Modeling socioeconomic effects on the development of brain and behavior

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Abstract

We used a population-level connectionist model of cognitive development to unify a range of empirical findings on the influence of socioeconomic status (SES) on behavior and brain development. The model captured qualitative patterns of *development* in behavior and brain structure, including reductions in connectivity across development (gray matter, cortical thickness) as behavioral accuracy increases. *Individual differences* in SES were implemented by altering the level of stimulation available in the environment. At the brain level, the model simulated non-linear effects of SES on cortical surface area (Noble et al., 2015), and faster cortical thinning across development in children from lower SES backgrounds (Piccolo et al., 2016). At the behavioral level, the model simulated the effect of SES on IQ, whereby gaps are observed to widen across development (von Stumm & Plomin, 2015). The model's main shortcoming was insufficient growth in connection magnitude across development in lower SES groups, implying that some aspects of the growth of connection strengths may be maturational (e.g., myelination) rather than experience dependent.

Keywords: socioeconomic status, brain, behavior, connectionist networks, multi-scale models, population modeling

Introduction

Differences in socioeconomic status (SES) have marked effects on cognitive development (Farah et al., 2006). These effects are not uniform across all areas of cognition and are stronger in the development of language and cognitive control (executive functions), where lower scores are observed in children from lower SES families. SES effects have been observed on intelligence (IQ) and indeed, it has been reported that gaps between children widen across development (von Stumm & Plomin, 2015; see Figure 1). SES refers to a marker for multiple potential causal pathways acting on cognitive development, among them effects on prenatal brain development, post-natal nurturing, and post-natal cognitive stimulation (Farah, 2017; Hackman, Farah & Meaney, 2017).

Recent work in neuroscience has focused on the impact of SES on measures of brain structure, demonstrating that cortical surface area and cortical thickness in children and adolescents show small but reliable associations with differences in family income and parental education; in some cases, associations have been observed between SES and the size of particular brain structures, such as the hippocampus and amygdala (e.g., Noble et al., 2015). Although small in size, these effects can be non-linear: for example, while lower SES is linked with reduced cortical surface area, the impact is larger for the lowest SES groups (Figure 2). Moreover, effects on brain structure are strongest in areas linked with language (temporal) and executive functions (prefrontal); and measures of cortical surface area (but not thickness) have been shown to mediate the relationship between SES and behavior (Noble et al., 2015). SES can be seen to influence the *rate of change* of brain structure over development. The cortex usually thins from mid-childhood onwards. In children from low SES backgrounds, thinning was observed to be faster. Piccolo et al. (2016) found that while cortical thickness showed no main effect of SES, it thinned more quickly in lower SES children; conversely, cortical surface area was reduced in the lower SES children, but showed similar rates of change across SES groups. Neuroscience data, then, confirm the impact of SES, but do they point to the causal pathways by which it operates?

Two challenges present themselves. First, we need a mechanistic account to explain how environmental influences produce linked effects on brain and behavior, which would provide a basis to evaluate competing accounts about causal pathways. Second, any putative causal explanation of SES effects must accommodate a range of other empirical phenomena: on developmental changes in brain structure, on the relationship between cognitive ability and various measures of brain structure, and on the origin of individual differences. The main qualitative patterns that must be captured are as follows.

First, although behavioral accuracy typically increases across development, this is not the case for all measures of brain structure: some measures increase (white matter volume, cortical surface area) but others decrease following a peak in early or mid childhood (gray matter volume, cortical thickness) (e.g., Giedd et al., 1999; Sowell et al., 2004). The mechanisms that drive these changes are still debated, but include myelination and pruning of local connectivity (synapses, dendrites, axons), but not generation or loss of neurons.

Second, although environmental measures such as SES predict individual differences, a large proportion of variance in cognitive ability, brain structure, and change in brain structure across development is predicted by the genetic similarity between people – that is, these phenotypes are

highly heritable (Plomin et al., 2013). Heritability may be modulated by SES: it has been observed that in individuals from low SES backgrounds, the heritability of IQ can be reduced (e.g., Tucker-Drob & Bates, 2016).

Third, brain structure is correlated with intellectual ability, with one meta-analysis showing correlations of 0.1-0.3 between brain volume and IQ (McDaniel, 2005). Ritchie et al. (2015) found that brain volume explained 12% of the variance in general cognitive ability, cortical thickness another 5%, and all structural measures together up to 21% of the variance. These individual differences data imply that having more neural resources is better for cognition. IQ is also related to the rate of thinning of the cortex with age (Shaw et al., 2006). Higher IQ is associated with faster thickening of cortex across early childhood, and then faster thinning of cortex from mid-childhood onwards. Since cognition improves as gray matter reduces, the developmental data imply that having fewer neural resources is better for cognition. This inconsistency is rendered more puzzling by the observation that faster thinning of the cortex is linked with lower SES (Piccolo et al., 2016). Lower SES is associated with lower IQ (von Stumm & Plomin, 2015). How can higher IQ and lower SES both be linked to faster thinning of cortex, when higher IQ is itself associated with higher SES? This complex set of effects is summarized in Table 1.

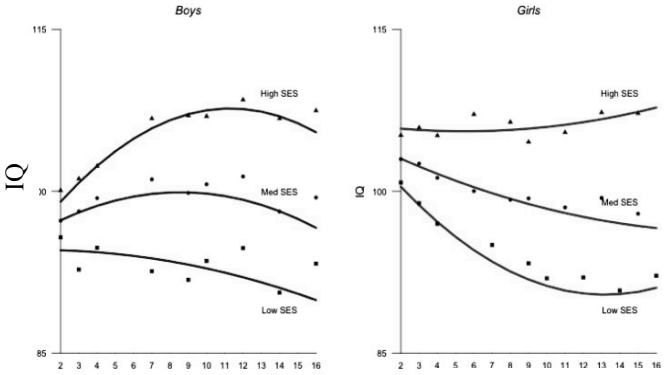

Figure 1: SES gaps in intelligence widen across development (von Stumm & Plomin, 2015)

In the current work, we use a multi-scale model to try and unify this complex pattern of data. The model is based on an artificial neural network (ANN) trained with backpropagation. In a multi-scale model, constrains are included at several levels of description (Thomas, Forrester & Ronald, 2016). Crucially, because the data concern both development and individual differences, it is necessary to simulate a population of individuals, and to model the influences on development that produce individual differences. Because the data span behavior, brain, SES, and genetics, the model must have analogues of each of these in its design.

In connectionist 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, 2008). Thomas (2016) argued

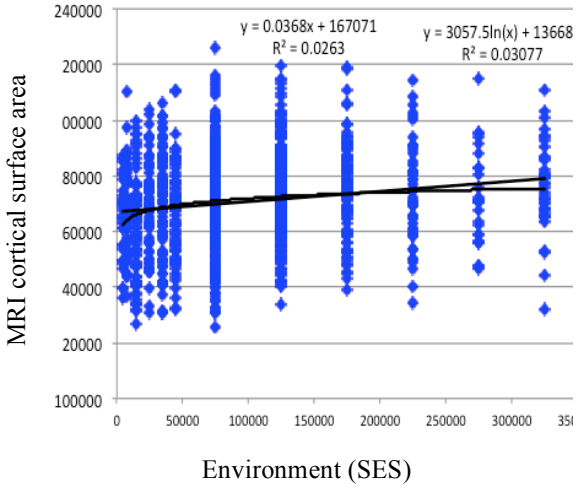

Figure 2: The link between cortical surface area and family income (data re-plotted from Noble et al., 2015).

Table 1: List of empirical phenomena to be simulated

1.	Behavioral accuracy increases across development
2.	Some measures of brain structure increase across development (white matter, cortical surface area)
3.	Some measures of brain structure reduce across development (gray matter, cortical thickness)
4.	Lower SES is associated with lower IQ and gaps widen across development
5.	Lower SES is associated with reduced cortical surface area, with larger effects at lowest SES levels
6.	Lower SES is associated with faster thinning of the cortex over development
7.	Lower SES is associated with reduced cortical surface area but no modulation of rate of development
8.	Cortical surface area partially mediates the relationship between SES and behavior
9.	Individual differences in behavior and brain structure are highly heritable
10.	Low SES can reduce the heritability of IQ
11.	Brain volume correlates with IQ
12.	Across development, higher IQ is associated with faster thickening and then faster thinning of the cortex

that with a simple addition – the onset of pruning of unused connectivity after a certain point in training – these models could give plausible analogues to measures of brain structure, where the total number of connections would serve as an analogue to properties that decrease over development (gray matter, cortical thickness) – under the view that unused connections are pruned away, causing a

loss of volume; and the combined magnitude of connection weights (excitatory and inhibitory) would serve as an analogue of properties showing increases (white matter, cortical surface area) – under the view that retained connections are optimized through myelination, causing an increase in volume. We use the same scheme here.

To capture genetic influences on behavior and structure, each network must have a genome and genomes must vary between individuals. To the extent that cognition is seen as information processing in the brain, genetic effects must translate to influences on neurocomputational properties. Accordingly, Thomas et al. (2016) used a method to simulate individual differences where the neurocomputational parameters of an ANN (e.g., number of hidden units, learning rate) were encoded in an artificial genome. Genetic variation produced parameter variation. In behavior genetics, the heritability of a phenotype such as behavior or brain structure is usually assessed using the twin design, where more heritable phenotypes show greater similarity between monozygotic (MZ) twins than dizygotic (DZ) twins. MZ twin networks can be simulated by networks with 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 networks versus DZ networks.

SES can plausibly be implemented in several ways (Thomas, Forrester & Ronald, 2013): it might influence how a network is constructed (equivalent to prenatal effects on brain development); it might influence the information on which the network is trained (equivalent to differences in levels of cognitive stimulation during post-natal development); or it might influence both factors. In the following simulations, we evaluated a model that implemented SES as differences in the richness of the training set.

An ANN trained with backpropagation has very limited biological plausibility. We should therefore be clear what are our key assumptions in relating measures of network structure to measures of brain structure. They are as follows: (1) neuron number is fixed so that changes in structure reflect changes in connectivity; (2) structural measures that increase over development (cortical surface area, white matter) reflect increases in connection strength, while structural measures that decrease over development (cortical thickness, grey 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); (6) we did not include an assumption that connection growth 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 will serve as a test of these assumptions.

Method

The following simulations use a base model taken from the field of language development, addressed to the domain of English past-tense formation. Here, the model was employed in an illustrative setting, intended only as an example of a developmental system applied to the problem of extracting the latent structure of a cognitive domain through exposure to a variable training environment. The intention was to capture qualitative characteristics of the empirical data rather than to exactly calibrate variances from genetic and environmental sources to fit empirically observed estimates of heritability in certain populations. In that capacity, the past tense accuracy of the networks was taken as a metric of behavioral development, and of intelligent behavior more widely (that is, of the type measured by cognitive ability tests). However, the base model has been used to specifically simulate data on the influence of SES on children's past-tense acquisition (Thomas et al., 2013). Full details of the current simulation can be found in Thomas (2016).

Network architecture: The basic model was a 3-layer backpropagation network, with 57 input and 62 outputs. The process of network growth was not modeled, only the outcome of this process. The number and size of initial connections was influenced by several factors, including number of weight layers, sparseness of connectivity, and range of initial random 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 free to vary between individuals. Pruning onset varied between 0 epochs and 1000 epochs, where 1000 epochs was 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, fourteen 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).

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 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.

Implementation of SES differences: Each simulated child was raised in a family with a given level of language stimulation, taken to be correlated to the family's SES (Hart & Risley, 1995). A family quotient parameter 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, networks could in principle be exposed to very few training patterns (see Thomas, 2016, for discussion).

Implementation of genetic differences: Differences in learning ability arose from the net effect of small variations in all the neurocomputational parameters, under a polygenic model of intelligence (Thomas, 2018). For this simulation, all variation in these parameters was considered to be under genetic control. There was a random association of family quotient to genotype, that is, we did not simulate gene-environment correlations.

Simulation design: A population of 1000 networks was created in sets of pairs, either MZ or DZ twins. Each network was trained for 1000 epochs. Performance on the training set (regular and irregular verbs) and two network measures, total number of connections and magnitude of connections, were assessed across training.

Results

Developmental changes in behavior: Figure 3 shows the monotonic improvement in accuracy in regular and irregular (vowel-change) verbs across training, averaged across the whole population (Table 1, #1).

Developmental changes in brain structure: Figure 4 plots the change in the magnitude of connections (gradually increasing) and the total number of connections (a non-linear decline) across training, averaged across the whole population. The plot captures the increase and decrease of different structural measures (Table 1, #2 and #3).

SES effects on behavior: The behavioral scores of the networks were split by their SES (upper quartile, family quotients >.75, lower quartile family quotients <.25). At each measurement point, the population distribution in accuracy values was used to convert accuracy to IQ scores, by deriving the population mean and standard deviation and transforming these to a mean of 100 and standard deviation of 15. Figure 5 plots developmental trajectories of IQs split by upper, lower and middle two quartiles. The plot captures a widening gap between the groups (Table 1, #4). In the simulation, this is the result of non-linear developmental trajectories, whereby the lower SES groups show earlier plateauing of performance.

SES effects on brain development: Figure 6 shows a scatter plot of each network's connection magnitude against SES (family quotient value), after 100 epochs of training. The simulations demonstrate a reliable association of SES to network structure. The pattern of a small effect size and non-linear relationship capture that shown in Noble et al.'s (2015) cortical surface area data, with larger reductions in area at the lowest SES levels (Table 1, #5).

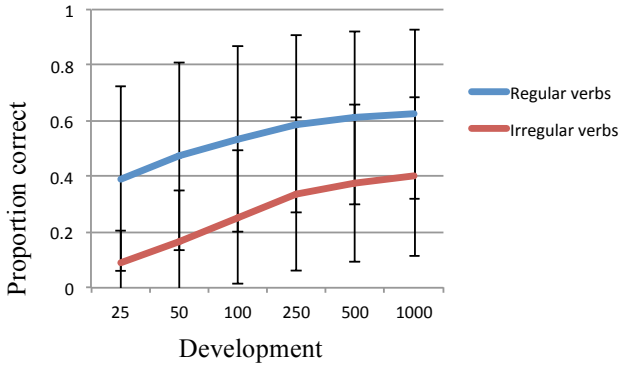

Figure 3: Average population development for two behaviors, regular verb and irregular verb performance. (Error bars show standard deviations)

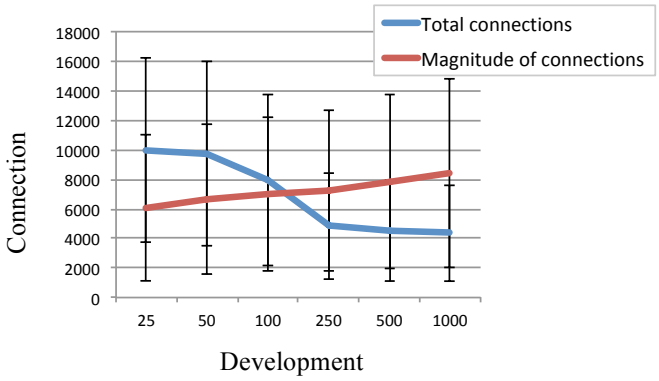

Figure 4: Average population changes in connection magnitude and number over development. (Error bars show standard deviations)

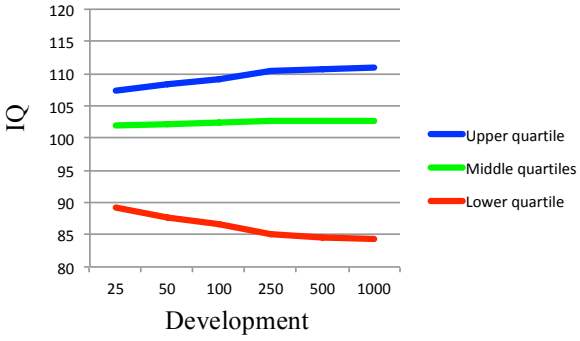

Figure 5: Development of behavior split by SES quartile.

Figure 7 separates the networks into the upper quartile and lower quartile according to SES and plots change in total number of connections across development (for simplicity, linking two points in training, epochs 25 and

250). There was no reliable main effect of SES on connection total (p=.547), but a reliable interaction, whereby connection total reduced more quickly in the lower SES quartile (F(1,498)=15.42, p<.001, η_p^2=.030). This occurred because lower SES networks received less stimulation, causing less strengthening of connections, and in turn greater vulnerability to later pruning processes. The result captures Piccolo et al.'s (2016) observation that cortex thins more quickly in children from a lower SES background, without overall differences in cortical thickness between groups (Table 1, #6).

Figure 8 plots the equivalent simulation data for connection magnitude, split by SES quartile. The model shows a main effect of SES, with smaller magnitudes in low SES networks (F(1,498)=13.33, p<.001, η_p^2=.026), but also an interaction, where magnitude in low SES networks improves much more slowly ((F(1,498)=150.88, p<.001, η_p^2=.233). The first effect captures the smaller cortical surface area observed by Piccolo et al. (2016) for lower SES children, but the interaction does not accord with the empirical data – SES does not modify rate of change of cortical surface area (Table 1, #7, not captured).

Brain structure mediates relationship of SES to behavior: Noble et al. (2015) found that cortical surface area mediated the relationship between SES and behavior but thickness did not. In the model, we observed increasing correlations between SES, connection magnitude, and behavior across training, such that a mediation effect was detectable by the end of training. Figure 9 shows that connection magnitude mediated associations between SES and regular verb performance (β=0.05, t(998)=8.44, p<.001, CI [.04; .07]). The Sobel test was significant, confirming partial mediation (Sobel-z=7.98, p<.001). Per Noble et al.'s findings, the analogue of thickness, connection number, did not show the mediation effect. This is because in the model, the correlation of SES to connection number did not reach significance (Table 1, #8).

Heritability of individual differences: at 100 epochs, the correlations between twin pairs were as follows: Regular verb performance: MZ=.99, DZ=.61; irregular verb (vowel change) performance: MZ=.97, DZ=.49; connection magnitude: MZ=1.00, DZ=.44; connection total: MZ=1.00, DZ=.33. Where MZ correlations are higher than DZ correlations, this implies genetic influence. The difference between the correlations can be used to estimate the heritability of the phenotype. Under an additive model, the respective heritabilities are .76, .97, 1.12, and 1.34 (that the latter values exceed 1 shows that the genetic effects violate an additive model and there are dominance effects operating). These values are higher than observed for behavior and measures of brain structure (Plomin et al., 2013). The simulations included no measurement error, which would appear as an environmental effect unique to each individual. Nevertheless, these high estimates of heritability imply the assumption that all neurocomputational parameter variation is under genetic control is not plausible, and that the environment contributes to variation in parameters (perhaps during prenatal brain development). However, the observed high heritabilities meant that effects of SES on brain and behavior were successfully simulated against a background of strong genetic influence on both measures (Table 1, #9).

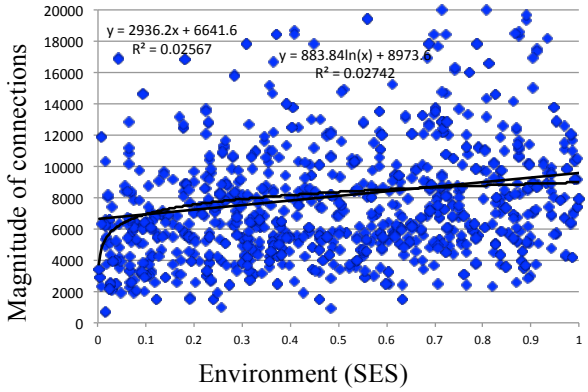

Figure 6: Connection magnitude versus SES

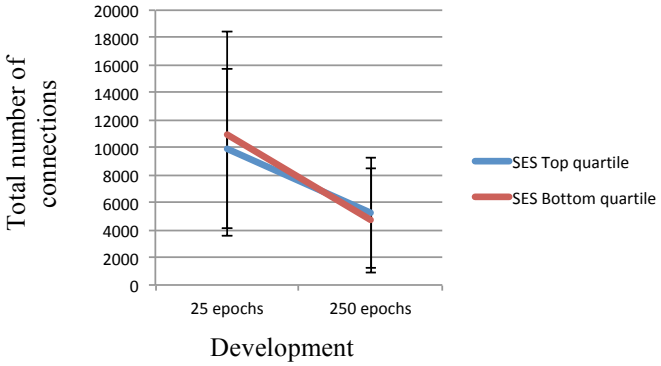

Figure 7: Change in number of connections across development, split by SES. (Error bars = STD)

Estimates of heritability were also observed to differ between upper and lower SES quartiles, with the lower SES quartile showing reduced heritability as the impoverished training set – rather than the neurocomputational parameters – became the limiting factor on performance. For example, for irregular verbs at 100 epochs, the upper quartile showed MZ correlation of .97, DZ .35, while in the lower quartile, these values were .95 and .60. The reduced gap between MZ and DZ correlations shows reduced genetic influence in the low SES group (Table 1 #10).

Relation of intelligence to brain structure: The 'ability' of each network was assessed based on its behavior. We chose to assess this based on irregular (vowel-change) verb performance at an early point in development (50 epochs), which gave good sensitivity to discriminate between individuals. At 100 epochs, the correlation of ability with total connections was .352, and with magnitude was .371. This captures the empirical observation of the small correlation between brain size and intelligence (Table 1, #11).

Based on the ability measure, we derived upper quartiles (top 25%) and lower quartiles (bottom 25%) of ability. Figure 10 shows the change in total number of connections between two points in development, epoch 25 and epoch 250. At epoch 25, high ability networks had reliably more connections ($t(458)=8.74$, $p<.001$, Cohen's $d=.81$). We did not simulate the growth of connectivity, only the outcome of this process. The higher peak captures the outcome of putative faster thickening of cortex across development for children with higher IQs (Shaw et al., 2006). Across development, connection number fell reliably more quickly in high ability networks than low ability networks ($F(1,458)=31.60$, $p<.001$, $\eta_p^2=.065$). The faster fall is a side effect of the higher peak – the greater ability arises from the greater computational power of having a larger network, while larger networks experience faster pruning. The result captures the observation by Shaw et al. (2006) that cortex thins more quickly in children with higher IQ (Table 1, #12).

Discussion

The model was successful in qualitatively capturing 11 of 12 target phenomena linking SES, IQ, brain development and behavioral development. The model used simple error-driven backpropagation networks, where connection strengths are altered to improve performance. Links to brain structure were established by adding a pruning process that, after a certain point early in development, removes unused connections. Measures of network connectivity gave analogous fits to brain structure measures that either show increases with age (white matter, cortical surface area) or decreases (gray matter, cortical thickness). The match of simulation and empirical data supports the view that these brain measures represent the results of experience-dependent strengthening of connectivity combined with intrinsic processes for connectivity growth and loss, where connectivity loss is dependent on the extent to which previous experience has strengthened connections.

The successful simulation of SES patterns in behavior and brain support the view that a key element of these effects is the level of cognitive stimulation. However, this is unlikely to be the full effect, and other environmental influences on prenatal and postnatal development undoubtedly contribute (see, e.g., Betancourt et al., 2016, for SES-related gray matter differences observed in babies at 1 month of age, where experience-dependent effects have had little time to act). Extension of the model presented here is necessary to explore the possibility that environmental effects on brain growth may interact with, and indeed may be correlated with, differences in cognitive stimulation.

The model failed in two regards. First, it did not capture the observed absence of SES effects in the rate of change of cortical surface area (Piccolo et al., 2016). The model did not show enough strengthening of connectivity across development in the low SES group. This implies that one of the assumptions of the model – that connection strength increases can only be experience dependent – is incorrect, and that there is a maturational contribution to connectivity increases (such as myelination). Second, its estimates of heritability were too high for individual differences in behavior and brain. In part, this is due to the absence of measure error in the simulations. But, consistent with above comments, it also demonstrates another initial assumption of the model is incorrect, that neurocomputational parameters are solely under genetic influence.

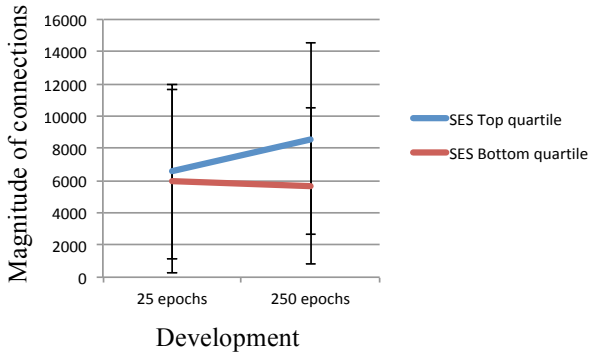

Figure 8: Change in magnitude of connections across development, split by SES. (Error bars = STD)

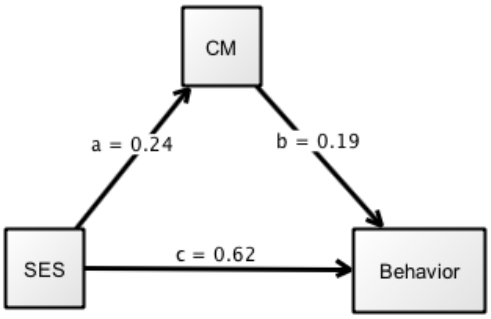

Figure 9: Partial mediation between connection magnitude (CM), SES and behavior (regular verb accuracy)

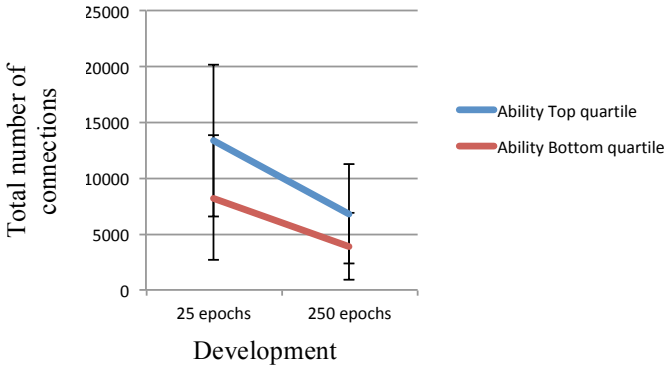

Figure 10: Change in number of connections across development, split by behavioral ability. (Error bars = STD)

A future extension of the model should investigate environmental influences on specifying network parameters, such as initial network growth. It should also be noted that the model set out only to simulate qualitative patterns, not to calibrate against precise ranges of genetic or environmental

variation, or to capture particular population mean levels of behavior at a given point in development. Some assumptions could be questioned, such as the extreme deprivation implied by training sets that could vary down to including no patterns.

Implementation of a mechanistic model provides the benefit that it can reconcile apparent paradoxes in the empirical literature. Why are high IQs associated with having a bigger brain (as if more neural resources were better for cognition) but also associated with faster gray matter loss and cortical thinning (as if fewer resources were better)? The answer is that the network size is driving ability (so more is always better), but that a higher peak of network size is then associated with faster connectivity loss during pruning of unused resources (in the manner that higher mountain peaks have steeper sides). How can faster cortical thinning be simultaneously associated with higher IQ and lower SES (which is associated with *lower* IQ)? The answer is that in the higher ability networks, there are more spare resources to be lost during pruning so thinning is faster; in low SES networks, the small training set (equivalent to lower cognitive stimulation) produces less strengthening of connectivity so that connections are more vulnerable to loss when pruning starts, leading once more to faster thinning. In other words, rate of change of structure isn't a direct marker of ability; ability is delivered by the full computational properties of the network and its developmental origins, not proxy measures like cortical thickness.

The model presented here is highly simplified, employing a single artificial network with very restricted biological plausibility. The range of the phenomena that the model captures probably reflects the fact that the existing observations we have on behavior, brain structure, and SES give limited insight into the detailed neural processes underlying behavior, development, and environmental influences. Nevertheless, we argue here for the importance of building multi-scale models that integrate individual differences within a developmental framework, and which can therefore evaluate causal mechanisms linking SES, brain and behavior. With causal, mechanistic accounts in hand, we are better able to consider interventions to ameliorate the impact of poverty and deprivation on children's development. The results here point to the importance of cognitive stimulation, and encourage interventions that seek to enrich that stimulation for children from poor backgrounds.

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