# 10

# USING NEUROCOMPUTATIONAL MODELLING TO INVESTIGATE MECHANISMS UNDERLYING SOCIOECONOMIC STATUS EFFECTS ON COGNITIVE AND BRAIN DEVELOPMENT

Michael S. C. Thomas

## Introduction

Poverty is about people's lives. Inequality, one of its major drivers, is a social issue. Cognitive neuroscientists have become increasingly interested in how being raised in poverty impacts children's brain and cognitive development. But how can it be useful to reduce people to instances of individual brain function? Poverty is the result of social structures and therefore a focus on neuroscience would appear to be a distraction (Farah, 2017).

There are at least three reasons why a cognitive neuroscience approach may be useful. First, as we shall see, socioeconomic

status (SES) – typically measured by a combination of family income, parental occupation, and parental education – has been found to correlate with differences in brain structure, brain function, cognitive ability, and educational achievement. However, many factors co-occur with low SES (see, e.g., Hackman et al., 2015). Mothers may be more stressed, have poorer diets, and more drug exposure while pregnant; children may be raised in less nurturing, more polluted, and more dangerous environments; there may be less social or neighborhood support, poorer schools, and less supportive attitudes to education; children may have fewer resources and opportunities for cognitive stimulation and learning. This array of factors may not all be equally responsible for producing health, cognitive, and educational outcomes. If the biological causal pathways of SES effects are identified, this can help to target the most efficient interventions to alleviate the downstream effects of poverty. Such interventions offer short-term measures, while the longer-term social goal of reducing poverty can be pursued.

Second, there is a straightforward sense in which evidence that poverty affects the brain in measurable ways is a powerful message to policymakers. A brain image is worth a thousand words. Brain data, however, represent a double-edged sword, because policymakers may be liable to think that effects observed on brain structure and function are then immutable. They are not, because we know that the brain is plastic, and behavioral interventions can improve outcomes. A study of brain mechanisms must also, therefore, emphasise this message and seek to identify pathways to remediate observed deficits.

Third, work in education, the social sciences, and the cognitive sciences has generated a large body of empirical data on outcomes that are *correlated* with SES. But these correlational data are open to misunderstanding and misinterpretation if the

underlying mechanisms are not understood. Here are three examples of empirical data and three respective possible interpretations.

(1) Gaps in children's IQs (cognitive ability) across levels of SES are evident from infancy and these gaps widen through childhood and adolescence (von Stumm & Plomin, 2015). *Some process must be getting worse across childhood to make the gaps widen.*

(2) When children are split into brighter and less bright groups around two years of age and then followed up, over time brighter children from poorer backgrounds fall back compared to their peers, and by age 10, they have been overtaken by less bright classmates from richer families (Feinstein, 2003). *With age, children's rank in their class is increasingly constrained by environmental factors such as SES*. From data like these, policymakers have concluded that early potential is lost through environmental factors such as poor childcare, poor early years education, poor schooling and lack of access to health services (HM Government, 2003).

(3) One way to measure social mobility is to assess whether children reach a higher level of educational attainment than their parents. On this measure, however, at least half the variability can be linked to genes (Ayorech et al., 2017). *Genetics would seem to place limits on how much social mobility can be influenced by interventions*. Do genes restrict whether children can escape poverty through education?

This chapter outlines one methodology within cognitive neuroscience to investigate the mechanisms underlying SES effects on brain and cognition: multi-level neurocomputational models of cognitive development. The model presented here was applied to each of the above empirical effects. It generated alternative interpretations of each set of empirical data (Thomas, Forrester & Ronald, 2013; Thomas et al., in preparation; Thomas & Meaburn, in preparation).

## SES effects on brain and cognitive development

We begin with a (very) brief overview of the existing empirical literature. We know that differences in SES have marked effects on cognitive development (Farah et al., 2006). These effects are not uniform across all areas of cognition, but are particularly marked in the development of language and cognitive control (often referred to as 'executive functions'). Hackman and Farah (2009) considered these differential effects in terms of relatively independent, anatomically defined neurocognitive systems in the brain. The strongest effects of SES were observed for the language system (left perisylvian regions) and the executive system (prefrontal regions, decomposed into working memory system [lateral prefrontal], cognitive control [anterior cingulate] and reward processing [ventromedial prefrontal]). SES explained 32% of the variance in the language composite behavioural measure, 6% in cognitive control, and 6% in working memory.

Effects of SES have been observed on measures of brain structure using magnetic resonance imaging. For example, Noble et al. (2015) reported effects of family income levels on cortical surface area in a cross-sectional sample of 1099 children in the USA aged 3-20 years. The relationship was non-linear, with the strongest effects observed in the lowest income families; differences in income at higher levels were associated with smaller changes in cortical surface area. However, SES only explained a few percentage points of the variance; there was a great deal of variation in brain structure measures not explained by SES. Notably, the strongest effects of SES on brain structure were found in regions supporting language, reading, executive functions, and spatial skills, consistent with behavioural evidence.

SES has also been found to impact neural development at much earlier ages. Betancourt et al. (2016) examined the relationship between SES measures (income-to-needs ratio and

maternal education) in a sample of African-American female infants aged 5 weeks. They observed that lower SES was associated with smaller cortical grey and deep grey matter volumes, pointing to the biological embedding of adversity very early in development.

The link between brain structure and function is indirect and not well understood. Nevertheless, researchers have observed differences in brain function associated with SES both with functional magnetic resonance imaging (regional oxygenated blood flow differences) and with electrophysiology (measurement of voltage potentials on the scalp associated with neural activity). For example, using functional magnetic resonance imaging, Raizada et al. (2008) found that the weaker language skills observed in 5-year-old children from lower SES backgrounds were associated with reduced hemispheric functional specialisation in left inferior frontal gyrus. Specialization to the left hemisphere is a marker of the functional maturation of language systems. Using electrophysiology with a sample of 3-8 year olds, Stevens, Lauinger, and Neville (2009) demonstrated reduced neural signatures of selective attention in children from lower-SES families (indexed by maternal education). In an auditory processing task where the children had to attend selectively to one of two simultaneously presented narrative stories, the neural processing differences that characterised the lower-SES children were related specifically to a reduced ability to filter out irrelevant information.

These few examples illustrate the general methods from a fast growing neuroscience literature (for wider reviews of structural and functional brain imaging and SES see Farah, 2017; Pavlakis et al., 2015). Importantly, cognitive neuroscientists do not yet understand the causal pathways of these cognitive and brain effects, not least because the SES measure represents a distal cause and does not isolate the proximal causes that influence cognitive

and brain development. Some differences associated with low SES may represent *deficits* (e.g., poorer brain development caused prenatally by poor maternal nutrition or postnatally by chronic stress). Others may represent *adaptation*s (e.g., apparent poorer selective attention may reflect higher vigilance appropriate to a more dangerous environment; impulsivity may reflect maximising short-term rewards because long-term rewards have proved unreliable).

Hackman, Farah, and Meaney (2010) classed potential causal mechanisms into three types, based on naturalistic research with humans and experimental research with animal models: (1) those operating prenatally on fetal development, (2) those affecting postnatal parental nurturing, and (3) those affecting postnatal cognitive stimulation. Explanatory models tend to distinguish what is lost from lower SES families (resources, good nutrition, learning opportunities) from what is added (stress, toxins, childhood adversity experiences) (Sheridan & McLaughlin, 2016). Causal explanations are likely to be complex: all three classes of factors could be responsible, or combinations could differ per brain system. The combination of factors may depend on details of the specific population and local factors, in terms of absolute levels of resources/poverty, where the economic and environmental restrictions lie in a particular society, and the relative levels of poverty (inequality).

Against this background of (hopefully) remediable environmental effects, we also know that in Western societies, a fair proportion of children's variability in cognitive and educational outcomes, and indeed brain structure, can be predicted by their genotypes – that is, abilities are 'heritable' (Plomin et al., 2016). The term heritable is often misunderstood to relate to necessary outcomes (because children's genes aren't changeable) but this interpretation is incorrect. In different environments, genetic

effects may be increased or decreased: observed genetic effects are not inevitable or deterministic. They show what is, not what can be. Nevertheless, we can take measures of heritability as current summary statistics: given the current range of family and educational environments that children are raised in, and which shape the world they can explore, heritability is a statistic that capture how much variance is currently being predicted by genetic similarity.

There has been a flurry of new findings with respect to life outcomes, SES and behavioural genetics. For example, researchers have reported that educational achievement is 'highly' heritable, with as much as 60% of the variance in examination results in 16 year olds explained by genetic similarity (Krapohl et al., 2014). These genetic effects appear general across topics rather than specific to different academic subjects (Rimfeld et al., 2015). Direct measures of DNA variation have pointed to regions of the genome associated with academic achievement, albeit with coarse educational measures as the outcome (years of schooling completed) and smaller amounts of variance explained (e.g., 11-13% variance; Lee et al., 2018). Notably, variations in SES have been reported to partly align with genetic variation (e.g., Trzaskowski et al., 2014). Moreover, social mobility – where an individual's SES differs from that of their parents, such as in educational attainment – has itself been reported as partly heritable, with one study observing that just under half of the variance in social mobility was linked to genetic variation (Ayorech et al., 2017), and another study reporting that direct measures of DNA variation could explain around 3% of the variance in upward educational mobility (Belsky et al., 2018).

Evidence of the role of genetic variation in influencing cognitive, educational, and life outcomes, and of the possible correlations between the genetic variation and SES gradients,

drives the debate between *social causation* and *social selection* accounts (Farah, 2017). Under a social causation account, SES effects and their persistence across generations are driven by the environments in which children are raised. Under a social selection account, SES-related differences in brain and cognition are under genetic control, with population stratification of genotypes according to SES.

Our concern here is not the competing merits of these accounts, but merely the challenge posed by respective data on the roles of environmental factors and genetic factors on brain and cognitive development. How can these bodies of empirical data be reconciled into a coherent causal account? Given the complexity and multi-faceted nature of both brain development and cognitive development, how can we begin to formulate and test competing explanations for the pathways by which SES effects operate – and their implications for intervention? Even under a social causation account, one must accept the role of genetic variation in contributing to differences in outcomes. Even under a social selection account, one must accept that differences in experiences will influence development.

## Neurocomputational modelling

One method used in cognitive neuroscience to formulate and test causal accounts is computational modelling. Models can be formulated at different levels of description: of individual neurons, of circuits of neurons, or of whole brain systems. In each of these cases, models seek to capture empirical evidence on patterns of brain activation or anatomical structure. Models can also be formulated at a cognitive level: although certain constraints may be included from neuroscience about the nature of computation, the target is then to capture empirical data on high-level behaviour. Multi-level models include constraints from several levels of

description and seek to capture data both at the level of brain and behavior (Thomas, Forrester & Ronald, 2016). Models may be constructed to simulate the characteristics of the static properties of a system at a given point in time, or they may be constructed to capture developmental change, where trajectories of behavior are simulated as they alter over time (Elman et al., 1996; Mareschal & Thomas, 2007).

How might we construct a multi-level computational model to explain SES effects on brain and cognitive development? Minimally, we need to stipulate a neutrally constrained developmental mechanism which acquires a target behavior through interaction with a structured learning environment; we need to stipulate how growth of that developmental mechanism and interactions with the structured learning environment might alter as a consequence of variations in SES; and we need to stipulate separately how genetic variation might alter the properties of the developmental mechanism, for example in terms of how it grows, operates, and responds to stimulation. Thomas, Forrester, and Ronald (2013) began this line of research by constructing an artificial neural network model of the effects of variation in SES on language acquisition, focusing on the specific domain of inflectional morphology (that is, altering the sounds of words to change their meaning, such as in forming the past tense of a verb). The model was able to simulate how children's language skills altered across the SES gradient, as well as generating testable predictions about children's language outcomes (see also, Thomas & Knowland, 2014; Thomas, 2018, for the model's extension to considering delay and giftedness). Thomas, Forrester, and Ronald (2016) and Thomas (2016) showed how the same model, treated more abstractly, could be extended into a multi-level format, to incorporate a genetic level of description and indices of brain structure as well as behavior. In the following sections, we

demonstrate how the model can be applied to considering SES effects on brain and cognitive development (Thomas et al., in preparation; Thomas & Meaburn, in preparation).

*Model assumptions and simplifications*

A schematic of the model is shown in Figure 1. In the model, cognitive development occurs through the interaction of an experience-dependent mechanism with a structured learning environment. The mechanism is an artificial neural network, which embodies computational constraints from neural processing (Elman et al., 1996). These constraints are, respectively, a network of simple non-linear integrate-and-fire processing units, distributed representations of knowledge, associative error-driven learning altering network connectivity strengths and unit thresholds, and network development including phases of growth and pruning. The structured learning environment is drawn from the field of language development. The single processing structure is assumed to lie within a larger cognitive architecture but is not intended in this model to correspond to any specific brain region.

The mechanism learns input-output mappings that drive behaviour relevant to its domain. Accuracy of input-output mappings is used as a measure of behavioral performance. Structural properties of the artificial neural network, including the total number of connections and the total strength of excitatory and inhibitory connections, are used as analogues of brain structure measures such as cortical thickness, cortical surface area, grey matter volume, and white matter volume (Thomas, 2016).

Individual differences factors, such as SES and genetic variation are not considered in isolation but in terms of how they modulate the above species-universal mechanisms that underpin development across all children. In this sense, the model construes individual differences as operating within a developmental

framework (Karmiloff-Smith, 1998). Various options are available to implement the effect of SES: as a modulation of the level of stimulation available in the learning environment (see Thomas, Forrester, & Ronald, 2013); as a modulation of the growth of the network and its processing properties; or both of these effects operating in a correlated fashion (see Thomas et al., in preparation). Each network represents a simulated child undergoing development in a family environment. Each family is assigned a value, between 0 and 1, to represent its SES, which is then used to modulate the learning environment or the network structure.

Genetic variation is assumed to operate by influencing the neurocomputational properties of the processing mechanism, in terms of its capacity, plasticity, and noisiness of processing (these are broad characterisations of the role of a larger set of parameters, show in Table 1). Since behavioral genetic research on cognition has indicated that common genetic variation amounts to large numbers of small genetic effects on a wide range of neural properties, genetic variation is implemented via a polygenic coding scheme: an artificial genome contains sets of genes which each influence variation on a neurocomputational property (14 properties, each influenced by 8-10 genes); the combination of small variations across a large set of properties produces networks with a normal distribution of learning properties (Thomas, Forrester & Ronald, 2016, for details). The combination of simulated children with different learning abilities, interacting with environments with different levels of stimulation, produces a population of children with different developmental trajectories in both behavior and brain structure. At any point in development, cross-sections can be taken of behavior or structure across the population, and correlations derived to SES or genetic variation.

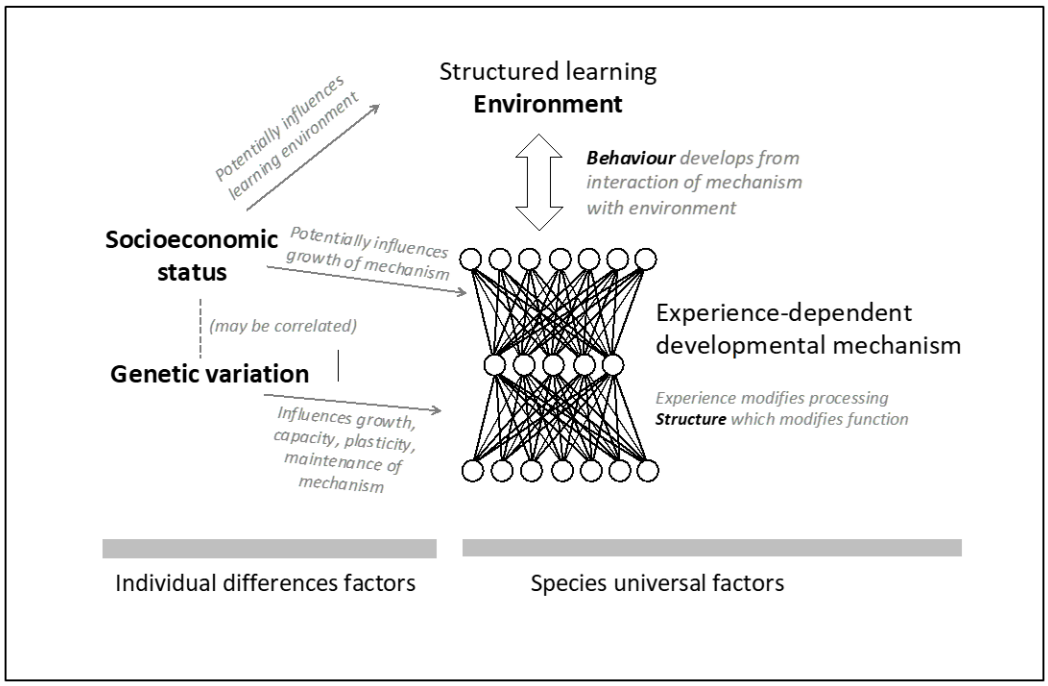


**Figure 1** – Structure of neurocomputational model simulating SES effects on cognitive and brain development. An experience-dependent developmental mechanism (artificial neural network) interacts with a structured learning environment to acquire a cognitive behavior. The multi-level model embodies constraints at the level of genes, brain structure (connections, units), behavior, and environment. Individual differences factors (SES, genetic variation) are considered with respect to how they modulate species universal mechanisms supporting cognitive development.

*Simulation design*

A single network was trained on its family-specific set of input-output mappings. Per its source cognitive domain, in this case the inputs were phonological representations of verb stems and the outputs were inflected forms of English verbs. Lifespan development corresponded to 1000 exposures (or 'epochs') of the network to the training set. The training set comprised a maximum of 500 input-output mappings. The development of 1000 individual children was simulated. Genomes were randomly initialised to produce genetic variation in learning ability across the population. Pairs of 'twin' networks were created which either

shared the same genome (identical) or shared 50% of genes on average (fraternal) and twin pairs raised in the same family. This design enabled the use of twin correlations to compute heritability levels. SES was allowed to vary widely across families to capture the potential effects of poverty. In the simulations described here, SES was implemented as modulation of the level of stimulation in the learning environment, and was allowed to vary between 0 and 1. A family with a value of 0.6 would generate a training set that only contained a (randomly sampled) subset of 60% of the full training set (see Thomas, 2016, for further details, including specification of neurocomputational properties and calibration of their range; results are reported for the G-wide E-wide condition in that paper).

*Simulation 1: SES effects on IQ change across development*

Thomas et al. (in preparation) first considered developmental trajectories of behaviour. The population was split into three groups, those in the upper quartile of SES (training sets with >75% of available experiences), those in the middle two quartiles, and those in the lowest quartile (<25% of available experiences). Figure 2(a) shows the latent growth trajectories of IQ for children from low, middle, and high SES groups in the empirical data of von Stumm and Plomin (2015), for around fifteen thousand UK children followed from infancy to adolescence. It shows diverging trajectories with age. The SES gap widens. Figure 2(b) shows simulated data of IQ scores in the model, where IQ was computed according to the population distribution at each measurement point [IQ score= ((individual performance – population mean)/population standard deviation X 15) + 100]. Figure 2(c) shows the developmental trajectories of performance without the transformation to IQ scores. The simulation is able to catch the

lower initial levels of performance at the youngest age, as well as the divergence of the trajectories across developmental time.

One might conclude from the empirical data that the conditions producing SES differences in cognitive development must worsen over time to produce the divergence. The simulations reproduced the diverging pattern with a consistent SES effect over time. In the model, divergence occurred due to non-linear trajectories of development. Increasing gaps between SES groups do not, then, necessarily imply worsening SES causal factors.

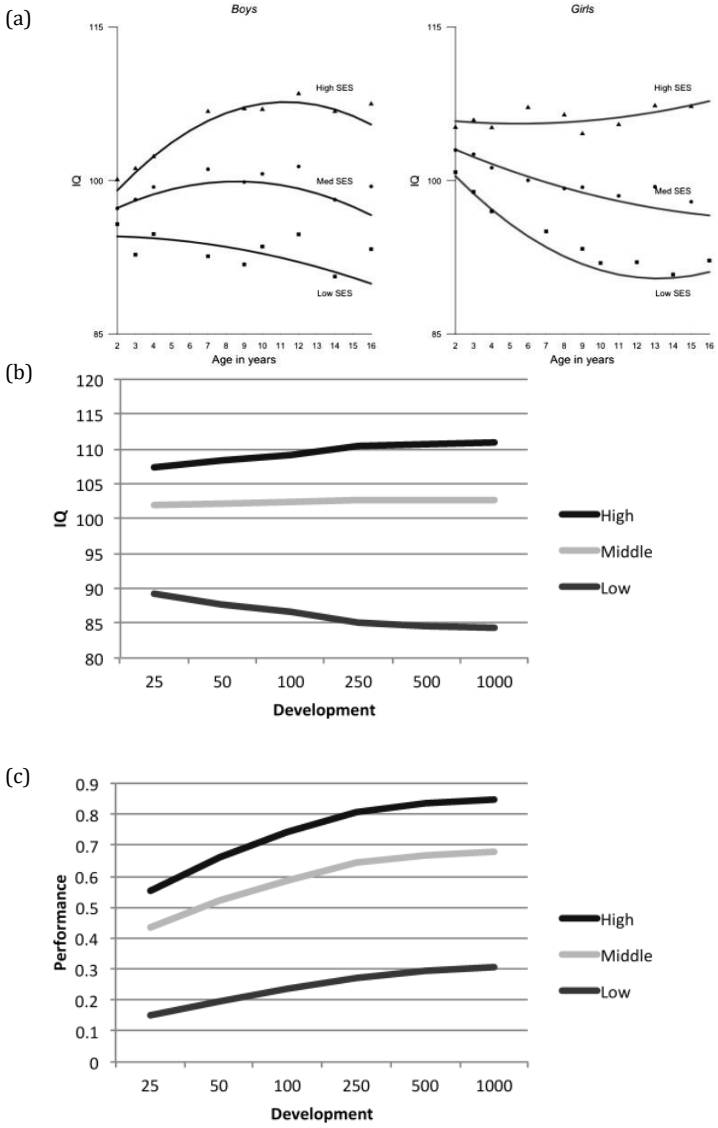


**Figure 2** – (a) Empirical longitudinal data from a UK sample of twins (N= 14,853 children) plotting IQ change over development from infancy to adolescence, split by socioeconomic status and shown separately by gender (reproduced with permission from von Stumm & Plomin, 2015). High SES = > 1 standard deviation (SD) above SES mean; low = < 1 SD below SES mean; middle = < 1 SD above SES mean and > 1 SD below SES mean. (b) Simulation data plotting IQ change across children's development where SES is captured by differences in cognitive stimulation. High SES = upper quartile, Middle SES = middle two quartiles, Low SES = lower quartile. (c) Equivalent mean performance on task (proportion correct) for simulated SES groups.

*Simulation 2: SES and developmental effects on population Rank order*

Thomas and Meaburn (in preparation) used the same model to simulate the analysis reported by Feinstein (2003). The empirical data from the 1970 Birth Cohort Survey are re-plotted in Figure 3. Around 1,300 UK children were classified into high (upper quartile) and low (lower quartile) cognitive ability at 22 months and then followed longitudinally to 10 years of age, with high SES (top 24%) and low SES (bottom 13%) subgroups tracked separately. Children are depicted by the mean population rank order of their group, where 100 is high performance and 1 is low performance. Somewhere between 5 and 10 years of age, initially high-ability/low-SES children fell below the rank of low-ability/high-SES children. Following publication of these data, the findings were criticised on two grounds. First, that they do not represent a real effect but instead regression to the mean of initially extreme scores through measurement error (Jerrim & Vignoles, 2013). Second, that the most emotive finding, of the cross-over of high-ability/low-SES and low-ability/high-SES groups between 5 and 10, was hard to replicate and depended on cut-offs used to define groups; for example, crossing-over was more likely under less extreme definitions of high and low cognitive ability (Washbrook & Lee, 2015; e.g., Figure 1).

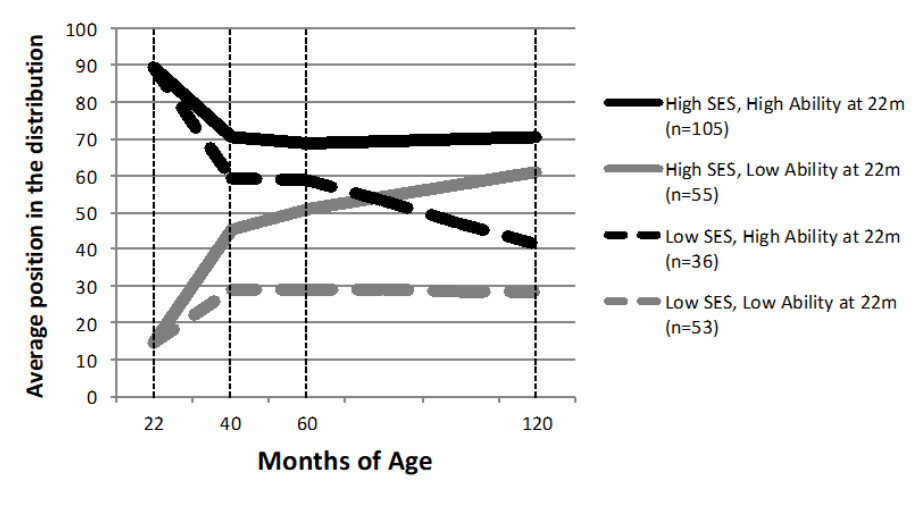


**Figure 3** – Longitudinal empirical data from the 1970 Birth Cohort Survey following the population rank of children on cognitive ability tasks, split by ability (high, low) at 22 months, and family socioeconomic status (re-plotted from Feinstein, 2003). Y-axis shows mean population rank of each group, where a higher rank marks better performance on age-appropriate cognitive tests.

Figure 4 depicts the computational simulation of these data (Thomas & Meaburn, in preparation). Early in training (25 epochs out of 1000 epochs), simulated children were split into high and low 'ability' groups based on behavior (accuracy of input-output mappings). High ability was defined as population rank >650 (where 1000 is good, 1 is poor), low ability as population rank <350. These groups were subdivided by SES, as a mean split (simulated SES varied 0 to 1; high SES>.5, low SES<.5). Performance of the groups was then followed over development. Figure 4(a) depicts the mean population rank of each group. As in the Feinstein (2003) data, high-ability/high-SES and low-ability/low-SES groups broadly held their mean rank. High-ability/low-SES showed declining rank and low-ability/high-SES show ascending rank, such that the groups converged. Notably, they did not crossover. Figure 4(b) shows the same data but for performance. It is included to emphasise that we are observing modulations in developmental trajectories, and that changes in

relative rank positions may exaggerate small differences in individuals who are nevertheless all showing developmental improvements with age.

Crucially here, there was no noise in the measurement of performance in the groups. The convergence of the trajectories, at least in the simulation, cannot have risen from regression to the mean following measurement error (Jerrim & Vignoles, 2013). It is a real reflection of the operation of constraints on development. Figure 4(c) takes the same population of children but now alters the definition of high and low ability to be less extreme (high ability: population rank >500; low ability: population rank <500) and the definition of SES more extreme (high: SES >.75; low: SES <.25). Now the trajectories of high-ability/low-SES and low-ability/high-SES did cross over. The simulations captured the empirical observation that the crossover pattern is sensitive to group definitions (Washbrook & Lee, 2015).

One simple interpretation of the Feinstein data is that changes in children's population rank performance in cognitive ability tests stem from environmental causes. For the simulation, we have available to us the full set of parameters that influences each simulated child's developmental trajectory: both the stipulated environmental effect, in terms of the level of cognitive stimulation, and the stipulated genetic individual differences, in terms of the neurocomputational patterns of each artificial neural network. We can then use these parameters in a multiple regression analysis to see which predicted population rank change across development.

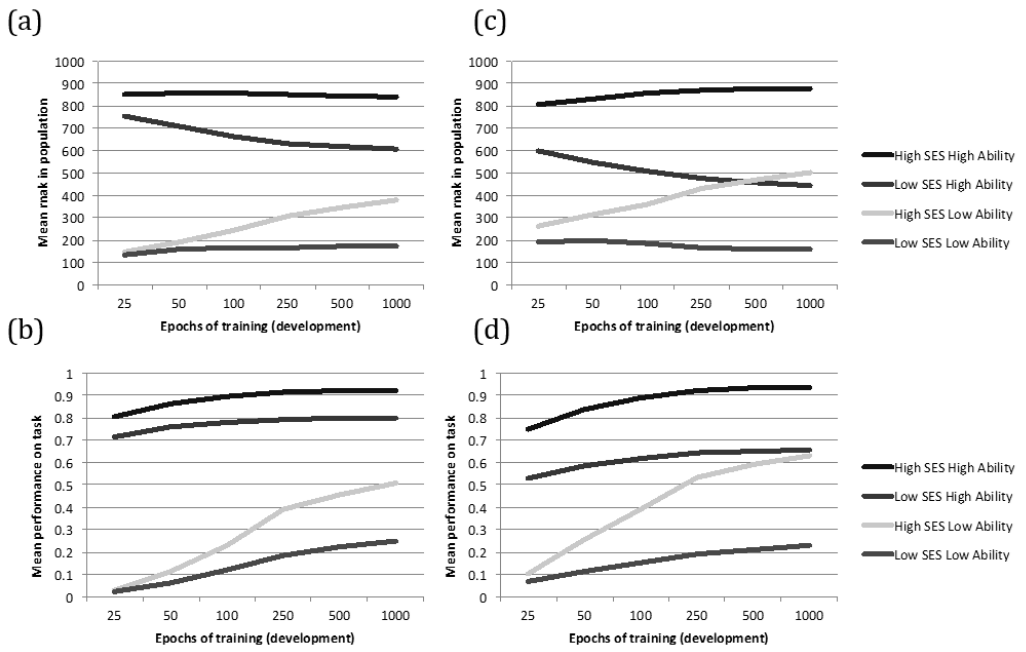


**Figure 4** – Simulations of longitudinal change in rank and change in performance across development in the computer model. Rank 1000 = best, rank 1 = worst. SES parameter varies between 1 (highest) and 0 (lowest). (a) Mean change in rank for high and low ability groups defined at time 1 (epoch 25), where high is rank >650 and low is rank <350, split by SES, where high >.5 and low <.5. (b) Equivalent performance on task (proportion correct). (c) Mean change in rank where high ability is time 1 rank >500 and low ability is rank <500, and where high SES >.75 and low SES <.25. (d) Equivalent performance on task for these group criteria.

Was all the rank change due to the environmental manipulation? Table 1 shows the results of this multiple regression, with the environmental parameter marked in bold, and the respective influence of each neurocomputational parameter below. First, it is worth noting that in the simulation, since environmental differences acted throughout development, they influenced measures of ability even at the early stage of development, here explaining 22.7% of the variance at the first time point. Early measurement does not give an unbiased measure of 'genetic' ability free from SES influences. Second, as expected, environmental differences did account for a significant amount of

variance in children's change in rank across development, up to 10% at the final time point. But notably, a number of neurocomputational parameters also contributed to change in rank. These included parameters influencing the capacity and plasticity of the mechanism, and consequently the shape of the developmental trajectory.

In other words, the model highlights that children develop at different rates. Some children are late bloomers, others slow later in development. This will cause changes in population rank order that are not solely related to variations in environmental stimulation. It is not necessary, therefore, to conclude from the Feinstein plot that the only cause of changes in children's population rank is due to environmental causes such as SES. In turn, this implies that not all the change in rank would be removed by reducing SES disparities.

**Table 1 –** Model of prediction of developmental change.

| Parameter | Neural network processing role | Predictors of developmental change in Population rank against Time 1 | | | | |
|---|---|---|---|---|---|---|
| | | Time 2 | Time 3 | Time 4 | Time 5 | Time 6 |
| Model fit ($R^2$) | | 0.181* | 0.312* | 0.368* | 0.379* | 0.384* |
| **SES** | **Environment** | **0.158*** | **0.274*** | **0.332*** | **0.337*** | **0.333*** |
| Hidden Units | Capacity | -0.069+ | -0.089* | -0.079* | -0.07* | -0.053+ |
| Architecture | Capacity | -0.185* | -0.212* | -0.171* | -0.142* | -0.129* |
| Sparseness | Capacity | 0.028 | 0.037 | 0.036 | 0.032 | 0.036 |
| Pruning Onset | Capacity | 0.044 | 0.074* | 0.077* | 0.074* | 0.067* |
| Pruning probability | Capacity | 0.021 | 0.017 | 0.004 | -0.002 | -0.006 |
| Pruning Threshold | Capacity | 0.033 | 0.013 | 0.006 | 0.023 | 0.025 |
| Learning algorithm | Capacity / plasticity | -0.064+ | -0.074* | -0.107* | -0.119* | -0.138* |
| Learning Rate | Plasticity | -0.148* | -0.159* | -0.177* | -0.186* | -0.199* |
| Momentum | Plasticity | -0.077* | -0.091* | -0.109* | -0.108* | -0.105* |
| Weight variance | Plasticity | 0.006 | 0.004 | 0.033 | 0.043 | 0.052+ |
| Unit activation function | Plasticity / signal | -0.107* | -0.147* | -0.178* | -0.184* | -0.188* |
| Noise | Signal | 0.019 | 0.036 | 0.069* | 0.101* | 0.116* |
| Response threshold | Signal | -0.223* | -0.292* | -0.304* | -0.308* | -0.309* |
| Weight Decay | Signal | -0.004 | -0.015 | -0.011 | -0.003 | -0.003 |

+ $p < 0.05$ * $p < .01$

**Note.** Level of environmental stimulation and neurocomputational parameters as predictors of *developmental change* in the model, measured by individual's change in population rank performance across development (scores show standardized beta coefficients from a linear regression model). Neurocomputational parameters are labelled according to their approximate processing role. Both environmental stimulation and network parameters explain variance in rank change (environment is marked by **bold**). The rightmost column indicates predictors of whether an individual's performance (rank) as an adult exceeds the rank of the

quality of their environment, as an indicator of *social mobility*. Time 1 = 25 epochs of training, Time 2 = 50, Time 3 = 100, Time 4 = 250, Time 5 = 500, Time 6 = 1000.

*Simulation 3: Genetic constraints on social mobility*

The model considered SES effects against the background of genetically influenced variations in learning ability. Thus, these simulations were able to capture the high heritability of behavior. For example, heritability of behavior shown in Figure 4(a) at the final measurement point was 51% under an additive model, computed from the twin design. The genetic component also allows the simulation to address data on social mobility. In the model, social mobility is defined as a developmental outcome that is greater or lesser than the SES of the family in which the child is raised (Thomas & Meaburn, in preparation). This can be measured as the difference in population rank order of a family's SES compared to the simulated child's population rank order ability at the end of training. For example, if the SES rank was 500 and the ability rank was 600, this would qualify as upwards social mobility; if the SES rank was 500 and the final ability rank was 400, this would qualify as downwards social mobility. Table 1, rightmost column, shows the results of a multiple linear regression predicting the rank disparity measure of social mobility from each simulated child's parameters. Notably, SES itself predicted a reliable amount of the disparity measure. Much of this relationship was driven by networks that fell below expected levels in high SES environments, less by networks that finished above expected levels in low SES environments. Several of the neurocomputational parameters relating to the network's capacity were reliable predictors of the disparity measure. These indexed whether the network had the capacity to best take advantage of the information that was available in the environment.

To the extent that the capacity of learning mechanisms is genetically influenced, this simulation therefore captured genetic influences on performance and on social mobility. It is the same simulation that captured empirical data on widening IQ gaps from SES across development. The same simulation that captured the restrictive effects of SES on children deemed high-ability early in development. These diverse behavioral effects were captured in a single mechanistic framework.

*Simulation 4: SES effects on brain structure*

Can the model also capture data on brain structure? The links between the model and brain structure can only be weak, because the model has a very limited degree of biological realism, necessitated by the requirement to make contact with high-level behavior. Moreover, there is still controversy how the physical properties that structural brain imaging measures relate to cognitive function. Despite the fact that cognitive ability shows broadly a monotonically increasing function with age, some of the brain structure measures reduce from middle childhood onwards (grey matter volume, cortical thickness), while others increase (white matter volume, cortical surface area); and the underlying biological mechanisms are still a matter of debate (Natu et al., 2018; Noble et al., 2015).

The model did not simulate the growth of each network, rather capturing variability in the outcome of the growth amongst its parameters in terms of network architecture (pathways linking input and output), number of processing units, and denseness of connectivity. It did, however, simulate a reduction in connectivity from mid-childhood onwards, in terms of a pruning process with variably timed onset that removed unused connections (see Thomas, Knowland & Karmiloff-Smith, 2011). For the artificial neural network, two structural measures offered possible analogs

to brain measures: the total *strength* of connections in the network and the total *number* of connections. During training, the total strength increases as those useful in driving behavior are strengthened, while the number of connections reduces as those not useful for driving behaviour are removed. These two network measures provide possible analogs to cortical surface area / white matter density and cortical thickness / grey matter density, respectively, by virtue of their similar developmental trajectories.

Figure 5 takes a mid-point in development for the simulated population considered in the previous sections. Figure 5(a) re-plots data from a sample of over 1000 US children aged 3-20 linking cortical surface area to family income (Noble et al., 2015). A small amount of variance is explained, with a non-linear function that exhibits stronger effects on brain structure at the lowest income levels. Figure 5(b) plots total connection strength for the simulated population against level of stimulation. Again, small amounts of variance are explained, and a non-linear function gives a best fit. Thus, the same simulated population that captures cross-sectional empirical data on SES effects on behavior can also capture cross-sectional patterns observed in brain structure data.

The model offers two benefits at this level. First, it provides a candidate hypothesis about the functional relevance of the brain structure measures – that they represent changes of connectivity arising from experience-dependent developmental change. Second, because the functioning of an artificial neural network is well understood – in terms of activations of networks of integrate-and-fire neurons, and learning algorithms that update connectivity and thresholds – it then demonstrates how indices of network structure only serve as an indirect measure of function, and how function modulates structure as a consequence of (variable) experience.

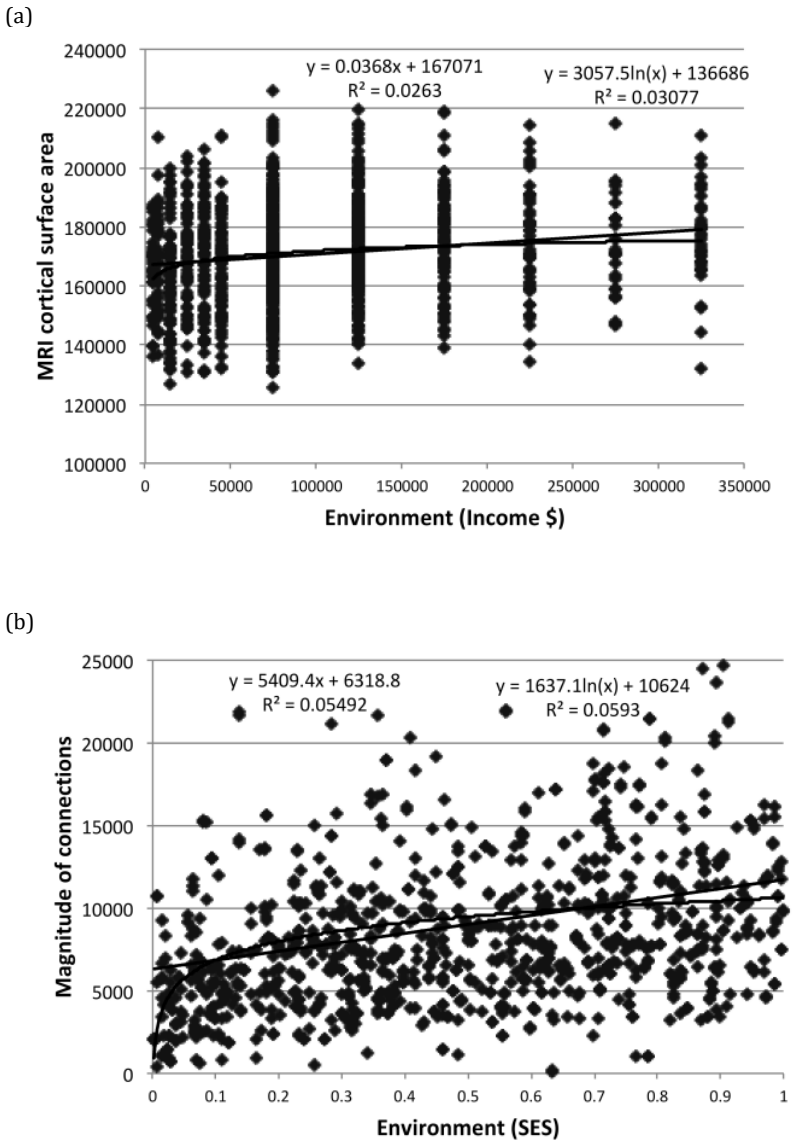


**Figure 5** – Empirical data re-plotted from Noble et al. (2015) showing the relationship between annual family income ($) and cortical surface area ($mm^2$) in a sample of 1099 US children between the age of 3 and 20. (b) Computer simulation data showing the relationship between level of cognitive stimulation in the environment in which children are raised, and the total magnitude of connection strengths in each artificial neural network, assessed at a mid-point in development (500 epochs of training). Both plots show a non-linear (log) relationship between the environmental measure and the structural measure, as well as much unexplained variability (linear and non-linear fits are shown, along with respective $R^2$ values).

## Discussion

A multi-level neurocomputational model was able to capture both behavioral data and brain structure data on the effects of differences in socioeconomic status on development. It did so while also incorporating the contribution of genetic variation to cognitive development, leading to high heritability of behavior; and by assuming that SES operates via differences in levels of cognitive stimulation. Variation between individuals was conceived as the modulation of trajectories of development, driven by species universal mechanisms.

In the simulation data presented, SES was implemented as variations in the level of cognitive stimulation. However, a modeling framework provides the opportunity to implement and compare alternative hypotheses, for example in how well they capture the effect size and shape (linear, log) of SES effects on particular measures of behavior and brain structure. Thomas et al. (in preparation) compared two alternative hypotheses: that SES may instead influence the growth of the networks themselves (per the findings of Betancourt et al., 2016), and therefore processing capacity; or that SES may influence both network growth and cognitive stimulation, in a correlated manner. The computational model therefore provides a foundation to hypothesis test different causal accounts of empirical data.

Thomas et al. (2019) have argued that once a basic developmental model of cognitive variation exists, it provides the basis to explore interventions, for example, by altering the quantity and quality of cognitive stimulation that individuals experience. The next step for the model, then, is to explore whether the gaps between individuals at different SES levels can be closed or eliminated by interventions that equalize environments, for instance by supplementing the stimulation received by children from low-SES families. Thomas and Meaburn (in preparation)

carried out these simulations, considering the extent to which opportunities to close gaps depended on the origin of individual differences (e.g., how heritable they were) and whether interventions were modulated by changes in plasticity with age (Thomas & Johnson, 2006). The broad pattern was that equalized and enriched environments improved population means under all conditions; when heritability was higher, improvements were smaller and gaps reduced less; but earlier interventions served to reduce gaps more than late interventions.

The research described here is presented to argue for the utility of neurocomputational modeling as one research tool to further the neuroscience of poverty. One should be cautious, however, to see such models in context. Models do not demonstrate what is actually the case: they demonstrate the sufficiency of particular mechanistic accounts to explain the observed empirical data; and therefore, indirectly, what any given pattern of empirical data must imply about causal mechanisms. By demonstrating the possible causal explanations of data, they do at least encourage the avoidance of misinterpretation of those data. For example, the pattern of widening IQ gaps across SES groups across development might be interpreted to mean that the action of SES differences worsens; the model showed the pattern would emerge even with static causal SES factors. The decline of population rank for early high ability children from low SES backgrounds could be interpreted to mean that population ranks are entirely dependent on environmental factors; the model showed that the empirical data are consistent with a limited role of environment in children's respective abilities. The influential role of SES on cognitive development and educational attainment might be taken as supporting a social causation account of SES differences, and of the primary role of environment in children's outcome. The model displayed realistic SES effects both on

behavior and network structure while displaying high heritability of individual differences, even indeed the heritability of differences in social mobility.

Clearly, the model presented here is highly simplified. While it shared some principles of neural processing, it is not a model of brain function. It is essentially a machine-learning mechanism that acquires a small set of input-output mappings, representing at best a single component of a larger system. A more realistic model of SES effects on development would need to depict a goal-oriented, adaptive, autonomous agent, with a repertoire of behaviors that can alter its subjective environment; to include separate cognitive, affective, and reward-based aspects; and provide a pathway for non-cognitive dimensions (diet, chronic stress, fitness) to alter its processing properties. And clearly, there is a great deal more to phenomena such as social mobility (and the societal structures that support or hinder it) than notions of cognitive stimulation and properties of developmental mechanisms.

Nevertheless, the key motivation for constructing a model of the current level of simplicity is to emphasise the importance of deriving causal, mechanistic accounts to explain the large body of correlational evidence that has accumulated on how SES is associated with differences in cognitive, educational, and life outcomes. Computational modeling is but one amongst several neuroscience methods that can shed light on mechanism, methods such as brain imaging, anatomy, animal models, and genetics. Mechanistic insights ultimately provide the basis to derive targeted interventions that can ameliorate the consequences of differences in SES, and especially poverty (Thomas, 2017). The potential of mechanistic insights to inform intervention is the motivating factor behind the involvement of neuroscience in a social issue such as poverty – even if the wider ambition is to alter societal structures that contribute to poverty in the first place.

***Acknowledgements.*** *This work was supported by MRC grant MR/R00322X/1 and a Wellcome Trust/Birkbeck ISSF Career Development Award held at the University of Western Ontario, Canada.*

## References

Ayorech, Z., Krapohl, E., Plomin, R., & von Stumm, S. (2017). Genetic influence on intergenerational educational attainment. *Psychological Science*, *28(9)*, 1302 – 1310.

Belsky, D.W., Domingue, B.W., Wedow, R., Arseneault, L. Boardman, J. D., Caspi, A., … & Harris, K. M. (2018). Genetic analysis of social-class mobility in five longitudinal studies. *Proceedings of the National Academy of Sciences*, Jul 2018, 201801238; DOI: 10.1073/pnas.1801238115

Betancourt, L.M., Avants, B., Farah, M.J., Brodsky, N.L., Wu, J., Ashtari, M., & Hurt, H. (2016). Effect of socioeconomic status (SES) disparity on neural development in female African-American infants at age 1 month. *Developmental Science*, *19(6)*, 947-956. doi: 10.1111/desc.12344.

Elman, J.L., Bates, E. A., Johnson, M.H., Karmiloff- Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist perspective on development*. Cambridge, MA: MIT Press.

Farah, M.J. (2017). The neuroscience of socioeconomic status: Correlates, causes, and consequences. *Neuron*, *96*, September 27, 2017, 56-71.

Farah, M.J., Shera, D.M., Savage, J.H., Betancourt, L., Giannetta, J. M., Brodsky, N.L., … & Hurt, H. (2006). Childhood poverty: Specific associations with neurocognitive development. *Brain Research*, *1110*, 166 –174.

Feinstein, L. (2003). Inequality in the early cognitive development of British children in the 1970 cohort. *Economica*, *70(277)*, 73–98.

Hackman D.A., Gallop, R., Evans, G.W. & Farah, M.J. (2015). Socioeconomic status and executive function:

Developmental trajectories and mediation. *Developmental Science*, *18(5)*, 686–702.

Hackman, D.A., & Farah, M.J. (2009). Socioeconomic status and the developing brain. *Trends in Cognitive Sciences*, *13(2)*, 65-73.

Hackman, D. A., Farah, M. J. & Meaney, M. J. (2010). Socioeconomic status and the brain. *Nature Reviews Neuroscience*, *11*, 651– 659.

HM Government (2003). *Every child matters*. Green Paper, Cm 5860

Jerrim, J. & Vignoles, A. (2013). Social mobility, regression to the mean and the cognitive development of high ability children from disadvantaged homes. *Journal of the Royal Statistical Society: Series A (Statistics in Society)*, *176(4)*, 887-906.

Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences*, *2*, 389–398. doi:10.1016/S1364-6613(98)01230-3

Krapohl, E., Rimfeld, K., Shakeshaft, N.G., Trzaskowski, M., McMillan, A., Pingault, J.B., Asbury, K., Harlaar, N., Kovas, Y., Dale, P.S., & Plomin, R. (2014). The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence. *Proc. Natl. Acad. Sci. USA 111*, 15273–15278.

Lee, J.J. et al. (2018). Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nature Genetics*, *50*, August 2018, 1112-1121. https://doi.org/10.1038/s41588-018-0147-3

Mareschal, D., & Thomas, M.S.C. (2007). Computational modelling in developmental psychology. *IEEE Transactions on Evolutionary Computation (Special Issue on Autonomous Mental Development)*, *11*, 137–150. doi:10.1109/TEVC.2006.890232

Natu, V.S., Gomez, J., Barnett, M., Jeska, B., Kirilina, E., Jaeger, C., Zhen, Z., Cox, S., Weiner, K.S., Weiskopf, N., & Grill-Spector, K. (2018). Apparent thinning of visual cortex during childhood is associated with myelination, not

pruning. *bioRxiv 368274*; doi: https://doi.org/10.1101/368274

Noble, K.G., Houston, S. M., Brito, N.H. et al. (2015). Family income, parental education and brain structure in children and adolescents. *Nature Neuroscience*, *18*, 773–778.

Pavlakis, A.E., Noble, K., Pavlakis, S.G., Ali, N., & Frank, Y. (2015). Brain imaging and electrophysiology biomarkers: Is there a role in poverty and education outcome research? *Pediatric Neurology*, *52(4)*, 383-388.

Plomin, R., DeFries, J.C., Knopik, V.S., & Neiderhiser, J.M. (2016). Top 10 replicated findings from behavioral genetics. *Perspectives on Psychological Science*, *11(1)*, 3 – 23.

Raizada, R.D.S., Richards, T.L., Meltzoff, A., & Kuhl, P.K. (2008). Socioeconomic status predicts hemispheric specialisation of the left inferior frontal gyrus in young children. *NeuroImage*, *40(3)*, 1392–1401.

Rimfeld, K., Kovas, Y., Dale, P.S., & Plomin, R. (2015). Pleiotropy across academic subjects at the end of compulsory education. *Scientific Reports*, *5*, 11713

Sheridan, M.A. & McLaughlin, K.A. (2016). Neurobiological models of the impact of adversity on education. *Current Opinion in Behavioral Sciences*, *10*, 108–113.

Stevens, C., Lauinger, B., & Neville, H. (2009). Differences in the neural mechanisms of selective attention in children from different socioeconomic backgrounds: An event-related brain potential study. *Developmental Science*, *12(4)*, 634–646.

Thomas, M.S.C. (2016). Do more intelligent brains retain heightened plasticity for longer in development? A computational investigation. *Developmental Cognitive Neuroscience*, *19*, 258-269.

Thomas, M.S.C. (2017). A scientific strategy for life chances. *The Psychologist*, *30*, 22-26.

Thomas, M.S.C. (2018). A neurocomputational model of developmental trajectories of gifted children under a

polygenic model: When are gifted children held back by poor environments? *Intelligence*, *69*, 200-212

Thomas, M.S.C. & Meaburn, E. (in preparation). Neurocomputational modelling of social mobility – Under what models of individual differences can interventions narrow the gaps?

Thomas, M.S.C. & Johnson, M.H. (2006). The computational modelling of sensitive periods. *Developmental Psychobiology*, *48(4)*, 337-344.

Thomas, M.S.C. & Knowland, V.C.P. (2014). Modelling mechanisms of persisting and resolving delay in language development. *Journal of Speech, Language, and Hearing Research*, *57(2)*, 467-483.

Thomas, M.S.C., Coecke, S., & Dick, F. (in preparation). A neurocomputational basis for socioeconomic effects on the development of behaviour and brain structure.

Thomas, M.S.C., Fedor, A., Davis, R., Yang, J., Alireza, H., Charman, T., Masterson J., & Best, W. (2019). Computational modelling of interventions for developmental disorders. *Psychological Review, 126(5)*, 693-726.

Thomas, M.S.C., Forrester, N.A., & Ronald, A. (2016). Multi-scale modeling of gene-behavior associations in an artificial neural network model of cognitive development. *Cognitive Science*, *40(1)*, 51-99.

Thomas, M.S.C., Knowland, V.C.P., & Karmiloff-Smith, A. (2011). Mechanisms of developmental regression in autism and the broader phenotype: A neural network modeling approach. *Psychological Review*, *118(4)*, 637-654.

Thomas, M.S.C., Ronald, A., & Forrester, N.A. (2013). Modeling socio-economic status effects on language development. *Developmental Psychology*, *49(12)*, 2325-43.

Trzaskowski, M., Harlaar, N., Arden, R., Krapohl, E., Rimfeld, K., McMillan, A., … & Plomin, R. (2014). Genetic influence on family socioeconomic status and children's intelligence. *Intelligence*, *42*, 83–88.

von Stumm, S. & Plomin, R. (2015). Socioeconomic status and the growth of intelligence from infancy through adolescence. *Intelligence*, *48*, 30–36.

Washbrook, E. & Lee, R. (2015). Beyond the Feinstein chart: Investigating differential achievement trajectories in a US cohort. *Longitudinal and Life Course Studies, 6*(3), 359-368.