavior, connection number, and connection magnitude, respectively, from the set of linear, logarithmic, exponential, and power. For behavior, all conditions showed logarithmic relationships between SES and behavior, with stronger effects of SES at lower SES levels. For connection number, the Stimulation condition showed no effect of SES ($p = .806$), while Structure and Stimulation+structure both showed linear effects. For

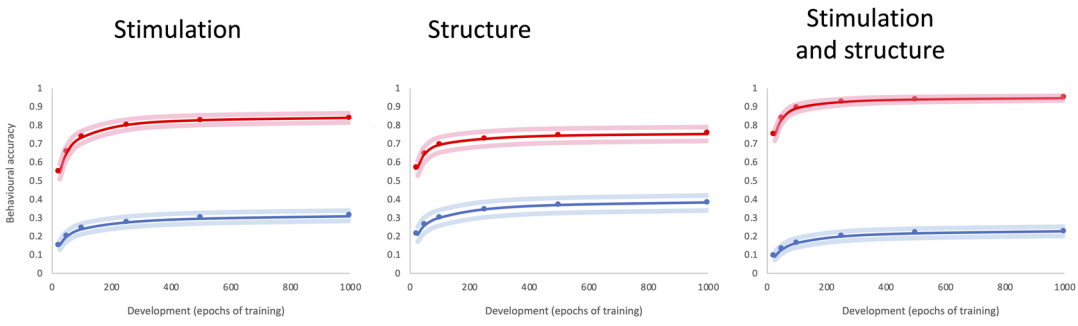

Fig. 4. The effect of simulated SES-linked influences on behavioral development for each condition. Trajectories plot the top (red) and bottom (blue) quartiles of SES. Shaded lines show 95% confidence intervals around the mean. Gaps reliably widen across development in all cases.

connection magnitude, Stimulation exhibited a logarithmic relationship, while for Structure, the best-fit relationship was linear, and for the combination of Stimulation+structure, an exponential growth was a better fit.

Overall, in terms of shape, the Stimulation condition gave the best fit to the empirical data for older children and family income (Noble et al., 2015), capturing small logarithmic effects on behavior and cortical surface area, and smaller effects on cortical thickness. By contrast, the Structure condition more closely resembled the empirical data for 1-month-olds (Betancourt et al., 2016), exhibiting larger linear effects of SES on connection number (under the resources-for-connectivity bridging assumption, the analog of gray matter); but as with the Betancourt results, smaller effects on connection magnitude (the analog of white matter).

3.3.3. SES-linked effects on developmental trajectories

The principal reported empirical effects here are early effects of SES on behavior, gaps which widen across development (von Stumm & Plomin, 2015); faster and more nonlinear thinning of cortex at lower levels of SES in some brain regions, such as left fusiform gyrus, and for average cortical thickness overall; but other regions such as left superior temporal gyrus demonstrating steeper age-related decreases in cortical thickness at higher levels of SES; and SES not altering the rate of change of cortical surface area (Piccolo et al., 2016).

Fig. 4 depicts the simulated developmental trajectories for behavior, for clarity showing the highest and lowest quartiles of SES (trajectories are shown for behavioral accuracy; trajectories for IQ-like standard scores are shown in Fig. S5). Gaps reliably widened across development in the conditions where stimulation contributed to SES differences but not Structure alone. To quantify the size of the interaction compared to the main effects of age and SES, ANCOVAs were performed on log-log transformed data with SES as a continuous variable. The SES main effect sizes for Stimulation, Structure, and Stimulation+structure, respectively, were $\eta_p^2 = .006$, $\eta_p^2 = .003$, and $\eta_p^2 = .006$, epochs $\times$ SES interactions were $\eta_p^2 = .001$ $p<.001$, and $\eta_p^2 = .003$ $p<.004$ for Stimulation and Stimulation+structure, but $\eta_p^2 = .000$

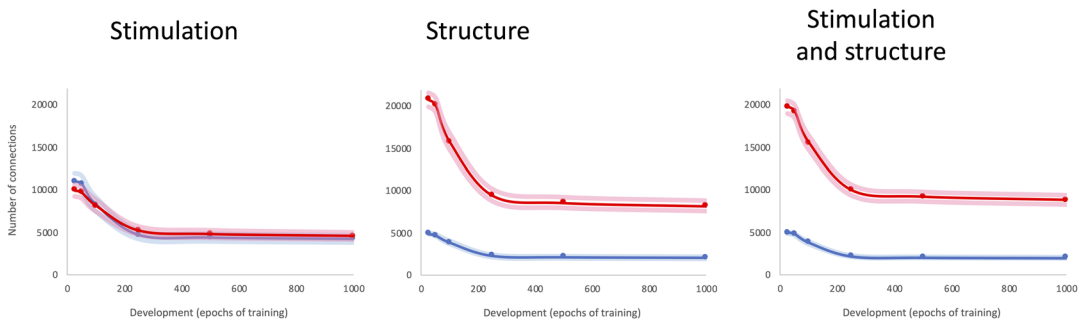

Fig. 5. The effect of simulated SES-linked influences on developmental trajectories of connection number for each condition. Trajectories plot the top (red) and bottom (blue) quartiles of SES. Shaded lines show 95% confidence intervals around the mean.

$p = .698$ for Structure alone. Different levels of stimulation, therefore, seemed sufficient to drive the developmental widening of behavioral gaps.

Fig. 5 shows the effect of SES-linked environmental influences on the trajectories of connection number. SES, by definition, had much larger effects on connection number in the Structure and Stimulation+structure conditions, though it was reliable for all conditions (effect sizes of $\eta_p{}^2 = .001$, $\eta_p{}^2 = .083$, $\eta_p{}^2 = .069$, for Stimulation, Structure, and Stimulation+structure). In the Stimulation condition, the connection number reduced at a faster rate in the lower SES individuals than in higher SES (epochs × SES interaction: $F(1,5996) = 16.09$, $p<.001$, $\eta_p{}^2 = .003$). For both Structure and Stimulation+structure, the reverse was the case; higher SES individuals showed faster rates of decline ($\eta_p{}^2 = .003$, $p<.001$, $\eta_p{}^2 = .002$, $p = .001$). Comparing top and bottom quartiles of SES between 25 and 250 epochs, the average connection loss for Stimulation was 4728 for high SES versus 6311 for low SES; for Structure was 11,302 for high SES versus 2597 for low SES; and for Stimulation+structure was 9792 for high SES versus 2773 for low SES. The Stimulation condition alone, then, showed faster connection loss for lower SES individuals, the global effect reported by Piccolo et al. (2016).

Fig. 6 depicts the effect of SES-linked environmental influences on trajectories of connection magnitude. Again, by definition, the SES effects on connection magnitude were much larger in the Structure and Stimulation+structure conditions, and indeed were not present as a main effect in the Stimulation condition (effect sizes of $\eta_p{}^2 = .000$, $\eta_p{}^2 = .049$, $\eta_p{}^2 = .072$). For the Stimulation and Structure conditions, the effect of SES on connection magnitude increased across development ($\eta_p{}^2 = .007$, $\eta_p{}^2 = .008$), but did not reach significance for Stimulation+structure ($p = .080$, $\eta_p{}^2 = .001$). The Stimulation condition showed no overall increase in connection magnitude across development for the bottom SES quartile (logarithmic function, $R^2 = .000$, $p = .739$; a reliable increase was observed for the top quartile $R^2 = .039$, $p<.001$)—that is, at low levels of cognitive stimulation, increases in the strengths of individual weights through training were balanced by the loss of weights through pruning and weight decay. By contrast, in the Structure and Stimulation+structure conditions, the lowest quartile did show reliable increases across development ($R^2 = .078$, $p<.001$;

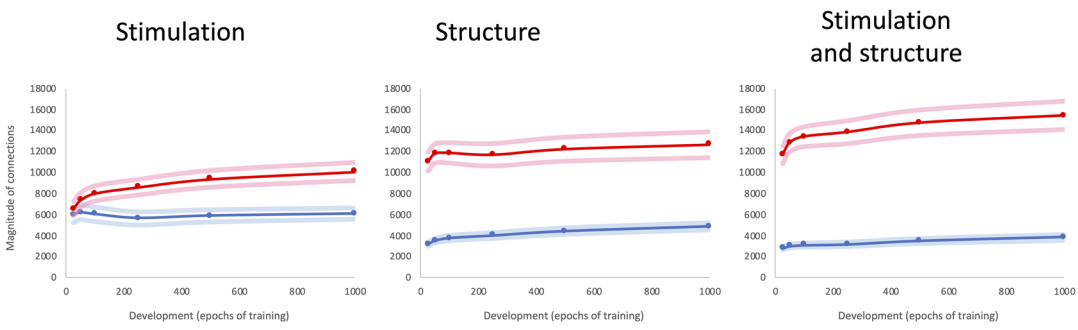

Fig. 6. The effect of simulated SES-linked influences on developmental trajectories of connection magnitude for each condition. Trajectories plot the top (red) and bottom (blue) quartiles of SES. Shaded lines show 95% confidence intervals around the mean.

$R^2 = .034$, $p<.001$). The Stimulation condition, then, showed weaker effects of SES on connection magnitude compared to conditions affecting the structure, but produced a stronger divergence in trajectories across development because of the flat profile of lower SES individuals. The parallel developmental trajectories in the cortical surface area reported by Piccolo et al. (2016) were only captured by the Stimulation+structure condition.

3.3.4. Associations between behavioral ability and developmental trajectories of network structure

In this section, we consider networks not by SES but by each individual's behavioural performance, irrespective of how it arose. The principal global empirical effects here are positive correlations between cognitive ability and brain size (including gray matter volume and white matter structure) (Deary et al., 2022; McDaniel, 2005; Ritchie et al., 2015); and differential rates of developmental change for cortical thickness and surface area: faster thickening and then thinning of the cortex from mid-childhood onward for higher ability children (Shaw et al., 2006); larger cortical surface area in more able children and earlier plateauing in adolescence (Schnack et al., 2015). Again, for the simulation to make contact with these data, we need to assume that such global effects would be reflected in the development of a single cognitive mechanism.

To assess behavioral ability in the model, we measured past tense accuracy in mid-development at 100 epochs. Viewed as a main effect on (log-log transformed) developmental trajectories, the effect sizes of ability on behavior were $\eta_p{}^2 = .047$, $\eta_p{}^2 = .029$, and $\eta_p{}^2 = .014$, respectively. Table 2 shows that at 100 epochs of training, more able networks had higher indices of structure, both in terms of the number and magnitude of connections. Fig. 7 displays developmental changes in connection number split by behavioral ability, while Fig. 8 shows the equivalent for connection magnitude.

Connection number tended to be higher in more able networks. The outcome of the growth phase of connectivity was modeled by a combination of parameters that could vary across individuals (sparseness of connectivity, architecture, number of hidden units, and the initial variance of weight randomization). At the onset of training, the correlations between

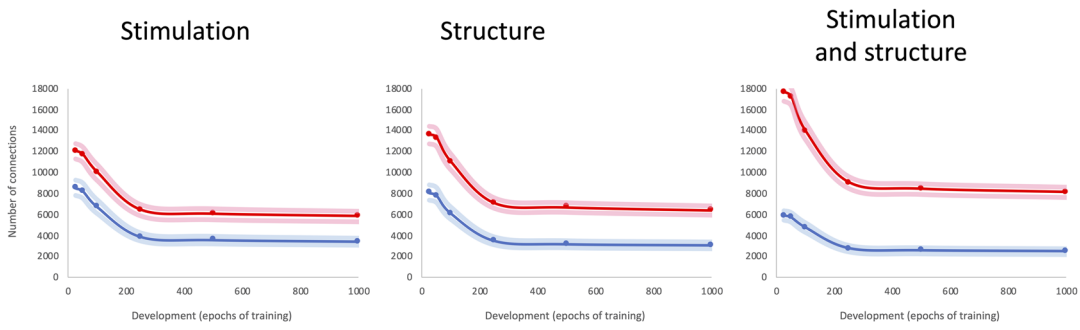

Fig. 7. The effect of behavioral ability on developmental trajectories of connection number for each condition. Trajectories plot the top (red) and bottom (blue) quartiles of behavioral ability, measured at 100 epochs of training. Shaded lines show 95% confidence intervals around the mean.

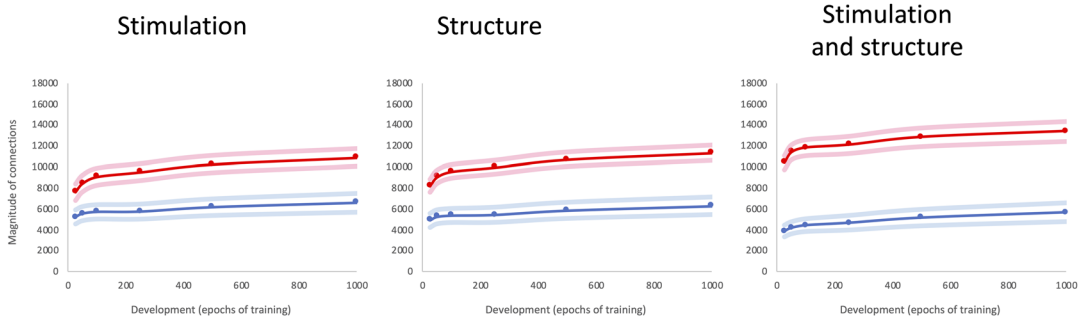

Fig. 8. The effect of ability on developmental trajectories of connection magnitude for each condition. Trajectories plot the top (red) and bottom (blue) quartiles of behavioral ability, measured at 100 epochs of training. Shaded lines show 95% confidence intervals around the mean.

connection number and later ability at 100 epochs were .242, .290, and .595 for the three conditions, respectively (the latter exaggerated by stimulation levels across the 100 epochs being correlated with initial structural differences). All conditions, therefore, captured the pattern that faster network growth was associated with higher behavioral ability. Analyzed across development, neither Stimulation nor Structure conditions exhibited a main effect of ability on connection number (Stimulation: $F(1,5996) = 1.80$, $p = .180$, $\eta_p^2 = .000$; Structure: $F(1,5996) = 2.31$, $p = .128$, $\eta_p^2 = .000$). The combination of influences did produce a reliable main effect of ability in Stimulation+structure ($F(1,5996) = 93.77$, $p = <.001$, $\eta_p^2 = .015$). In all conditions, however, there was a faster decline in connection number for more able networks (epoch × ability: $\eta_p^2 = .005$, $\eta_p^2 = .007$, $\eta_p^2 = .005$). For example, comparing the top and bottom ability quartiles between 25 and 250 epochs, the average connection loss for Stimulation was 5617 for high ability versus 4648 for low ability, for Structure was 6442 versus 4593, and for Stimulation+structure was 8610 versus 3136. All conditions, therefore, captured the pattern of faster growth and faster thinning for individuals who had the higher behavioral ability.

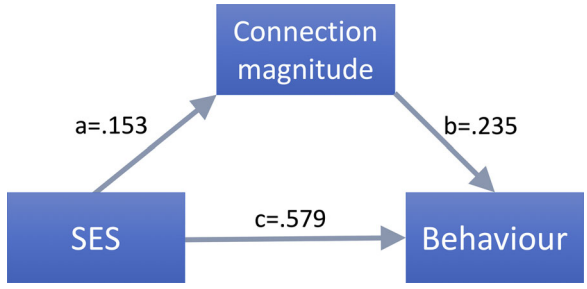

Fig. 9. Example simulated mediation analysis, showing the extent to which a measure of network structure, here connection magnitude, mediated the relationship between SES and behavior in the model (100 epochs, Stimulation condition). See Table 3.

Turning to connection magnitude, this also tended to be higher in more able networks. With respect to early growth, the correlations between initial connection magnitude and later behavioral ability at 100 epochs were .068, .113, and .316, respectively. Analyzed across development, ability differences were reliably reflected in connection magnitude differences in all conditions, but were largest in the Stimulation+structure condition (effect sizes of $\eta_p{}^2 = .002$, $\eta_p{}^2 = .002$, $\eta_p{}^2 = .026$). Trajectories reliably diverged in the Stimulation and Structure conditions, with an increasing effect of ability on connection magnitude differences (epoch × ability: $F(1,5996) = 13.17$, $p<.001$, $\eta_p{}^2 = .002$; $F(1,5996) = 26.02$, $p<.001$, $\eta_p{}^2 = .004$, respectively), but remained parallel in the Stimulation+structure condition. No condition showed convergence of the trajectories, which would be expected if higher ability networks plateaued in connection strengths earlier than lower ability.

3.3.5. Do network structure measures mediate SES–behavior relationships?

Example empirical data are that measures of whole brain cortical surface area (but not cortical thickness) were found to mediate the relationship between an SES-linked measure (family income) and two behavioral scores on executive function tasks (flanker task and working memory; Sobel $z = 2.4$, $p = .02$ and $z = 2.6$, $p = .009$, respectively), thus increasing confidence that structural differences linked with SES were implicated in functional differences (Noble et al., 2015). Khundrakpam et al. (2019) found that cortical thickness mediated the link between SES and language abilities in the same sample, reporting a nonlinear pattern with the strongest effect observed in early adolescence.

Fig. 9 shows an example mediation analysis for the simulation data, in this case in the Stimulation condition, testing the extent to which the measure of connection magnitude mediates the relationship between the SES index and behavioral accuracy. The direct effect between SES and behavior here was .579, while the indirect effect was .036, a small but reliable mediation effect according to the Sobel test (Sobel $z = 4.1$, $p < .001$). Table 3 shows these statistics for all conditions, for connection number and connection magnitude, for two points in development, 100 epochs and 250 epochs, to give an indication of developmental change. For the Stimulation condition, connection number did not reliably mediate SES–behavior relationships, but connection magnitude did, and the mediation increased across development.

Table 3
Simulation data: Mediation analyses showing the extent to which measures of network structure (connection total CT, connection magnitude CM) mediated the relationship between SES and behavior for each condition, at two points in development 100 and 250 epochs of training

Condition	Epoch	Path analysis				Sobel test			
		Structural measure	a	b	c Direct effect	Indirect effect	Sobel-z	Std. error	*p*
Stimulation	100	CT	−.008*	.232	.579	−.002	−.245	.008	.806
		CM	.153	.235	.579	.036	4.102	.010	<.001
	250	CT	.061^{+}	.275	.637	.017	1.878	.010	.060
		CM	.214	.295	.637	.063	5.371	.013	<.001
Structure	100	CT	.823	.290	.293	.239	8.382	.000	<.001
		CM	.604	.272	.293	.164	6.303	.000	<.001
	250	CT	.722	.319	.282	.230	11.905	.000	<.001
		CM	.508	.308	.282	.156	10.326	.000	<.001
Stimulation +structure	100	CT	.847	.344	.397	.291	15.191	.039	<.001
		CM	.633	.241	.397	.153	12.031	.032	<.001
	250	CT	.777	.363	.399	.282	19.771	.026	<.001
		CM	.540	.268	.399	.145	17.482	.020	<.001

Notes: *$p = .806$ $^{+}p = .054$. All other correlations for a, b, and c: $p<.01$. *c* represents the direct effect from SES to behavior; *a* and *b* are the two legs of the indirect pathway via the structural measure (Fig. 10).

Connection magnitude was, therefore, a stronger mediator than connection total. For Structure and Stimulation+structure conditions, there were strong mediation effects, as one would expect given that SES-linked environmental influences were implemented through initial structural differences. In these latter conditions, however, connection number showed stronger mediation effects than connection magnitude; all mediation effects again increased across development. Unlike in the empirical case, for the computational model, we can be sure that *all structural measures are involved in function*, albeit we know that function is affected by other computational parameters (such as, say, learning rate or processing noise) that manifest only indirectly in structural measures through developmental change. Nevertheless, the empirical pattern of stronger mediation effects of cortical surface area than cortical thickness was best captured by the Stimulation condition, where connection magnitude showed reliable mediation, but connection total did not.

3.3.6. Do SES-linked environmental influences moderate the association between network structure measures and behavior?

SES measures have been found to moderate the relationship between brain function and behavior (e.g., Demir et al., 2015). Moderation effects have been more frequently explored in functional studies because they can reveal differences in neurocognitive processing that underly performance differences between SES levels. Here, in the context of a single cognitive mechanism, we tested whether certain properties of networks, reflected in their connection number or magnitude, were more able to influence behavioral development in the presence of higher SES levels. Table 4 shows the results of moderation analyses, using regression models to test whether SES as a continuous variable modulated the relationship between each structural measure and behavior, at two points in development. Reliable moderation effects were found in many cases, but these were much larger in the Structure and Stimulation+structure conditions, and stronger for connection number than magnitude. They demonstrated that there was a stronger relationship between these network measures and behavior for lower SES levels, because the associated reduced processing resources became the limiting factor on performance.

3.3.7. Genetic effects on behavior and network structure

Key empirical data here indicate that individual differences in cognitive ability and indices of brain structure are influenced by genetic variation (Deary et al., 2022). In the sample where SES effects on cognitive development were reported by von Stumm and Plomin (2015), the heritability of cognitive ability varied developmentally from 40% to 70% (Davis et al., 2009; Haworth et al., 2010). In a review paper, Jansen et al. (2015) reported child and adolescent heritability of structural brain measures ranging from 50% to 90%, including indices such as total brain volume, cortical volume, gray matter volume, white matter volume, and cortical thickness. Grasby et al. (2020) reported a heritability of 91% for cortical surface area and 64% for average cortical thickness. Some studies have indicated a partially shared genetic basis for differences in cognitive ability and brain structure (Posthuma, De Geus, & Baaré, 2002, 2003; Schmitt et al., 2020; though see Wallace et al., 2010, for lack of effects). Again, we

Table 4
Simulation data: Moderation analyses showing the extent to which SES moderated the relationship between measures of network structure (connection total CT, connection magnitude CM) and behavior at two points in development 100 and 250 epochs of training

Condition	Epoch	Structural measure	Stepwise regression model predicting behavior				
				Main effect of structure	Main effect of SES	Moderation of structural measure by SES	Direction of effect
Stimulation	100	CT	ΔR^2	5.36%	33.68%	.32%	Stronger relationship between CT/CM and behavior for higher SES
			p-value	<.00001	<.00001	.02176	
		CM	ΔR^2	5.54%	30.14%	.02%	
			p-value	<.00001	<.00001	.57728	
	250	CT	ΔR^2	7.54%	38.63%	0.25%	
			p-value	<.00001	<.00001	.03145	
		CM	ΔR^2	8.73%	34.51%	.00%	
			p-value	<.00001	<.00001	.89051	
Structure*	100	CT	ΔR^2	8.40%	.93%	5.27%	Stronger relationship between CT/CM and behavior for lower SES
			p-value	<.00001	.00145	<.00001	
		CM	ΔR^2	7.39%	2.62%	4.13%	
			p-value	<.00001	<.00001	<.00001	
	250	CT	ΔR^2	10.15%	.57%	6.48%	
			p-value	<.00001	.01187	<.00001	
		CM	ΔR^2	9.49%	2.13%	4.96%	
			p-value	<.00001	<.00001	<.00001	
Stimulation +structure	100	CT	ΔR^2	27.89%	26.79%	8.05%	Stronger relationship between CT/CM and behavior for lower SES
			p-value	<.00001	<.00001	<.00001	
		CM	ΔR^2	19.72%	33.87%	6.01%	
			p-value	<.00001	<.00001	<.00001	
	250	CT	ΔR^2	29.67%	27.02%	10.19%	
			p-value	<.00001	<.00001	<.00001	
		CM	ΔR^2	20.58%	35.59%	7.38%	
			p-value	<.00001	<.00001	<.00001	

Notes ml[: * The small size of additional variance explained when SES is added to the structural predictor occurs because in this condition SES is implemented via a structural manipulation; most of this variance is accounted for when the structural predictor is added first. A stepwise regression predicted behavior (% correct on regular verbs). The structural measure (CT or CM) was entered first, followed by the SES measure, followed by the interaction term to test for moderation. Values show the increase in variance explained and significance.

Table 5
MZ and DZ correlations for behavior and network structure measures for each condition at 100 epochs of training

		Behavior	Connection number	Connection magnitude
Stimulation	MZ correlation	.993	1.00	1.00
	DZ correlation	.617	.325	.439
	Difference	.376	.675	.561
	95% CI	.372	.674	.558
		.380	.677	.563
		Behavior	Connection number	Connection magnitude
Structure	MZ correlation	.569	.995	.945
	DZ correlation	.415	.849	.575
	Difference	.153	.146	.370
	95% CI	.149	.142	.365
		.158	.149	.375
		Behavior	Connection number	Connection magnitude
Stimulation+structure	MZ correlation	1.00	1.00	1.00
	DZ correlation	.794	.866	.739
	Difference	.203	.134	.261
	95% CI	.202	.132	.258
		.205	.137	.264

Notes: Under an additive model, the heritability is estimated at twice the difference between MZ and DZ correlations (Falconer & Mackay, 1998). An additive model holds where DZ correlations are more than half MZ correlations. The additive model fit all conditions except connection measures for the Stimulation condition, where a dominant model was appropriate. Assuming consistency of genetic effects across proximal points of development, values show correlations averaged from epochs 90 to 110 epochs to improve robustness. 95% confidence intervals are shown for MZ–DZ differences across these epochs.

explore whether these types of global effect would be reproduced in a model of an individual mechanism.

Heritability measures in the simulation were assessed by comparing the correlation between MZ twin networks and DZ twin networks, where zygosity influenced the similarity of network parameters, and twin pairs were exposed to the same "family" training set. Table 5 shows the MZ and DZ correlations for each condition, for the measures of behavior and network structure, at 100 epochs of training. The heritability of behavior for Stimulation was .75, for Structure, with environmental variation both in hidden unit number and training set, lower at .31. The Stimulation+structure condition showed a heritability of .41. The heritability of connection number and magnitude was high for the Stimulation condition, such that a dominant rather than additive genetic model would be more appropriate. For Structure and Stimulation+structure conditions, the heritability of network structure measures was between .28 and .74 and fit an additive model. These estimates of heritability for network structure are

unsurprisingly dependent on whether the shared environmental effect implementing SES-linked influences targeted structural indices, as in the latter conditions; or whether network structure was predominantly determined by the artificial genome as in the Stimulation condition. The Structure and Stimulation+structure conditions captured the higher heritability of connection magnitude (analogous to cortical surface area) than connection number (cortical thickness), while the Stimulation condition did not. Nevertheless, all conditions successfully captured the simulation of SES effects on behavior and brain structure against a background of significant heritability of these metrics.

The simulations were less successful in capturing patterns of bivariate heritability, that is, estimating the overlap between the inferred genetic influence on pairs of measures. In particular, the simulations produced cross-twin cross-trait correlations for behavior/network structure that were higher for DZ than for MZ (correlations are shown in Table S1), an unusual pattern not reported in the literature (see, e.g., Brans et al., 2010; Posthuma et al., 2003; Schmitt et al., 2020; Wallace et al., 2010, e.g., genetic and environmental correlations for intelligence and brain). This pattern can occur if three conditions hold: both traits are strongly heritable, there is a negative genetic correlation (whereby genetic influences that increase one trait will tend to decrease the other trait), and there is a strong shared environment effect (Purcell, 2002). These were the conditions that arose here. For example, in the Structure condition, the cross-twin cross-trait correlations between behavior and connection number were best fit by a genetic correlation of –.60 and shared environment correlation of +.96; those between behavior and connection magnitude by a genetic correlation of –.40 and shared environment correlation of +1.0; and between connection number and magnitude by a similar high shared environment correlation of +1.0, but a positive genetic correlation of +.62.[1] These bivariate estimates are consistent with the strong environmental manipulation to network structure used to implement SES in this condition.

For empirical data, it is necessary to infer the shared genetic influences contributing to phenotypic correlations because genetic influences, and the biological processes to which they contribute, remain largely unknown (Elliott et al., 2018), the best window currently being offered by genome-wide association analyses (e.g., Grasby et al., 2020). For the simulation, however, the relationship between the artificial genome and the computation parameters was well characterized. Fig. 10, therefore, depicts the actual correlations between neurocomputational parameter settings and phenotypic variability for each condition, for behavioral and network structure measures. The picture these simple correlational data give is not perfect: in many cases, the relationship between parameters and outcomes is nonlinear, while Fig. 10 shows the size of simple linear correlations; and the parameters can interact to produce effects not captured in independent correlations (Thomas et al., 2016). Nevertheless, it is apparent that some parameter effects on behavior are in opposite directions to those on structure (e.g., noise, sparseness, and weight variance), consistent with the observed negative genetic correlations. Moreover, the contribution of parameters to variability was very similar across SES conditions—different ways of implementing environmental influence did not alter associations.

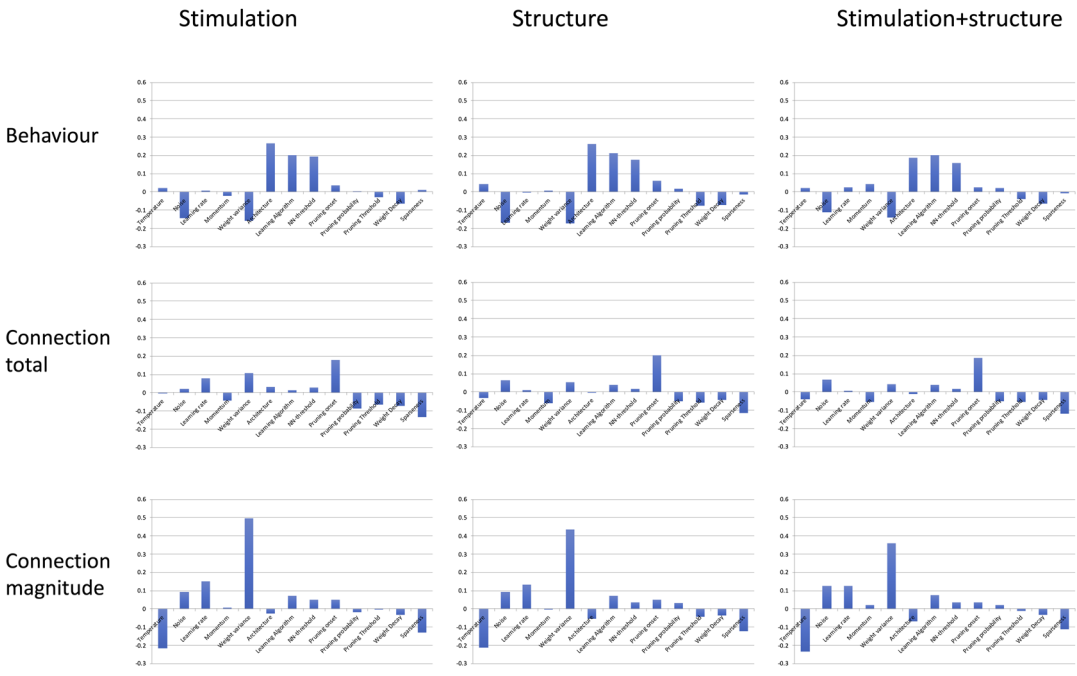

Fig. 10. Linear correlations between parameter values and phenotypic outcomes for each condition at 100 epochs of training. The parameters shown are: sigmoid activation function temperature, netinput noise, learning rate, momentum, initial weight variance, architecture, backpropagation error metric, nearest neighbor response threshold, pruning onset, pruning probability, pruning threshold, weight decay rate, and sparseness of initial connectivity. Hidden units are not included here as this parameter was only specified by the artificial genome for the Stimulation condition.

3.3.8. Modulation of heritability according to SES

One gene × environment interaction reported in the literature is between SES and the heritability of intelligence, notably in U.S. twin samples, where the heritability is reduced at the lowest levels of SES (Turkheimer et al., 2003). The interaction was driven by DZ twin correlations decreasing more substantially as a function of higher SES than MZ correlations (Giangrande et al., 2019). However, the interaction has not been replicated in European populations, suggesting that it may depend on the range and absolute level of SES variability (Tucker-Drob & Bates, 2016).

Table 6 shows simulation data for MZ and DZ correlations for behavior, split by SES quartile. For all conditions, the SES quartile modulated the MZ-DZ difference, and, therefore, the heritability of individual differences in behavior. For the Stimulation condition, the lowest SES quartile showed the lowest heritability and, in line with the empirical data, this interaction was driven by a faster reduction in DZ than MZ correlations with higher levels of SES. For Structure, when there were sufficient hidden units, performance was increasingly dependent on stochastic differences in stimulation (shown by reducing MZ correlations, see Table S3). For Stimulation+structure, the lowest SES quartile aligned with constraints of structure,

Table 6
Simulated MZ and DZ correlations for behavior for each condition split by quartile of SES, at 100 epochs of training

Behavior		SES quartile			
		Bottom	Lower middle	Upper middle	Top
Stimulation	MZ correlation	.994	.994	.991	.987
	DZ correlation	.502	.427	.322	.443
	Difference	.491	.567	.668	.544
	95% CI	.486	.561	.662	.538
		.497	.574	.675	.550
		Bottom	Lower middle	Upper middle	Top
Structure	MZ correlation	.924	.452	.330	.334
	DZ correlation	.234	.147	.319	.363
	Difference	.690	.305	.011	−.029
	95% CI	.684	.295	.005	−.035
		.696	.315	.017	−.022
		Bottom	Lower middle	Upper middle	Top
Stimulation +structure	MZ correlation	.992	.993	.994	.989
	DZ correlation	.359	.391	.358	.495
	Difference	.634	.603	.636	.495
	95% CI	.630	.598	.628	.481
		.637	.607	.644	.508

Notes: Assuming consistency of genetic effects across proximal points of development, values show correlations averaged from epochs 990 to 110 epochs to improve robustness of estimates. 95% confidence intervals are shown for the differences in correlations (see Fig. S8 for plots).

while the highest SES quartile aligned with constraints of stimulation, leading to a flatter profile. In short, the attenuation of heritability in the lowest SES group reported in the literature was only captured in the Stimulation condition.

3.3.9. Heritability across development

Empirical data show an increase in the heritability of cognitive ability across childhood (Davis et al., 2009). There is some evidence that the heritability of measures of brain structure slightly increases across age (Jansen et al., 2015). For example, Schmitt et al. (2014) reported an increase in the heritability of cortical thickness through late childhood and adolescence. The predictive power of SES on behavior also increases with age (von Stumm & Plomin, 2015). We tested whether these global effects could be captured by a model of a single mechanism.

None of the conditions showed an increase in heritability of behavior across development––instead, levels declined (see Table S2 and Fig. S9 for MZ and DZ correlations). For Stimulation and Stimulation+structure, the influence of SES on behavior increased across development, as observed in the empirical data on IQ, while SES effects were broadly flat across development for Structure. Variable patterns were observed for the change in the heritability of network connection measures. Stimulation and Structure captured the increasing heritability of the analog of cortical thickness, connection number, while this was reduced in Stimulation+structure. For connection magnitude, Structure and Stimulation+structure conditions predicted increasing heritability across development, while heritability of magnitudes fell for Stimulation. The developmental pattern of heritability for the connection measures is complex because there can be early environmental effects on structure, lifespan effects due to stimulation, and differential effects across the development with the onset of pruning, itself genetically influenced but with results that are sensitive to both early environment effects and lifespan stimulation effects.

In sum, no condition captured age-related increases in the heritability of behavior, but some captured the developmental increase in SES influence, and the heritability increases for brain structure measures.

3.3.10. Summary

We contrasted different implemented pathways for proximal environmental influences on development that reflect competing hypotheses for how the distal effects of SES operate. These pathways were evaluated in a simple developmental model that captured trajectories of behavioral, cortical surface area and cortical thickness change with age (given certain bridging assumptions for the latter). Within the highly simplified framework of the language model, SES associations were readily simulated in behavior when SES-linked environmental influences were implemented either in terms of differences in cognitive stimulation or in prenatal brain development, and simulations also captured widening behavioral SES gaps across development. The cognitive stimulation account gave a better fit to the small nonlinear SES effects on the cortical surface area in mid and later childhood (Noble et al., 2015), as well as the faster thinning of the cortex in children from low SES families (Piccolo et al., 2016). The prenatal brain development account gave a better fit to the larger linear SES effects on cortical gray matter in infancy (Betancourt et al., 2016). In the model, network structure measures partially mediated the relationship between SES and behavior as with human studies, and there was some indication that SES could moderate the relationship between network structure measures and behavior.

All conditions simulated these qualitative effects against a background of strong polygenic genetic influence on individual differences in behavior and brain structure (Deary et al., 2022). The cognitive stimulation account also captured reduced heritability of behavior in the lowest SES groups (Turkheimer et al., 2003). The simulations were less successful in capturing the increase in heritability with age, and bivariate measures of shared genetic influence on brain and behavior.

4. Building on the model: Intervening to narrow the gaps

The model we have described is highly simplified and very limited in its scope. Nevertheless, the implemented multiscale model provides a foundation to explore what would happen to the individual differences in the population under different circumstances, for example, in response to an intervention that sought to narrow the gaps between children's developmental outcomes. We can ask two types of questions of the model: how much does the success of such an intervention depend on the causal pathway by which SES-linked effects are operating, prenatal brain development versus postnatal cognitive stimulation (or both)? And, to what extent can network structure differences predict the extent to which behavioral outcomes can be improved?

Globally, increasing work has considered how interventions may promote early childhood development, targeting the sectors of health, nutrition, education, child protection, and social protection, as well as the optimal developmental timing for each type of intervention (Britto et al., 2017; Noble, 2021). For example, studies have explored the relative merits of nutrition interventions for children living in poverty compared to cognitive stimulation, contrasting outcomes such as physical growth with behavioral metrics like socioemotional, cognitive, language, and motor development (e.g., Grantham-McGregor, Powell, Walker, & Himes, 1991; McCoy et al., 2017; Prado et al., 2019). Studies have also tested possible interactions between interventions, such that later schooling might buffer the effects of early nutritional deficits (e.g., Gorman & Pollitt, 1996; Stein et al., 2008). A recent study proposes that, despite the importance of nutritional interventions for health and early development, improving long-term cognitive outcomes requires interventions that specifically target early cognitive ability through enrichment, in other words, that target cognitive stimulation (Stein et al., 2023). From a cognitive perspective, interventions have been proposed that specifically target SES-related differences in children's executive function and language development through cognitive stimulation. For example, there are interventions that seek to increase parent–child language interactions (Neville et al., 2013; Pace et al., 2017; Romeo et al., 2018b) or target factors that might buffer the child against adverse environments, such as through caregiver support or training the child's attention (Luby et al., 2013; Markant, Ackerman, Nussenbaum, & Amso, 2016). From a cognitive neuroscience perspective, the focus has recently turned to tracing the longitudinal consequences on children's brain function of interventions that directly address poverty, such as through cash payments to mothers (e.g., Troller-Renfree et al., 2022).

Interventions operate on the whole child. They may have complex effects on the physical, cognitive, and emotional state of the individual, as well as causing changes to the social and educational systems within which the child is embedded that may attenuate or exaggerate the impact of the interventions. Within our narrow model perspective, we can only consider how such global effects may be reflected in developmental changes within specific cognitive mechanisms (see Thomas et al., 2019, for a review of computational modeling approaches to simulating interventions).

We implemented an intervention early in development that sought to maximize and equalize cognitive stimulation across the population, akin to the Thirty Million Words initiative

(https://cri.uchicago.edu/portfolio/thirty-million-words/) which seeks to address disparities in language development caused by different levels of language experience. In terms of the modeled past tense domain, we applied the intervention at a point approximately equivalent to 5 years of age. At that point, all networks were provided with the full training set. To be clear, the intervention occurred at the *same developmental point* in each of the three SES conditions. However, the timing of when SES-linked environmental influences impacted development differed between the conditions. For the Structure condition, they operated early, during network growth, while for the Stimulation condition, they operated later, during experience-dependent development. The intervention relied on equalizing cognitive stimulation, which was ex hypothesi the cause of SES-related gaps in the Stimulation condition, but was not the cause of the gaps in the Structure condition. We might, therefore, predict that the largest population-level behavioral improvements would be found in the Stimulation condition where the intervention directly addressed the cause of the gaps, and the smallest in the Structure condition where it did not. For the Stimulation+structure condition, the intervention would address one of the original causes of the SES-associated gaps (stimulation) but not the other (structure), so we predicted an intermediate outcome. As we shall see, these predictions turned out to be wrong.

4.1. Method

At 50 epochs of training, each network in each population was given the full training set. The performance of each individual under the intervention was compared to the same individual's original (control) performance at an equivalent point of development. The comparison was made after 100 epochs of the intervention (150 epochs overall), to minimize any asymptote effects in measurement.

4.2. Results

Fig. 11 shows density plots for each condition, for behavior and for the network structure measures, respectively. All measures showed reliable intervention effects (paired *t*-tests, all $p<.001$). These effect sizes were large for behavior (Cohen's d for Stimulation = .61, Structure = .59, Stimulation+structure = .45), medium for connection magnitude (.22, .23, .15), and small for connection number (.05, .06, .03). Mean intervention effects are shown in Table 7. Intervention served to increase behavioral accuracy, connection number, and connection magnitude. For connection number at least, then, the intervention did not *accelerate* development, since the developmental change in the model is associated with fewer connections, not more. Instead, the intervention strengthened connections sufficiently to resist pruning and retain additional resources for connectivity.

Surprisingly, the Stimulation and Structure conditions showed comparable mean intervention effects on behavior (nonsignificant condition × intervention interaction: $F(1, 1998) = .03$, $p = .861$, $\eta_p^2 = .000$). The intervention effect for the Stimulation+structure condition was reliably smaller than each (vs. Stimulation: $F(1, 1998) = 23.93$, $p<.001$, $\eta_p^2 = .012$; vs. Structure: $F(1, 1998) = 22.29$, $p<.001$, $\eta_p^2 = .011$).

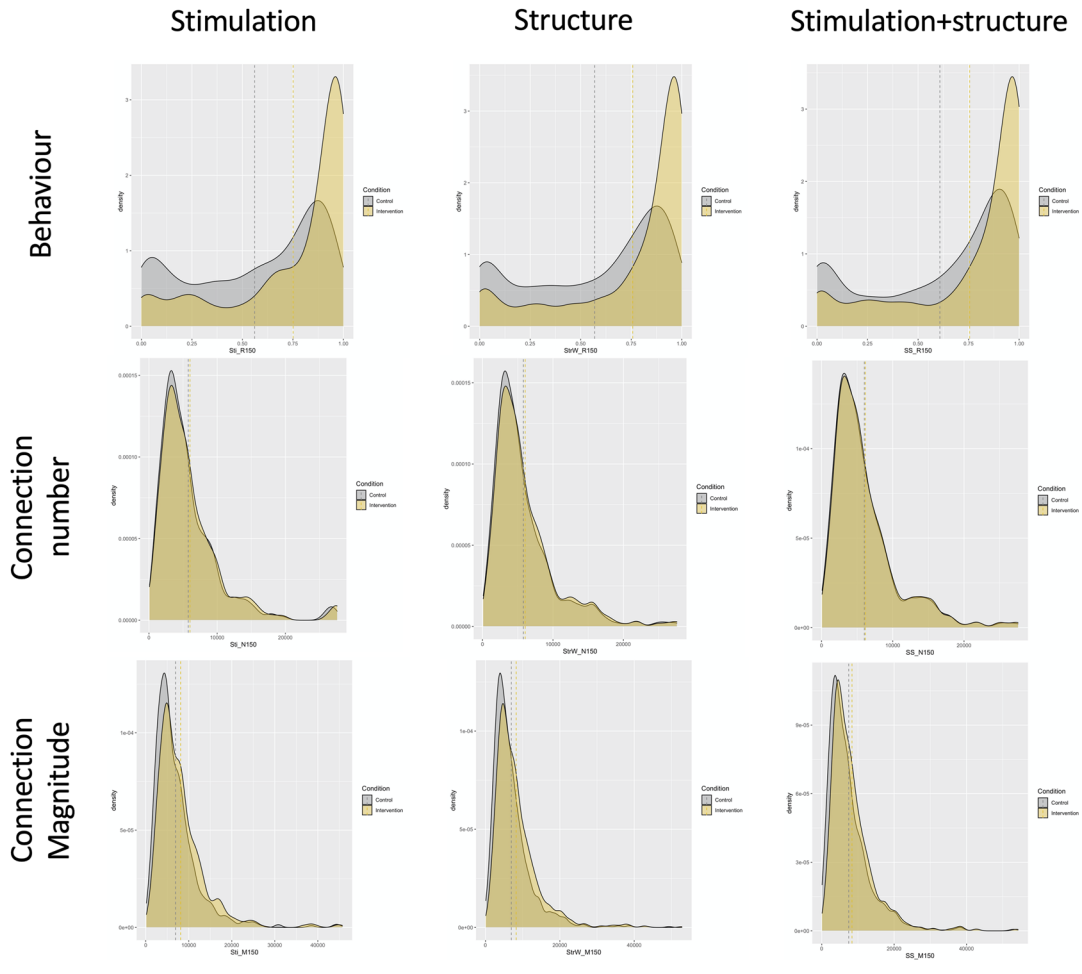

Fig. 11. Density plots for behavior and network structure measures, demonstrating the effects of intervening to maximize and equalize cognitive stimulation. Interventions were applied at 50 epochs and results show population measures 100 epochs later. (Control = gray; Intervention = yellow. Vertical dashed lines show population means.)

Table 7
Intervention effects on behavior (proportion correct), connection number, and connection magnitude for each condition (data show intervention minus control)

		Stimulation	Structure	Stimulation+structure
Behavior	Mean	.191	.189	.147
	SD	.236	.233	.148
Connection	Mean	255	265	148
number	SD	700	683	534
Connection	Mean	1197	1311	918
magnitude	SD	1490	1820	799

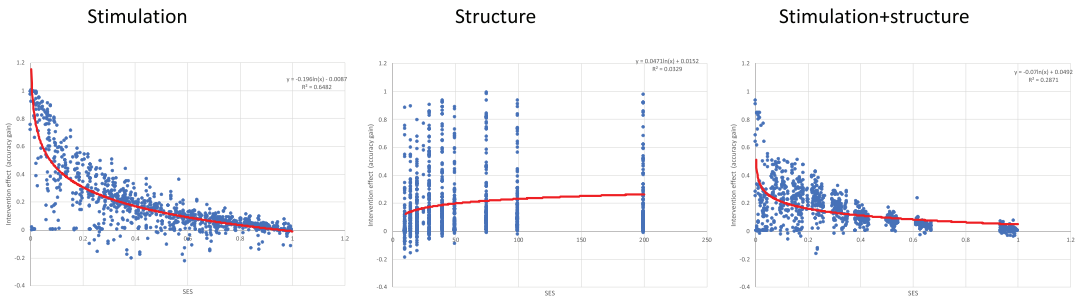

Fig. 12. The relationship between behavioral intervention effect size (gain in accuracy between control and intervention) plotted at an individual level for each condition.

Comparison of intervention effects for network structure measures showed equivalent changes in both connection number and magnitude for the Stimulation and Structure conditions (nonsignificant connection number interaction of condition × intervention: $F(1, 1998) = .10$, $p = .753$, $\eta_p^2 = .000$; connection magnitude: $F(1, 1998) = 2.34$, $p = .126$, $\eta_p^2 = .001$). Both of these conditions showed larger effects of the intervention on network structure than the Stimulation+structure condition (connection number: vs. Stimulation: $F(1, 1998) = 14.97$, $p<.001$, $\eta_p^2 = .007$; vs. Structure: $F(1, 1998) = 18.17$, $p<.001$, $\eta_p^2 = .009$; connection magnitude: vs. Stimulation: $F(1, 1998) = 27.15$, $p<.001$, $\eta_p^2 = .013$; vs. Structure: $F(1, 1998) = 38.96$, $p<.001$, $\eta_p^2 = .019$).

Our predictions for the model, then, were not borne out. Despite different developmental timings of SES-linked effects in Stimulation and Structure conditions, the populations were equally able to respond to a cognitive stimulation intervention applied at the same point across conditions. By contrast, there were weaker effects in the Stimulation+structure condition, indicating that *cumulative* SES-linked influences were a driving factor. However, these results report mean population scores. When we look at individual intervention effects, shown in Fig. 12, we see a different pattern. For Stimulation, there was a clear indication that the largest intervention effects occurred for individuals at the lowest SES level, consistent with differences in stimulation being the primary cause. This is in line with the idea that the smallest intervention effects occur for those previously with the strongest development (Jolles & Crone, 2012). For Structure, the weakest effects were observed at the lowest level, but there was a large amount of variability (here, driven by a combination of stochastic differences in the stimulation to which the individual had been exposed, and variation in genetically influenced parameters). Stimulation+structure depicted a combination of these functions, including larger effects at lower SES levels but greater variability in response.

Lastly, in the narrow domain of the model, could network structure measures predict individual intervention effects? This speaks to a deterministic view of the role of brain structure: perhaps differences in brain structure measures will predict how successful interventions are, with smaller brains responding more poorly to increases in cognitive stimulation? The correlations between the network structure measures at 50 epochs and the subsequent individual behavioral intervention effects at 150 epochs were as follows. For connection total:

Stimulation $r = .229$, $p<.001$; Structure $r = .108$, $p = .001$; Stimulation+structure, $r = -.494$, $p<.001$. For connection magnitude, the equivalent correlations were $r = .085$, $p = .007$; $r = -.023$, $p = .467$; and $r = -.398$, $p<.001$. Whether and how network structure at the onset of intervention could predict response to intervention was, therefore, dependent on how SES-linked effects were implemented. Network structure measures in isolation gave no clear view of the potential for behavioral intervention.

In sum, contrary to expectations, for the intervention it did not matter at a population level whether the causal pathway for SES-linked differences was early network growth or cognitive stimulation. There was the same effect of a postnatal intervention that served to equalize and maximize cognitive stimulation. The poorest population response occurred through cumulative effects of different proximal causes of SES influence. In all cases, changes to network structure induced by the intervention were subtle. Preintervention measures of network structure were no better able to predict which individuals would respond best to intervention in the Stimulation or Structure conditions. However, the conditions were distinguished in the individual response to intervention, with markedly stronger behavioral effects observed at the lowest SES levels in the Stimulation condition.

5. Discussion

The application of developmental science to the impact of poverty on child development intends to inform ways to intervene to improve developmental outcomes. The move from correlations, for example, between SES and measures of brain structure (Noble et al., 2015), to intervention relies upon causal models (Troller-Renfree et al., 2022). Without a causal understanding, correlations risk being misinterpreted. Policymakers might view brain differences between children from poor and rich families in a deterministic light, the gaps perhaps less amenable to reduction via cognitive or educational interventions. However, at this time, causal accounts linking SES effects on behavior and brain structure represent a significant challenge, since the integrated multiscale developmental explanations within which they must be nested do not exist, and to be valid, such explanations must also accommodate a range of findings on genetic effects on behavior and brain development. For example, if SES effects are partially markers for correlated genetic variation in stratified populations, environmental manipulations may have reduced effects.

In this article, we considered the contribution of computational modeling to the construction of causal accounts of SES effects on brain and behavior. We started with a simple ANN model of language development, restricting our focus to a single mechanism. We added neurobiological constraints to capture the developmental processes of network growth, activity-dependent change, and pruning, as well as genetic and environmental sources of variation. We introduced bridging concepts that, while speculative, enabled us to link between properties of the ANN's structure (based on its connectivity) and brain structure measures usually derived from MRI scans. This model allowed us to contrast possible pathways of SES-linked influences on development, targeting network growth (prenatal effects; Betancourt et al., 2016; Hanson et al., 2013), activity-dependent change (postnatal cognitive stimulation; Brito, 2017;

Romeo et al., 2018a), or a combination of both (compounded influences; Farah, 2017). We were able to compare which pathways were best able to qualitatively reproduce empirical data spanning development, individual differences, behavior, and brain structure (albeit contingent on the assumption that effects in specific mechanisms scale to global effects). Finally, with these versions of the model in hand, and retaining our narrow single mechanism focus, we turned to simulating the effects of a cognitive enrichment intervention to narrow the gaps in behavioral outcomes. The model produced unexpected findings. A simulated intervention equalizing and maximizing cognitive stimulation in early childhood produced similar mean population improvements in behavioral outcome whether SES-linked influences had acted on early prenatal network growth or on later postnatal cognitive stimulation. The poorest outcomes were associated with the cumulative effects of both influences. Unexpected outcomes demonstrate the value of computational modeling. In this case, we had not anticipated that individuals with smaller networks nevertheless often retained the plasticity to take advantage of cognitively enriched environments.

This line of research should not be taken to underestimate the complexity of the construct of SES nor what it means for child development; what it means, for example, for a particular child to be raised in a poor neighborhood in Los Angeles in a family with an annual income of less than $5000; how this might influence the nutrition and healthcare available to the child, the nature of her parenting, the opportunities for play and cognitive stimulation, the quality of the housing in which she lives, the risk of adverse experiences, interactions with siblings and peer group, the values and resources in the community, and the nature of her schooling. Yet, the emerging neurocognitive literature on SES has been influential (see, e.g., Mariani, 2017; Noble & Giebler, 2020). This raises the question of the translational implications of the cognitive neuroscience findings. Based on their brain structural data, Noble et al. (2015) commented:

> "*Our results should in no way imply that a child's socioeconomic circumstances lead to an immutable trajectory of cognitive or brain development . . . by elucidating the structural brain differences associated with socioeconomic disparities, we may be better able to identify more precise endophenotypic biomarkers to serve as targets for intervention, with the ultimate goal of reducing socioeconomic disparities in development and achievement*." (p. 778).

Yet, what *do* such results imply? Other authors have been more forthright in identifying implications for intervention. In two samples of over 3000 10-year-old children in the United States, Tomasi and Volkow (2021) observed that of their SES indices, family income was a stronger predictor of cortical volume and cortical thickness than the indices of parental education and neighborhood deprivation. They concluded:

> "*These results suggest that lack of supportive/educational stimulation in children from low-income families might drive the reduced cortical volume and cortical thickness. Thus, strategies to enhance parental supportive stimulation and the quality of education*

> *for children in low-income families could help counteract the negative effects of poverty on children's brain development*." (p. 6619)

One might argue (as we consider below) that morphometric measures of brain structure derived from MRI, such as cortical thickness and surface area, are so highly derived and conflate so many micro-level developmental properties that it is too great a leap to assign functional or mechanistic roles to these measures. In that case, SES-linked differences in brain structure simply have *no implications for policy* because they cannot be used to identify how outcomes could be changed. The alternative, which we prefer, is to begin to construct mechanistic models that link levels of description and use these to test how gaps could be narrowed.

The use of neurocomputational models to investigate interventions is also in its infancy. Thomas et al. (2019) began to consider how such models could be used more generally to consider behavioral interventions. For example, in a similar population model to that presented here, they explored the effects of removing differences in cognitive stimulation at different points in development. The result was that gaps closed between individuals, but the strength of the effect depended on timing, with later interventions less effective. Due to maturational changes in connectivity, early deficits in cognitive stimulation became long-term differences in structure, which were then more resistant to behavioral interventions. Similar empirical data have been reported for children experiencing very deprived early environments: even when these deprived conditions were removed, they left lifelong detectable changes in brain structure (Mackes et al., 2020). Childhood SES can be a stronger predictor of adult neural activity than the adult's concurrent SES status (Kim et al., 2013). The model we present must be strongly caveated given its simplifications and narrow single-mechanism focus. The operationalization of the intervention was simply in terms of equalizing stimulation to a single associative system, and we would be cautious about the extent to which the findings generalize. Nevertheless, the modeling supports previous arguments for considering both the maturational state of the system, and the origin of the individual differences in the target population, for predicting the outcome of behavioral interventions (Jolles & Crones, 2012). However, work is only just beginning in this area, and as our results showed here, the outcomes of implemented simulations of complex systems can be unexpected.

5.1. Model limitations

Our initial model was limited in a number of ways. It depicted a single cognitive mechanism; it learned through passive exposure to stimulation; it was not an autonomous agent that could select its environment and so develop adaptive strategies; and the modeled system had no emotional or reward-learning aspects, which are likely influential in the response of the developing brain to deprivation (Yaple & Yu, 2020). Whole system properties, such as adaptive and reward-based responses, are likely to be central to understanding the full impact of variations in SES-linked influences on cognitive and brain development. The model assumed that environmental and genetic variation were independent, though there is evidence that they can be correlated (e.g., Allegrini et al., 2020). The model could only connect with global

properties, such as general intelligence or brain volume, inasmuch as variations in these properties are reflected at the local level in the structure and function of individual mechanisms, which remains speculative.

The model was also based on a three-layer feedforward ANN and the limited biological plausibility of this architecture is well known (see Crick & Asanuma, 1986; Lillicrap, Santoro, Marris, Akerman, & Hinton, 2020, and Thomas & McClelland, 2023, for a more recent discussion). The model does not incorporate aspects of neural circuitry that may be key in producing changes in measures like cortical thickness and surface area, such as cortical columns, six-layer laminar structure, and intracortical myelination versus fiber tract myelination; and functional properties, such as diverse neurotransmitters and dynamic local circuit oscillations. The model simplifications were motivated by the primary goal to make contact with behavioral data from a concrete domain where associations with SES have been observed, rather than with reported patterns of neural activity. Nonetheless, alternative architectures could have been selected for the target system, such as deep convolution neural networks, which have more recently been proposed to better capture neural processing (e.g., in the ventral visual system, Lindsay, 2021). Deep convolution neural networks have a more biologically plausible hierarchical structure and organization of connectivity around receptive fields, although they still rely by and large on forms of backpropagation for training. Another line to develop the current work would be the use of neuroanatomically constrained ANN models of language development (e.g., Ueno, Saito, Rogers, & L. Ralph, 2011). This would allow closer links to specific empirical effects, such as the association between language exposure and white matter pathways in the language system (left arcuate and superior longitudinal fasciculi; Romeo et al., 2018a). However, such models have yet to be scaled to the population level necessary to capture environmental and genetic influences.

The model was assessed based on qualitative rather than quantitative patterns, and qualitative fits are to an extent subjective. The model was not calibrated to produce specific effect sizes, nor to match epochs of training to specific chronological ages. In one way, this is a strength—no parameters in the model were directly tuned to simulate any of the empirical effects; where the model captured developmental, SES, or genetic effects, these arose solely from design assumptions rather than parameter fitting. However, it also meant that some effects were ill-calibrated: SES/shared-environment effects were too powerful, so the stimulation conditions allowed almost zero levels of cognitive stimulation, and the structure conditions produced very large effects on network structure. The Stimulation and Structure conditions were matched to each other on their population performance but the combination of Stimulation+structure then produced exaggerated environmental effects. Genetic effects on variation in the simulated populations were quite strong, and measurement noise was largely absent.[2] Perhaps the main drawback of the lack of model calibration is uncertainty on whether all the observed effects would hold were the model more realistically calibrated (albeit finding appropriate empirical data to constrain the model at each scale would not be a trivial exercise). Nevertheless, one could also argue that some of the empirical findings, particularly for SES, are inconsistent and not well replicated, so it may be premature to undertake an exercise involving quantitative fits.

Turning to the brain level, the model collapsed structural indices into two types: *resources for connectivity*, which are irrevocably lost as part of a developmental process of optimization, and *operational connectivity*, which increases in line with the function. This coarse rendition generates bridging concepts that allow the model to connect data at different levels of description, but it means that there are phenomena that it cannot capture, such as where different resource measures like gray matter and cortical thickness diverge (e.g., Gogtay et al., 2004; Schnack et al., 2015), the temporary local increases in gray matter produced by training (Draganski, Gaser, Busch, Schuierer, & Bogdahn, 2004), or lifespan trajectories of cortical change including aging (Schnack et al., 2015). For example, Schnack et al. (2015) demonstrated that following developmental expansion, the cortical surface area shrinks from early adolescence into adulthood. In terms of individual differences, Brans et al. (2010) reported that in contrast to the pattern observed in mid-childhood/adolescence, adults with higher intelligence showed attenuated cortical thinning and more pronounced cortical thickening over time compared to lower intelligence. With respect to SES, Chan et al. (2018) argued that younger and older adults do not exhibit consistent SES-related differences in brain measures. Such lifespan and transient training-induced changes are beyond the scope of the simple model described here.

The enterprise of linking MRI measures of brain structure with functional properties must also be viewed in its proper context. There are a number of necessary explicit assumptions in the model that may not square with the neurobiology and physics underlying the MRI measures used as proxies for connection strength, pruning, and regressive events. First, cortical thickness measures (as implemented in Freesurfer and other MRI analysis platforms) are entirely dependent on local changes in the arbitrary image values measured via T1-weighted (T1w) MRI scans. In particular, the border between "gray matter" and "white matter" is defined by a relatively steep gradient in T1w values from higher ("white matter") to lower ("gray matter"). Crucially, this inferred border can be shifted up toward the pial surface via an increase in T1w values within the deepest cortical layers. Such increases in deep cortical T1w values, and resulting cortical "thinning," can be driven by increases in cortical myelination near the gray–white boundary, as shown by Natu et al. (2019) in the lateral ventral temporal cortex. Notably, in the same study, Natu et al. (2019) showed that age-related cortical thickness changes in the medial ventral temporal cortex could not be straightforwardly explained by myelin-related developmental changes, suggesting regional dependence. The interpretation of T1w values is important for the current account, in that cortical thinning—here theorized to reflect a subtractive or "pruning" process—may conversely sometimes reflect increased local myelination in deep cortical layers, a putatively additive process. While quantitative MRI approaches to development and lifespan changes (e.g., Carey et al., 2018; Natu et al, 2019; Erramuzpe et al., 2021; Taubert et al., 2020) can help disambiguate these potential processes, as can combined in-vivo and ex-vivo assays, the ambiguity of links between structural and functional properties renders modeling in this field necessarily speculative.

In addition, in interpreting the current body of imaging findings, the very small SES-related differences in area and volume reported in older children and adults also need to be taken in the context of other measures that may or may not have been accounted for, such as height, body mass, and intracranial volume. Finally, subtle differences in MRI scanning protocols,

head position/rotation within the head coil, or postprocessing steps may drive potentially artifactual differences that masquerade as SES effects.

5.2. Model strengths

The model was set a wide range of phenomena to integrate across multiple levels of description. To the extent that it could capture developmental trajectories in behavior and brain structure, and individual differences effects linked to ability, SES, and genetic variation, this supports the adequacy of its assumptions. These assumptions included: experience-dependent change in distributed neural networks; the sources of genetic and environmental influences on individual differences in neurocomputational parameters, network growth, and cognitive stimulation; and the first approximation of separately matching *resources for connectivity* and *operational connectivity* to indices of brain structure. The model was able to simulate small nonlinear SES effects in the cortical surface area under the Stimulation condition, and larger linear SES effects on the cortical gray matter under the Structure condition, supporting the view that the SES effects on brain and cognition reported by Noble et al. (2015) are plausibly the result in differences in cognitive stimulation, while those reported by Betancourt et al. (2016) in 1-month infants are the result of prenatal brain development.

Where there were shortcomings, the model assumptions need to be altered. For example, the failure of the Stimulation condition to show developmental connection magnitude increases in low-ability individuals suggests the floor stimulation level must be higher or connection magnitude must also have a maturational (nonexperience dependent) component. The lack of increase in heritability of intelligence with age suggests an emerging gene–environment correlation should be added to the model, where the richness of training sets increasingly aligns with the computational power of the learning systems[3] (see, e.g., Allegrini et al., 2020, and Selzam et al., 2019, for evidence of SES-related gene–environment correlations in educational achievement and attainment).

The model also demonstrated candidate ways that structure and function can be linked. As we saw in the Introduction, there is empirical evidence both that cortical surface area differences cause differences in educational attainment (structure influences subsequent function) and that educational attainment causes differences in cortical surface area (function influences subsequent structure) (Grasby et al., 2020). In the Stimulation condition, differences in the acquisition of function led to subsequent differences in structure. In the Structure condition, initial differences in structure led to subsequent differences in the acquisition of function. This led to different predictions of developmental change, such as in how network structure mediated the relationship between SES and behavior (Section 3.3.5). As with MRI measures of the brain, the structural measures of the connectivity of the ANN gave only an oblique view of its function. Because many key neurocomputational parameters influencing function are hidden (such as learning rate, processing noise, and sigmoid activation function temperature), these can only be viewed indirectly as their influence manifests on structure across development. However, in contrast to the brain, the model's operation is well understood. Therefore, it is possible to assess the actual contribution of functional parameters to variations in behavior and the structural measures as discussed in Section 3.3.7.

The model generated explanations of empirical observations. Both Stimulation and Structure conditions showed a nonlinear logarithmic relationship between SES and behavior. The Stimulation condition did so because of the systematic nature of the information available in the environment (in this case, the regular past tense rule). Below a certain amount of information in the training set, this regularity could not be extracted. The Structure condition captured the same pattern for a different reason. The relationship between hidden unit number and function is itself nonlinear, so there is a faster drop in function as the level drops below the minimum required for successful learning, and a slower benefit as the minimum is exceeded.

Only the Stimulation condition captured how low SES reduces the heritability of intelligence (Turkheimer et al., 2003). It did so because only below a certain size of training set did the training set become the principal limiting factor on development—the level experienced by the lowest quartile of SES. This generates a possible explanation for the intermittent observation of SES $\times$ heritability interactions (Tucker-Drob & Bates, 2016). The interaction depends on the absolute level of the lowest SES group sampled. It only emerges when cognitive stimulation becomes the limiting factor in developmental outcomes compared to the genetic influence on variation in the population. In the model, the Structure condition did not reproduce the interaction because low SES was implemented as the fewest hidden units; the ability of the model to learn successfully with few hidden units depends on the settings of other neurocomputational parameters, whose variation in the implementation was genetically influenced.

Farah (2017) argued for the importance of distinguishing different ways in which SES might modulate developmental trajectories in order to identify underlying causal pathways: delayed onsets might imply early operating effects, widening gaps might imply compounding, and narrowing gaps might imply common trajectories followed at different rates. The model demonstrated that early operating effects could produce widening of gaps across development (shown by the Structure condition); that compounding is not required to produce widening gaps (observed in the consistent lifelong effects in the Stimulation condition); that compounding, however, can exaggerate widening (observed in the Stimulation+structure condition); but that narrowing/catch-up was not exhibited as an outcome of SES influences in any condition for any measure in the implementation.

Implementation sometimes allows apparent paradoxes to be resolved. Take an 8-year-old and a 16-year-old. The 16-year-old has less gray matter but is more cognitively sophisticated than the 8-year-old (Gogtay et al., 2004; Inhelder & Piaget, 1958). Take two 8-year-olds. The one with more gray matter is likely to be slightly more cognitively able (McDaniel, 2005). Is it better, then, for cognition to have more or less substrate? Developmental and individual differences perspectives give inconsistent answers. Development says less is better, individual differences says more is better. The model resolves the inconsistency by demonstrating that a bigger network is almost always better. Although network size reduces across development through optimization (perhaps, biologically, also to reduce on the metabolic costs for maintaining resources for connectivity and the risks of aberrant function associated with high connectivity, such as epilepsy), at any point in development, the larger network is better off (Section 3.3.4).

Here is another paradox. It has been argued that the cortex thins more quickly in lower SES groups (Piccolo et al., 2016) and indeed this may represent accelerated brain maturation induced by the increased stress and reduced cognitive enrichment associated with economic and social disadvantage (Tooley et al., 2021). However, the cortex is also observed to thin more quickly in more intelligent individuals (Shaw et al., 2006). But higher IQ is associated with higher SES (von Stumm & Plomin, 2015), and for higher SES, the cortex thins more slowly. Can all these observations be correct? They appear inconsistent. Nevertheless, the Stimulation condition reproduced all three effects.

In the model, "thinning" arises from the process of pruning, which stochastically removes connection weights below a certain magnitude. For the lower SES individuals, pruning removed connections more quickly because lower levels of stimulation caused the weights to grow less strong, rendering them more vulnerable to pruning. The high-ability networks tended to be larger (see above, larger is always better). Larger networks tended to have smaller connection weights, because the required function was spread across more connections.[4] Smaller weight size again left these larger networks vulnerable to faster pruning. In this way, in the model, both low SES and high IQ produced faster thinning because of the common causal factor of connection-size-dependent pruning, even while low SES was linked to lower IQ in the same model. Only the Stimulation condition captured this effect because in the Structure conditions it was high SES individuals—manifested as larger networks with attendant smaller connections—that experienced faster connection loss.[5] Computational implementation, therefore, demonstrated how this paradox can be resolved under certain conditions.

6. Conclusion

In this work, we used a highly simplified multiscale population-level computational model to capture a diverse set of findings on SES-linked environmental effects on cognitive and brain development, integrating them with findings on intelligence and on the genetic contribution to individual differences in brain and behavior. The model provided support for both prenatal brain development and postnatal cognitive stimulation as proximal pathways of environmental influence through which distal differences in SES influence cognitive and brain development. Nevertheless, it is important not to simplify the effects of SES into a single dimension—as Farah (2017) argues, SES is a social construct that corresponds to a complex set of partially aligned environmental influences reflecting the contribution of possible advantages, disadvantages, and adaptations that children experience in being raised in physical and social environments with different levels of economic (dis)advantage. The work presented here sought to identify effects on brain and behavior that the operation of certain causal pathways would predict, under a particular set of implementation decisions. In doing so, we demonstrated how mechanistic accounts can contribute to the consideration of interventions aimed at closing environmentally produced gaps in developmental outcomes.

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Notes

1 Estimated via the *mult* bivariate heritability modeling application: Purcell, S. (2002). Behavioural Genetics Interactive Modules. http://zzz.bwh.harvard.edu/bgim/tuts/mult.exe. Accessed September 10, 2020.
2 For reference, the original model of SES effects on English past tense formation that formed the starting point of the current work was calibrated against a sample of 400 twin 6-year-old children, which was oversampled for risk of developmental language disorder (Bishop, 2005). The condition of the model that best matched the empirical data had the same range of genetic variation as the Stimulation condition here, but variation in cognitive stimulation between 60% and 100% of the training set (Thomas, Forrester, & Ronald, 2013). For the sample of 6-year-old children, individual differences were highly heritable, with MZ correlation of .67 and DZ correlation of .11, and there were unique environment effects of over 30% of variance.
3 Thomas (2016) showed that developmental increases in heritability could be an intrinsic property of the current model rather than requiring external manipulation of the training set. However, the heritability × age interaction was not found under the condition of wide variation of environmental influence employed in the current simulations, only with stimulation varying between 60% and 100% of the training set.
4 Fig. S6 illustrates this relationship, plotting total connection number against average magnitude.
5 Detailed in Fig. S7.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Supplementary material
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