Supplementary Material

To accompany the article 'Do more intelligent brains retain heightened plasticity for longer in development? A computational investigation' by M. S. C. Thomas

Sections:

1. Introduction

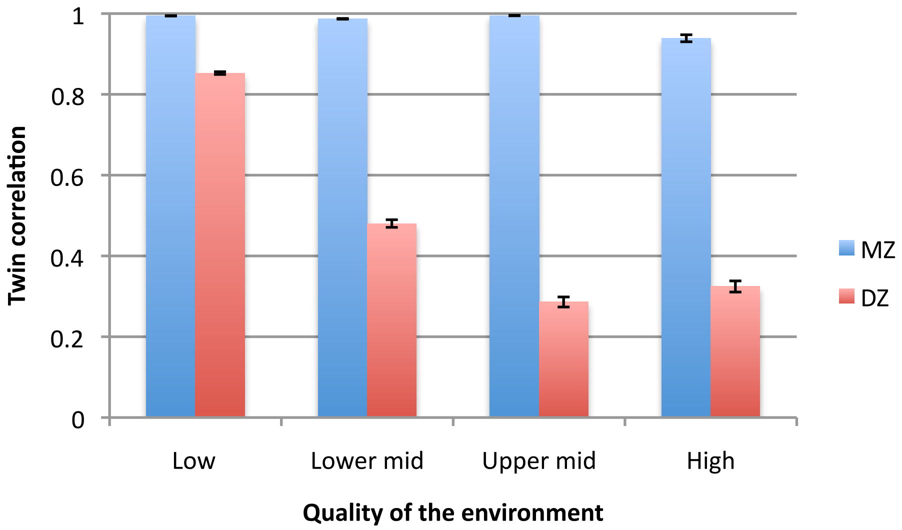
These supplementary materials provide supporting data for the article ‘Do more intelligent brains retain heighted plasticity for longer in development? A computational investigation’ by M. S. C. Thomas. Section 2 covers additional findings for the model, while Section 3 provides additional detail about the design of the model, the derivation of its parameters, the calibration of the range of variation of the parameters, and the encoding of the parameters in the artificial genome.

2. Additional results

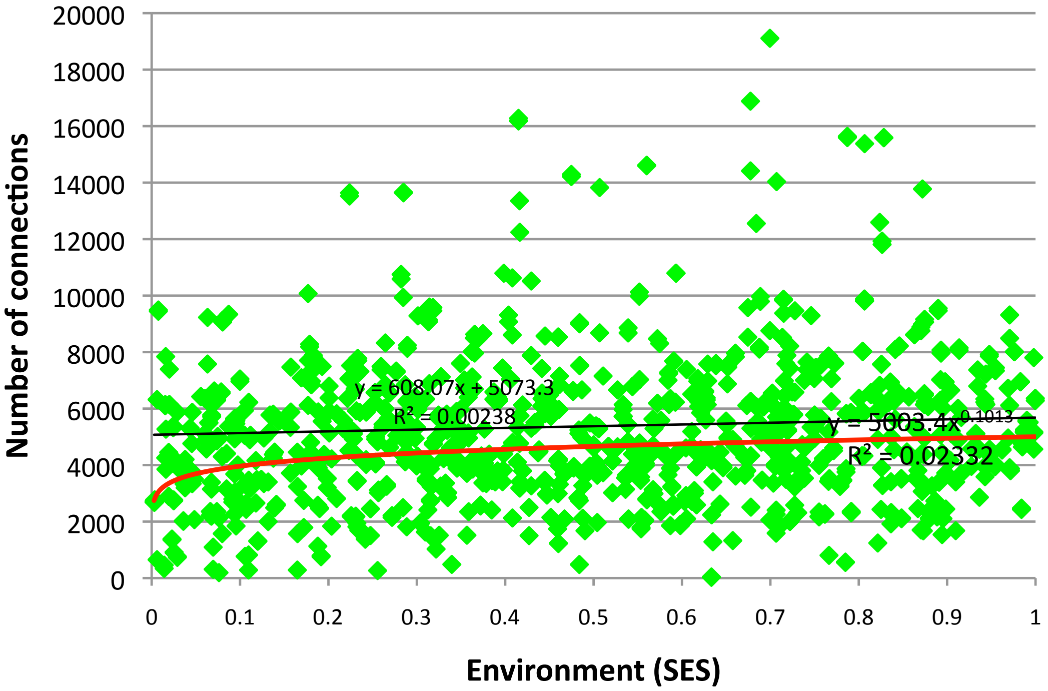
2.1 Simulated interaction of the heritability of cognitive ability and socio-economic status

Figure S1 (a): Interaction of the quality of the environment (implementing the effects of variations of socio-economic status) with the heritability of behavior, for the genetic-wide / environment-wide population; (b) relationship between quality of the environment and total number of connections in the network. Both measures are reported for regular verbs at 500 epochs of training (data from Thomas & Coecke, in prep.)

(a)



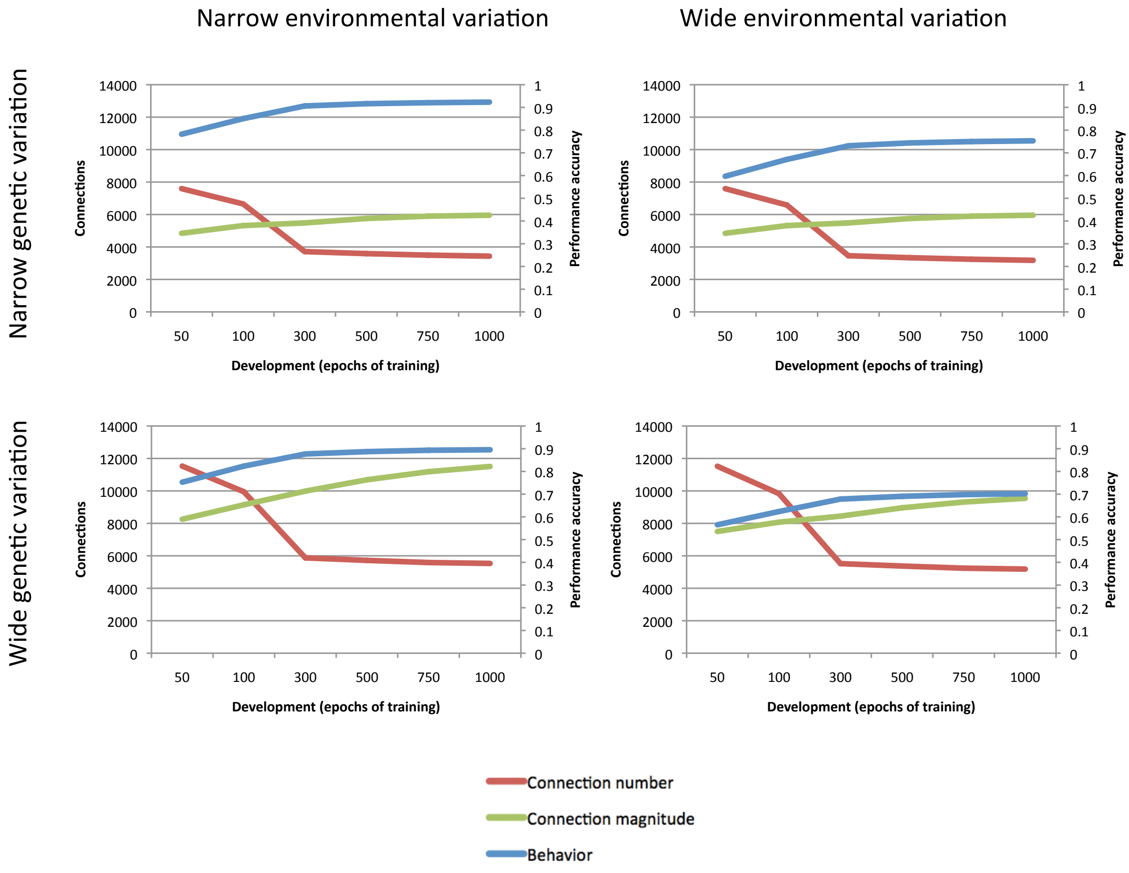
(b)



It has been reported that the heritability of intelligence is reduced in individuals from low SES background (Turkheimer et al., 2003; though see Hanscombe et al., 2012). In the genetic-wide / environmental narrow population, where all environments were sampled in the range 0.6 to 1 (where the possible range was 0 to 1), there was little influence of SES on heritability. In the genetic-wide / environment-wide population, environments could be much more impoverished. Figure S1(a) shows MZ versus DZ correlations split by quartiles of SES (number of twin pairs per group: lower = 55, lower-mid=66, upper-mid=67, higher=61). The reduction in heritability is clearest in the lower quartile, with an attendant increase in shared environment. Therefore, the modulation of heritability by SES was dependent on the absolute level of stimulation from the training environment. Noble et al. (2015) reported effects of SES on cortical surface area. The association with income was reliable but non-linear, such that the steepest gradient was present at the lower end of the income spectrum, and the effect sizes of the relationships were 1-2% of the variance. Figure S1(b) plots the total number of connections in each network against SES for the genetic-wide / environment-wide condition. A non-linear relationship is also demonstrated, accounting for around 2% of the variance in structure.

2.2 Population mean developmental trajectories for behavior, total network connections, and total connection magnitude, for the four populations

Figure S2. Population means for behavior (regular verbs), and the two network structure indices of total connection number and total connection magnitude, for the four populations in the 2x2 design across the full lifespan of development (1000 epochs of training).



2.3 Average connection strength in high ability and low ability networks across development

Low ability networks tended to have stronger connection weights. This disparity was not present at the start of training but emerged, as networks with fewer representational resources attempted to learn equivalent input-output mapping sets.

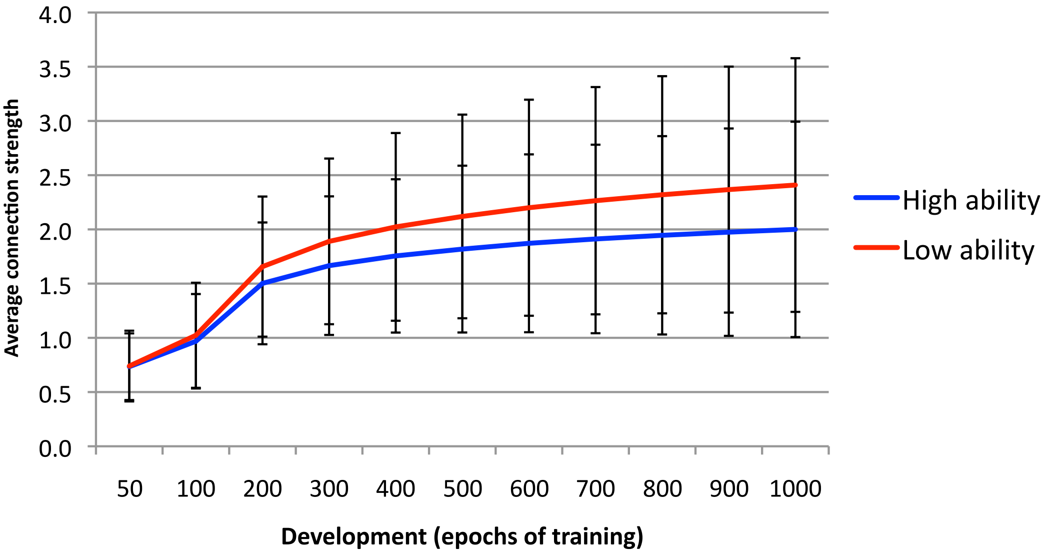
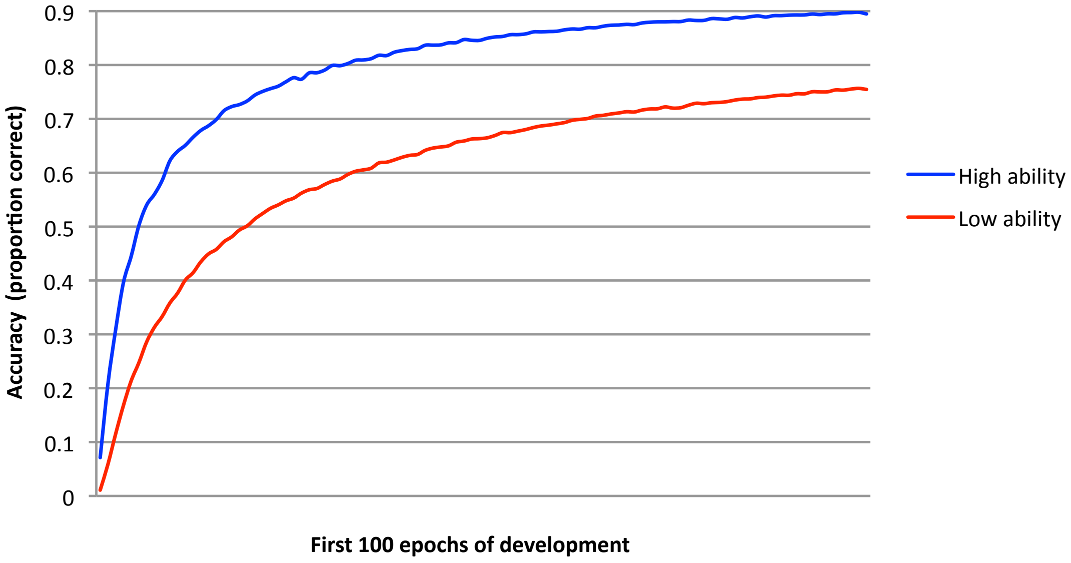


Figure S3: Average connection strength (total connection magnitude divided by total connection number) for high ability networks and low ability networks across development for the genetic-wide / environment-narrow population. Error bars show standard deviations. (Main effect of Ability, F(1,998)=29.91, p<.001, η_p^2=.029; main effect of Development (quadratic) F(1,998)=2545.43, p<.001, η_p^2=.718; interaction of Ability x Development, F(1,998)=28.83, p<.001, η_p^2=.028).

2.4 Mean performance trajectories of high ability and low ability groups early in development

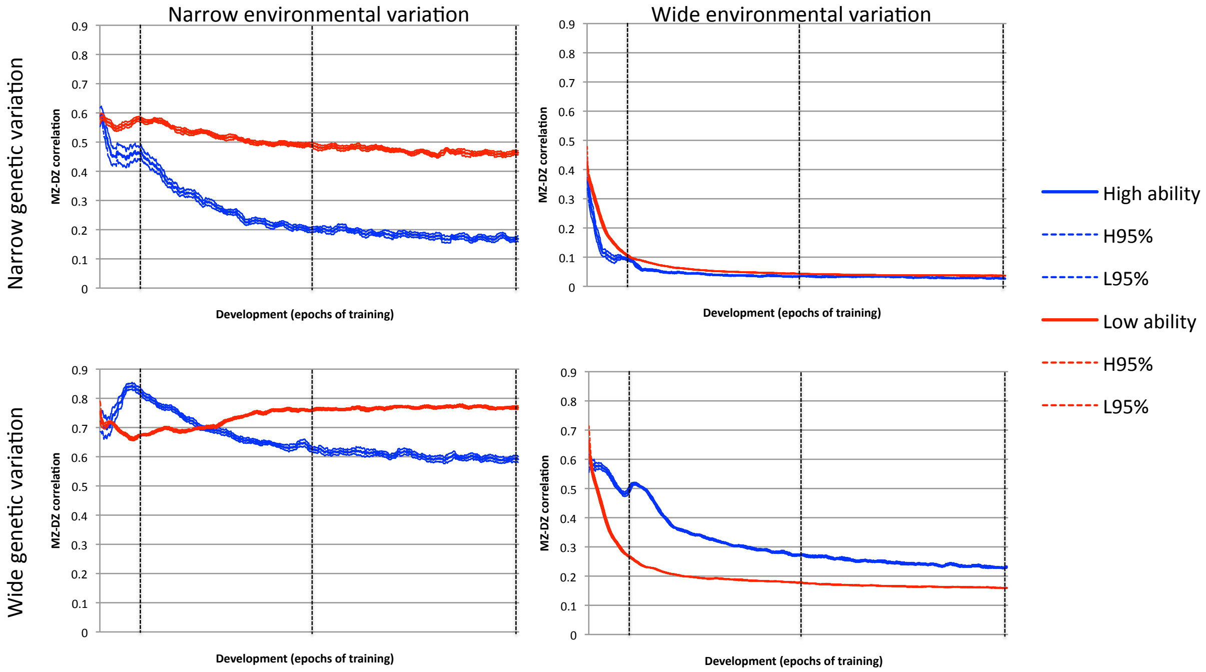
Figure S4: Accuracy differences between high ability and low ability networks in the first 100 epochs of training, for the genetic-wide / environment-narrow population.



These data demonstrate that accuracy differences between high and low ability networks emerge very early in development. They are already present during the first 100 epochs of training prior to the time 1 measure of heritability.

2.5 MZ-DZ correlation differences for the four populations using a more environmentally sensitive measure of ability

Figure S5: The difference between MZ and DZ correlations across development, using untransformed performance on irregular verbs at epoch 50. This measure is normally distributed and discriminating between individuals, but is sensitive to variations in environmental quality. Data are shown for the 2x2 design.

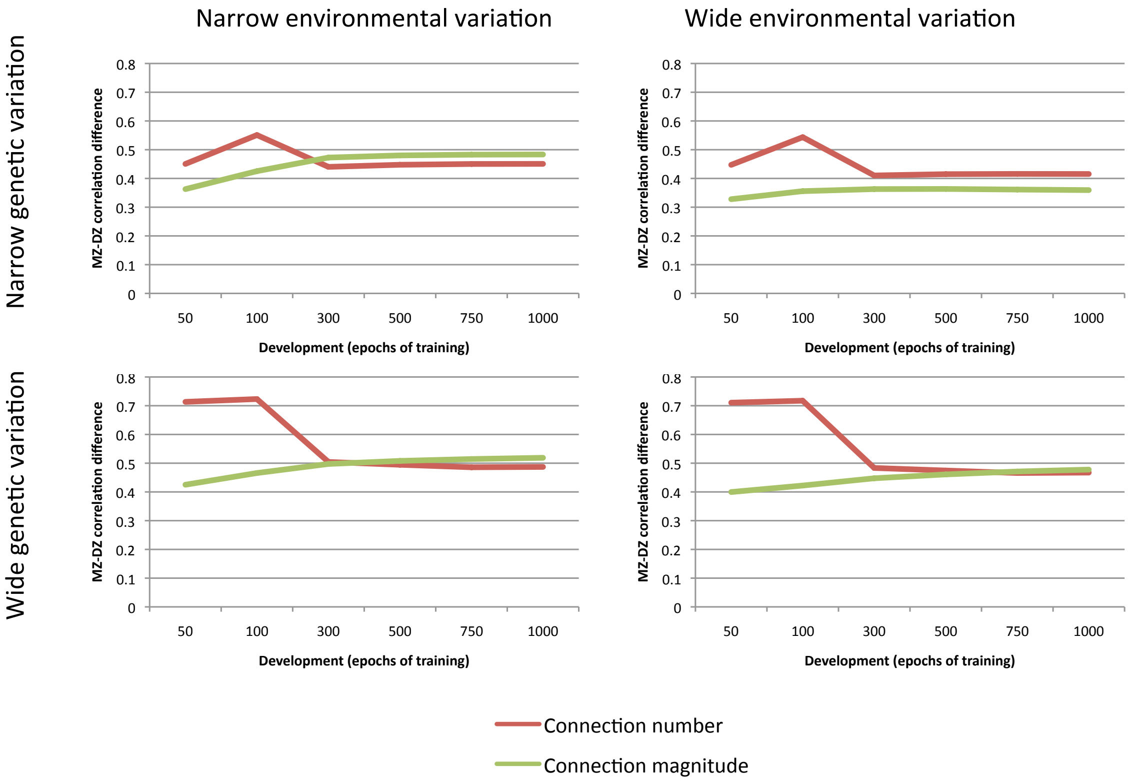


The main impact of using untransformed irregular verb performance is to reduce the heritability of high ability groups in the conditions with narrow environmental variation. This is because formerly, ability was determined by the 'culture-fair' measure removing environmental influence, and therefore mainly determined by internal neurocomputational properties. With the untransformed measure, high ability is more dependent on how good the environment is, hence reducing heritability. The correlation of environmental quality for the 'culture-fair' ability measure was:

GWEN=-.004; GWEW=-.032; GNEN=.023; GNEW=-.032. For the more environmentally sensitive measure, it was GWEN=.113 ; GWEW=.450; GNEN=.178; GNEW=.455.

2.6 MZ-DZ correlation differences for the four populations across development for total network connections, and total connection magnitude

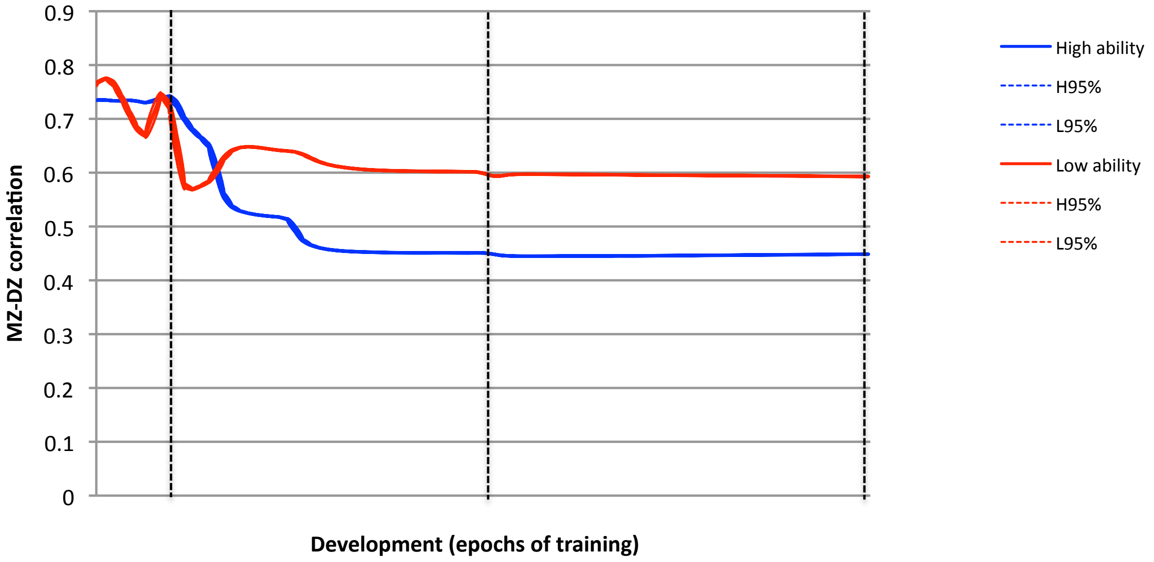
Figure S6: MZ-DZ twin correlation difference, proportional to heritability, for the two indices of network structure, total connection number and total connection magnitude, across development for the 2x2 design.



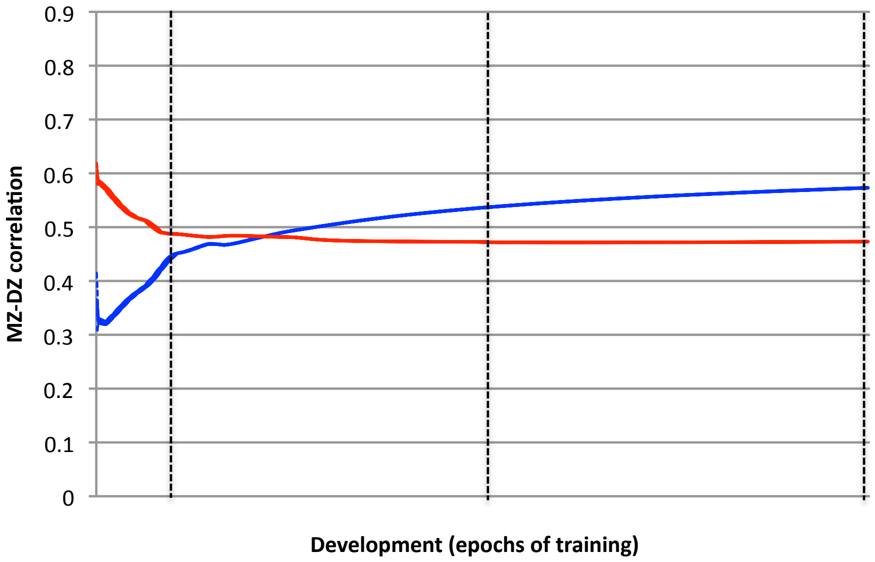
Total connection number shows an initial rise in heritability, followed by a fall during pruning (where the richness of the environment influences how many connections are lost), followed by a plateau. Magnitude shows increasing heritability across development.

Figure S7: MZ-DZ twin correlation difference across development for the genetic-wide / environment-narrow condition, for the two structural indices, split into high and low ability groups. (a) Total connections. (b) Total magnitude. Ability group is defined using the 'culture-fair' measure.

(a) Total connections



(b) Connection magnitude



Connection magnitude (strength gain) indicated higher heritability for the high ability group, while connection number (connection loss) indicated higher heritability for the low ability group.

2.7 Correlation between behavior, structural measures, environmental quality, and timing of plasticity changes for the four simulated populations

Table S1: Pearson correlation between behavior, structural measures, environmental quality, and timing of plasticity changes for the four simulated populations experiencing different relative range of variation of genetic and environmental factors. Correlations were computed at the mid point in development (500 epochs of training). Perf = performance accuracy on regular verbs; CoN = total number of network connections; CoM = total magnitude of network connections; Env = quality of the training environment; Prn = timing onset of connection pruning / reduction in plasticity.

			Environmental variation									
			Narrow					Wide				
			CoN	CoM	Env	Prn			CoN	CoM	Env	Prn
Genetic variation	Narrow	Perf	.348**	.116**	.562**	.070*		Perf	.241**	.457**	.850**	.054
		CoN		.584**	.059	.069*		CoN		.691**	.200**	.061
		CoM			-.031	.056		CoM			.496**	.056
		Env				.005		Env				.059
	Wide	Perf	.157**	.136**	.143**	.024		Perf	.105**	.257**	.774*	.014
		CoN		.613**	-.022	.296**		CoN		.653*	.049	.303**
		CoM			.049	.063*		CoM			.234**	.084**
		Env				.035		Env				.006

* p<.05 2-tailed

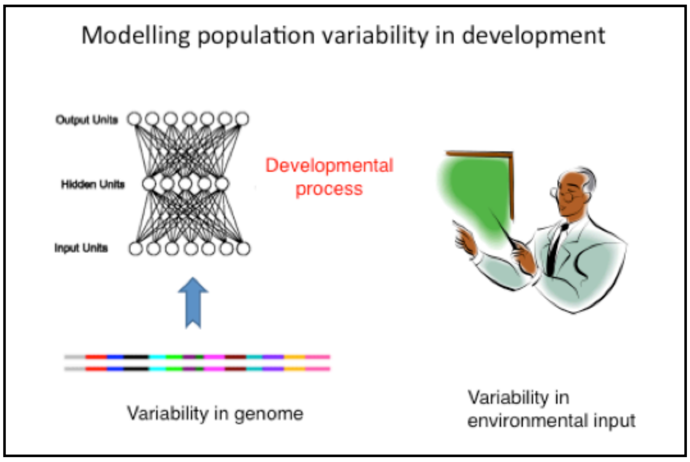
** p<.01 2-tailed

Pruning onset didn’t explain much of the variance in performance. Correlations between structural indices were stronger than between structural indices and behavior. Environment quality explained more of the variance in behavior than in the structural indices. Effect sizes for the implemented SES effect on behavior ranged from 2.04% of the variance in the genetic-wide / environment-narrow condition to 72.25% of the variance in the genetic-narrow / environment-wide condition. Effect sizes for the implemented SES effect on structure ranged from .05% of the variance in the genetic-wide / environment-narrow condition to 24.60% of the variance in the genetic-narrow / environment-wide condition.

3. The base model

These simulations explored a population of networks acquiring the past tense domain, where individual variability was included both in the parameters of the artificial neural networks which modeled the children's learning systems, and the learning environment to which they were exposed. The parameters of the artificial neural networks were encoded in an artificial genome. Population variability in parameters was created by generating and breeding populations of artificial genomes. Each genome was realized as a parameterized network. The network was exposed to an individualized learning environment, generating a trajectory of behavioral development. The inclusion of an artificial genome level in the simulations allowed two avenues of exploration. First, it permitted us to study the associations that can arise between values on the artificial genome and behavioral variability that is the product of an implemented developmental process (see Figure 2). These results are considered in Thomas, Forrester & Ronald (2015). Second, it allowed us to generate individuals with different levels of genetic similarity, such as parents, siblings, monozygotic (identical) twins, and dizygotic (fraternal twins); siblings could be exposed to a shared family learning environment, unique learning events, or a combination of the two. Measurements of the similarity of behavior between related individuals then permitted the simulation of behavior genetic designs, such as twin studies. The simulations therefore provided a framework to study heritability of behavior within a developmental framework.

Figure S8: Schematic of the population modeling simulations

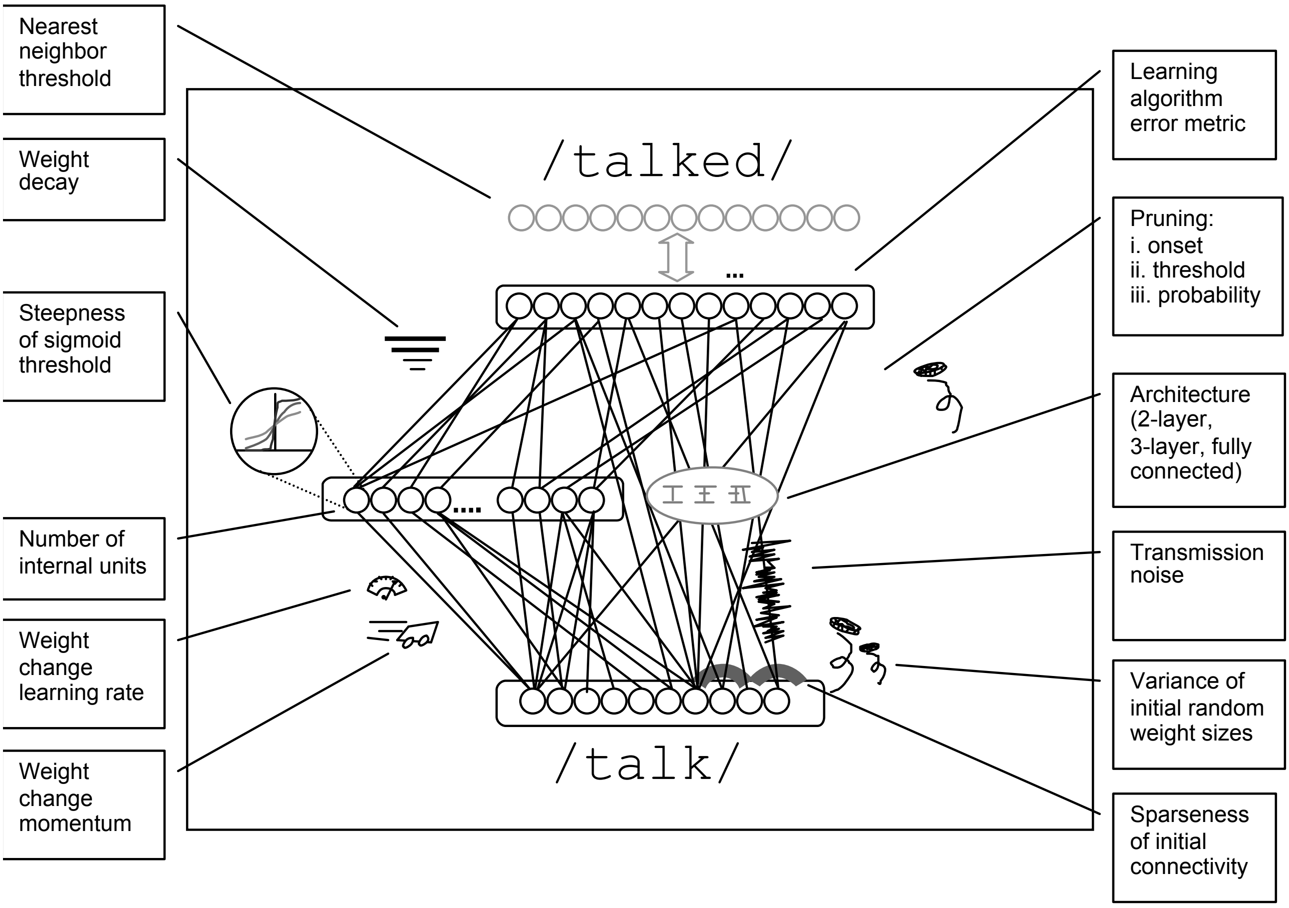

Modelling population variability in development
Output Units
Hidden Units
Input Units
Developmental process
Variability in genome
Variability in environmental input

This document outlines the computational parameters that varied in the artificial neural networks. It details how the range of variation for each parameter in the population was established. It then describes the method for designing the artificial genome, and the assumptions that this method embodies. The details of the breeding process are described. Finally, a set of lookup tables is included detailing how values on the artificial genome were mapped to computational parameter values in the artificial neural networks. Two sets of tables are included, one that specifies a wide range of computational parameter variation in the population, a second that specifies a relatively narrow range of variation. By combining these with training sets that also can vary widely or narrowly in quality, the relative contributions of internal versus external influences on individual differences in behavior can be gauged.

3.1 Model architecture and parameters

The original connectionist model employed a three-layer artificial neural network, comprising an input layer, a layer of internal or 'hidden' units, and an output layer. It was trained using the backpropagation algorithm (Rumelhart, Hinton, & Williams, 1986), a type of supervised learning. The free parameters in the model were the number of hidden units, the learning rate, and the momentum (see below). An expanded set of 14 parameters was employed in the current simulations, in many cases to allow for additional analogues to known neurocomputational properties. However, backpropagation itself is not viewed as fully biologically plausible. We use it here in place of a more biologically plausible error-correction algorithm (see Thomas & McClelland, 2008, for discussion). An introduction to the idea that parameters in connectionist models can explain types of cognitive variability can be found in Thomas and Karmiloff-Smith, 2003a). The parameters and model architecture are depicted schematically in Figure S9.

Figure S9: Architecture of the connectionist model of English past-tense acquisition, showing the internal parameters that varied in the population.


Nearest neighbor threshold
Weight decay
Steepness of sigmoid threshold
Number of internal units
Weight change learning rate
Weight change momentum
/talked/
...
....
/talk/
Learning algorithm error metric
Pruning:
i. onset
ii. threshold
iii. probability
Architecture (2-layer, 3-layer, fully connected)
Transmission noise
Variance of initial random weight sizes
Sparseness of initial connectivity

The parameters were as follows:

Building the network:

- *Architecture*: In addition to the 3-layer network, a 2-layer network without a layer of hidden units, and a fully connected network were used. A 2-layer network has less computational power than a 3-layer network but learns more quickly. A fully connected network contains both direct connections from input to output and a hidden layer, and produces a computationally more powerful system. Networks could therefore have 1, 2, or 3 layers of connection weights. Previous connectionist models have proposed single or multiple pathways may be available to connect input and output (e.g., Westermann, 1998; Zorzi, Houghton & Butterworth, 1998), and that differential use of routes may explain individual differences in behavior (Harm & Seidenberg, 2004; Plaut, 1997; Thomas & Karmiloff-Smith, 2002). Recent functional brain imaging of reading lend support to this proposal (e.g., Richardson et al., 2011; Seghier et al., 2008).
- *Hidden units*: For networks with a hidden unit layer, the number of hidden units could vary. Variations of the number of hidden units have been proposed to account for developmental deficits such as dyslexia (e.g., Harm & Seidenberg, 1999) and autism (e.g., Cohen, 1998), as well as individual differences (Richardson et al., 2006a, b). We did not vary the number of hidden layers. More hidden units within a layer increases computational power and the rate of learning, while more layers of hidden units increases computational power but slows down learning, since error must be propagated from the output more deeply into the network to improve learning (see Richardson et al., 2006a,b, for a comparison of these conditions).

- *Sparseness*: The architecture determined how many layers of connection weights existed. Of the potential connections in a layer, only a certain proportion was created. The sparseness parameter set the probability that any given connection would be created. Greater connectivity increases computational power, but can lead to slower learning. Under some conditions, it can also lead to poorer generalization, since greater integration of information causes more item-specific and context-specific learning (see McClelland, 2000, for a proposal that conjunctive coding may cause autistic symptoms; and conversely, Beversdorf, Narayanan & Hughes, 2007, for a proposal that the symptoms arise from sparse connectivity).
- *Weight variance*: Connection weights were assigned an initial random value within a range depending on this parameter. E.g., if set to 0.5, weights would be randomized between +/- 0.5. Large initial weights take time to unlearn, which slows learning (an effect known as entrenchment; see Munakata & McClelland, 2003, for discussion).

Processing dynamics:

- *Processing noise*: The net activation a receiving unit receives from a given sending unit is a product of the sending unit's activation and the connection strength between them. Transmission noise was added to this net activation. Gaussian noise was used and the parameter specified the standard deviation of the noise distribution around zero. Noise has been used to simulate under-specified representations in development (e.g., to simulate Specific Language Impairment: Joanisse & Seidenberg, 2003; or as a candidate explanation of autistic symptoms:

Simmons et al., 2007), and has also been proposed as an essential primitive in neural processing (McClelland, 1993).

- *Unit threshold function*: A receiving unit sums the net activation from all sending units and uses an activation function to determine its consequent output. We used a common non-linear activation function, the sigmoid or logistic function, equivalent to a smoothed threshold. This function has a free parameter, the ‘temperature’, which makes the smoothed threshold either steeper or shallower. The activation function was:

$$Output = \frac{1}{1 + e^{-temperature \times (netinput + bias)}}$$

where *netinput* is the summed activation to a unit, *bias* is the negative of the unit’s threshold, and *Output* is the unit’s activation state in response to this input. A shallow function (low temperature) denies a unit the opportunity to make large output changes in response to small changes in net input, whereas a steep function (high temperature) approximates a non-smoothed threshold, thereby producing a unit with binary response characteristics. Variations in the slope of the sigmoid function have been proposed as candidate explanations of disorders such as specific language impairment (Thomas, 2005) and schizophrenia (Cohen & Servan-Schreiber, 1992), as well as ageing (Li & Lindenberger, 1999). Changes to the slope of the sigmoid have a number of effects on learning. A shallow slope means that processing units are less sensitive to small differences in their input. This poor discriminability means they will be slow to learn categorizations that rely on small distinctions in the input. Secondly, in the backpropagation algorithm, weight update for a given error signal is proportional to the slope on the sigmoid (the differential of the function). If the function resembles a gentle S-shape, then the slope across the range of unit activations will be small. A shallow sigmoid will lead directly to slower learning. Conversely, if the temperature is very high, producing a sigmoid similar to a step function, for most inputs to a unit, it will be jammed on or off ('saturated') rather than in its dynamic range. When a unit is saturated, the slope on the sigmoid function is flatter (the regions below or above the step). When it is in its dynamic range it is steep (the step). If a unit is predominantly saturated due to a high temperature, the flat slope will again lead to small weight changes for a given error signal and therefore slow learning. Finally, units with high temperatures flip between being saturated on or off. They are therefore ill suited to learning mappings requiring graduations of activation states. In sum, temperatures that are either too high or too low can delay learning.

Network maintenance:

- *Connection weight decay*: each connection's magnitude was reduced by a small proportion on each presentation of a training pattern, according to the weight decay parameter. The approximate range of weight decay values was derived by estimating a percentage of weight value that could plausibly be lost overall all of training (e.g., 50%), and then dividing this proportion by the number of training epochs (e.g., 1000) and the number of training patterns presented on each epoch (e.g., 508), to give a proportional reduction in the connection weights to be applied on each pattern presentation (e.g., $0.5/1000/508=9.84 \times 10^{-7}$). To our knowledge, weight decay has not been used as a candidate mechanism to explain individual variability.
- We did not simulate the increase in synaptic density observed in human cortex during infancy and early childhood, instead simulating the outcome of this process through variations in the sparseness of connectivity; we did, however,

implemented the pruning of spare resources from mid-childhood (Huttenlocher, 2002). The *pruning process* eliminated small connection weights. Variations in pruning have been proposed as an explanation of autistic symptoms, and specifically developmental regression (Thomas, Knowland & Karmiloff-Smith, 2011). The pruning process involved three parameters: onset, threshold, and probability:

- *Connection pruning – onset*: Connections that were not being used were probabilistically pruned away after a certain point in training. The onset parameter determined the point in training when pruning began (see Thomas & Johnson, 2006, for simulations of pruning applied to sensitive periods in plasticity).
- *Connection pruning – threshold*: Connections stood a chance of being pruned after onset only if their magnitude fell below a threshold determined by this parameter. The rationale is that small weights are assumed not to transmit strong activations and therefore not to be playing a key role in computations. They may therefore be removed to save on resources.
- *Connection pruning – probability*: If the magnitude of a connection fell below threshold after pruning had begun, it was eliminated probabilistically based on this parameter. High probability leads to faster loss of unused connections. Low probability leads to slower loss.

Network adaptation:

- *Learning algorithm error measure*: The backpropagation algorithm was used with two different metrics to determine the error signal marking the disparity between the network's current output and its intended target. These were Euclidean distance and cross-entropy (Hinton, 1989). The Euclidean distance metric produces less weight change for a unit when it is committed to an erroneous response than the cross-entropy measure. That is, when a unit is stuck on in a saturated state but the learning algorithm requires it to be off, or vice versa, cross-entropy will lead to faster changes to its weights to change its activation state than Euclidean distance. Under some conditions, cross-entropy can therefore be a more plastic learning algorithm, leading to faster learning and higher ceiling performance.

- *Learning rate*: This parameter determined how much the connection weights were altered in response to a certain disparity between output and target during supervised learning. A large learning rate produces a system that learns more quickly but that also may be unstable, flipping between good performance on different parts of the problem domain. Differences in learning rate have been proposed as explanations of individual differences in cognitive ability (Richardson et al., 2006a,b) and general intelligence (Garlick, 2002), as well as developmental deficits (e.g., dyslexia; Harm & Seidenberg, 1999).
- *Momentum*: This parameter allowed some proportion of the weight change on the previous learning trial to be carried over. It serves a smoothing function to prevent learning from getting stuck in local, sub-optimal solutions. While a parameter often varied in connectionist models of development, it has not to our knowledge been used as a candidate explanation for individual differences in learning.

Network response:

- *Nearest neighbor threshold*: Network output comprised a vector of continuous activation values between 0 and 1, while legal responses of the network were binary vectors. An algorithm determined which legal phoneme was closest to the activation patterns at onset, nucleus, and coda. However, the phoneme was only recognized as a response if the activation was sufficiently close to the legal phoneme (using a root mean square or RMS measure). This was determined by the nearest neighbor threshold. (The legal phonemes could of course still be the incorrect ones for the target verb). The nearest neighbor computation may be viewed as equivalent to the settling of an unimplemented recurrent attractor network into a particular response state (see Plaut et al., 1996, for a model of reading development in which this attractor network was implemented). The nearest neighbor threshold parameter then indexes the efficiency of this attractor network to generate a response within some notional deadline. A high threshold allows an approximate output to be recognized as correct (i.e., larger error is tolerated); a low threshold requires a more exact initial output. The use of a nearest neighbor algorithm allowed the network to generate accuracy levels. Differences in the functioning of the attractor network (sometimes called 'clean-up' units) have been proposed as a candidate explanation of developmental deficits (e.g., dyslexia; Harm & & Seidenberg, 1999).

3.2 Calibrating parametric variation

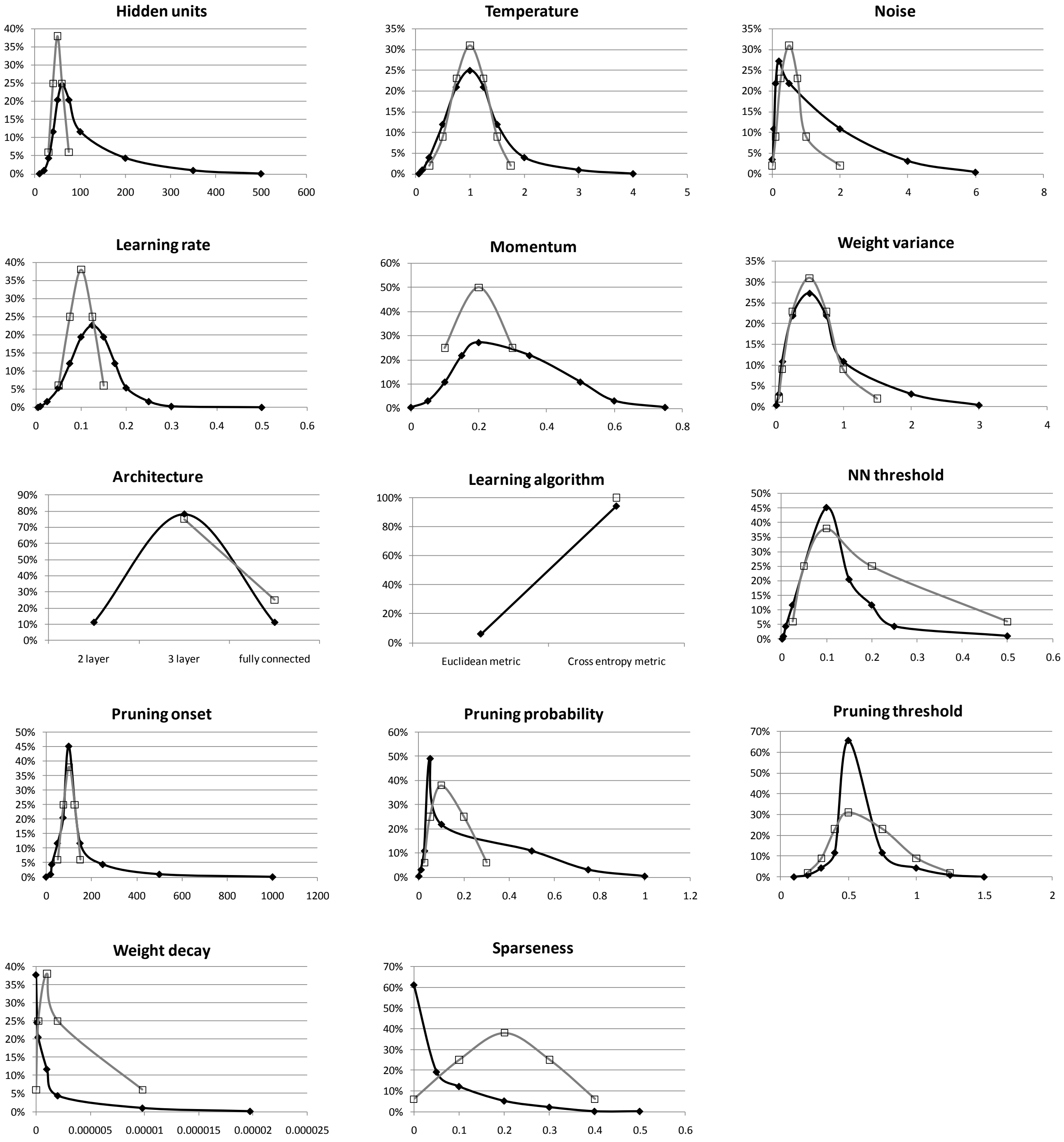
Calibration was carried out to establish the full range of variation for each parameter over which the artificial neural network exhibited some degree of learning. In general, the network was fairly robust to variation in its parameters, as illustrated in Appendix A.

Two of the network parameters were categorical: the architecture and learning algorithm metric. The others were continuously valued. In order to produce variability in the population according to these remaining parameters, they were calibrated as follows. An initial 'normal' set of parameters was defined. These were estimated based on previous research. Each of the continuously valued parameters was then varied in turn, holding the all other parameters at their initial values. For each parameter, the range was derived that produced failure of learning up to highly successful learning. In some cases, parameters had a monotonic relationship to performance (e.g., hidden units, where more was better); in other cases, there was an optimal intermediate value (e.g., activation function). The functions linking a given parameter and behavioral outcomes, with all other parameters held constant, are included in Appendix A. The aim was to determine an average or adequate value for each parameter, which was defined heuristically as 'just enough to succeed and then a little bit more'. Values were then derived that would cause increasingly poorer or increasingly better performance around this value. We attempted to make poorer and better performance roughly symmetrical around average performance for each parameter. This caused some parameter ranges to be skewed. For example, 50 hidden units was determined as the average value in a 3-layer network. Values of 40 or 30 would cause poorer performance. However, to achieve equivalent differences above average level, 100 or 200 hidden units might be necessary. We chose to emphasize behavioral symmetry around the average parameter value rather than parametric symmetry, on the grounds that the symmetrical bell curve is a common pattern observed in human abilities. The ranges for each parameter for the phonology-to-phonology network are included in Figure S10.

We chose not to vary the input and output coding scheme. Our previous work suggests that, within certain limits, varying the problem encoding has similar effects on the developmental trajectory to altering computational parameters (Thomas & Karmiloff-Smith, 2003b). However, recoding the problem domain can in principle have extreme effects on learnability, if key distinctions in the input or output are lost in the recoding. Some models of developmental language impairment and dyslexia propose that differences in the representation of phonology cause subsequent behavioral deficits in grammar and reading acquisition (e.g., Harm & Seidenberg, 1999; Hoeffner & McClelland, 1993; Joanisse, 2004).

Although only main effects of each parameter were considered as sources of variability during calibration, we expected interactions between these neurocomputational parameters in subsequent learning. To pick four examples: (i) large numbers of hidden units can partially compensate for a shallow sigmoid function in those processing units; (ii) having a more sparse initial connectivity is likely to reduce the amount of weights eliminated via pruning because their magnitudes will be larger; (iii) high weight decay can be countered by a higher learning rate; (iv) an over-aggressive pruning process (e.g., with a high threshold and high probability) can be alleviated if its onset occurs very late in training when weights have become large, but exacerbated if the onset is early. Large numbers of parameter combinations were possible within our scheme: given the number of levels specified for each parameter, approximately two trillion unique parameter combinations were available.

Figure S10: Parameter values and target population frequencies for genetic-wide (black) and genetic-narrow (grey lines) conditions.



In Figure S10, dark lines show parameter values (x-axis) and their target frequencies in the population (y-axis) for each of the 14 computational parameters. Each gene had two alleles, coded as binary values. Several genes coded for each parameter value. Sets of binary values were summed and a look-up table used to derive each parameter value. The numbers of binary alleles for each parameter were as follows. *G-Wide* = 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; sparseness: 12 (total 126 bits). *G-Narrow* = hidden units: 4; temperature: 6; noise: 6; learning rate: 4; momentum: 2; weight variance: 6; architecture: 2; learning algorithm: 4; nearest neighbor threshold: 4; pruning onset epoch: 4; pruning probability: 4; pruning threshold: 6; weight decay: 4; sparseness: 4 (total 60 bits).

3.3 Specifying an artificial genome for the model

The use of genetic algorithms entails creation of an artificial genome to encode the neural network's parameter values, such that all possible genomes correspond to legal parameter sets. In creating the genome, we made the following assumptions:

- There were two copies of each gene, with genes residing on pairs of chromosomes.
- For simplicity, each gene had only two variants or alleles.
- The two alleles produced different outcomes in the functionality of the neurocomputational parameter which they encoded.
- The influence of genes was intended to be *additive*: we did not include dominant or recessive effects, and genes had the same effect in combination as in isolation. This constraint was motivated by the finding within behavioral genetics that the effect of gene variants is predominantly additive on phenotypic outcomes (Plomin et al., 2008). Nevertheless, our method of

implementing the mapping between gene variants and neurocomputational parameters did inadvertently produce some non-additive effects.

- All neurocomputational parameters were *polygenic*. That is, their value was determined by the additive action of a collection of genes.
- In the first instance, we assumed that the action of genes was not *pleiotropic*; that is, with respect to neurocomputational parameters, we assumed that no gene affected the value of more than one parameter at once. This simplification likely will not hold in many cases, and certainly the current theoretical view is that the relationship between genes and *cognitive processes* is pleiotropic (see, e.g., Kovas & Plomin, 2006).

The assumption of polygenicity was motivated by the fact that we are using computational models to capture cognitive-level phenomena, and is a point worth emphasizing. We expect many low-level neural variations to influence neurocomputational functions at the level of cognitive processes in neural circuits. We therefore viewed it as unlikely that a single gene would modulate a neurocomputational parameter responsible for normal cognitive variation.

We assumed, for reasons of simplification only, that the combination of alleles for each polygenic neurocomputational parameter had a deterministic relation to the value of that parameter in the instantiated network: that is, the allele set alone determined the parameter value. Alternatively, this may be viewed as the assumption that the relation between alleles and parameter setting relied on an environment that did not vary across the individuals in the simulated population. We assumed (and did not instantiate) a much larger part of the genome that was species universal and was responsible for the basics of, for example, creating the processing units, the connections, the activation dynamics, the sensorium, the input-output connectivity pathways, and the mechanics of experience-dependent systems.

Turning to mechanisms of breeding, we assumed that there was sexual reproduction, so that each gene had a 50% chance of being passed on to a gamete (egg or sperm), which combined with a gamete of another individual to create a new offspring. Although reproduction was sexual in this sense, we did not consider sex effects in these simulations (i.e., there were no genetic differences between males and females). During breeding, we assumed that there was uniform crossover and no linkage disequilibrium, the latter falling beyond the scope of our simulations. That is, the presence or absence of a given allele in a gamete was independent of the presence or absence of any others. This assumption is violated in humans because genes on the same chromosome have a greater than 50% chance of being transferred into a gamete together, and the closer they lie on a chromosome, the higher the chance.

When Genetic Algorithms are used for machine learning optimization, the most successful individuals of the previous generation are often retained in the next generation. In our case, after breeding, the previous generation died. Breeding enabled the creation of individuals with different degrees of relatedness, for instance as twins or siblings. For some conditions, we created identical (monozygotic; MZ) or fraternal (dizygotic; DZ) twin pairs. MZ twins shared the same genome, while DZ twins and siblings were created by generating two offspring from the same set of parents, but from a different sperm and egg. DZ twins and siblings shared 50% of their alleles on average. Also in contrast to the more common use of Genetic Algorithms, we did not include genetic mutation during reproduction. In humans, the mutation rate is extremely low (e.g., Strachan & Read, 2003, cite a rate of between 1 and 4 mutations per 100,000 genes per generation). Mutations serve to reduce the average genetic similarity of siblings below 50%, and our preference was to maintain retain the 50% value, since it is the one deployed in standard behavioural genetic models. Several other aspects fell beyond the scope of the simulations. We did not model the effects of epistasis (interactions between genes) or epigenetic effects on gene expression; we did not model assortative mating – in our simulations, mates were selected at random from the population; and we did not model gene-environment correlations (Plomin et al., 2008) – variation in the composition of the environment had no correlation with the nature of an individual's genotype.

3.4 Parameter values and their link to the artificial genome for the past tense network

For the basic past tense network, the total of number of genes used to encode the value of the 14 computational parameters was 126 (or two copies of 63) 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; sparseness: 12 (total 126 bits).

Figure 3 plots the range of values for each parameter against their target frequency of occurrence in the population. The translation of a genome into a parameter set was implemented by assigning alleles the value of 1 or 0, and then deriving the total for all the genes influencing the parameter (thereby ensuring additivity). The parameter value was calculated from the total using a lookup table, created by hand for each parameter to reflect the range of values identified during the calibration stage. The lookup tables for the 14 parameters (in the Wide Genetic) condition are shown below.

Table S2. Lookup table linking the artificial genome to the Hidden Unit parameter, for the Wide Genetic Variation condition

	Hidden Unit Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	0.001	0.01	0.04	0.12	0.21	0.25	0.21	0.12	0.04	0.01	0.001
Parameter value	10	20	30	40	50	60	75	100	200	350	500

Table S3. Lookup table linking the artificial genome to the Temperature parameter, for the Wide Genetic Variation condition

	Temperature Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	0.001	0.01	0.04	0.12	0.21	0.25	0.21	0.12	0.04	0.01	0.001
Parameter value	0.0625	0.125	0.25	0.5	0.75	1	1.25	1.5	2	3	4

Table S4. Lookup table linking the artificial genome to the Noise parameter, for the Wide Genetic Variation condition

	Noise Parameter Value								
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8
Population probability	-	0.04	0.11	0.22	0.27	0.22	0.11	0.03	0.00
Parameter value	0	0	0.05	0.1	0.2	0.5	2	4	6

Table S5. Lookup table linking the artificial genome to the Learning Rate parameter, for the Wide Genetic Variation condition

	Learning Rate Parameter Value												
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10	11	12
Population probability	0.0002	0.0029	0.02	0.05	0.12	0.19	0.23	0.19	0.12	0.05	0.02	0.0029	0.0002
Parameter value	0.005	0.01	0.025	0.05	0.075	0.1	0.125	0.15	0.175	0.2	0.25	0.3	0.5

Table S6. Lookup table linking the artificial genome to the Momentum parameter, for the Wide Genetic Variation condition

	Momentum Parameter Value								
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8
Population probability	0.004	0.03	0.11	0.22	0.27	0.22	0.11	0.03	0.004
Parameter value	0	0.05	0.1	0.15	0.2	0.35	0.5	0.6	0.75

Table S7. Lookup table linking the artificial genome to the Weight Variation parameter, for the Wide Genetic Variation condition

	Weight Variation Parameter Value								
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8
Population probability	0.004	0.03	0.11	0.22	0.27	0.22	0.11	0.03	0.004
Parameter value	0.01	0.05	0.1	0.25	0.5	0.75	1	2	3

Table S8. Lookup table linking the artificial genome to the Architecture parameter, for the Wide Genetic Variation condition. (0 = 2-layer, 1 = 3-layer, 2 = fully-connected)

	Architecture Parameter Value						
Number of 1-valued alleles	0	1	2	3	4	5	6
Population probability	-	0.109	-	0.781	-	0.109	-
Parameter value	0	0	1	1	1	2	2

Table S9. Lookup table linking the artificial genome to the Learning Algorithm parameter, for the Wide Genetic Variation condition. (0 = Euclidean distance error metric, 1 = cross-entropy error metric)

	Learning Algorithm Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population	0.063	0.938	-	-	-

probability					
Parameter value	0	1	1	1	1

Table S10. Lookup table linking the artificial genome to the Nearest Neighbor Threshold parameter, for the Wide Genetic Variation condition

	Nearest Neighbor Threshold Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	0.001	0.010	0.044	0.117	0.451	-	0.205	0.117	0.044	0.011	-
Parameter value	0.0025	0.005	0.01	0.025	0.1	0.1	0.15	0.2	0.25	0.5	0.5

Table S11. Lookup table linking the artificial genome to the Pruning Onset parameter, for the Wide Genetic Variation condition

	Pruning Onset Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	0.001	0.01	0.04	0.12	-	0.45	0.21	0.12	0.04	0.01	0.001
Parameter value	1000	500	250	150	100	100	75	50	25	20	0

Table S12. Lookup table linking the artificial genome to the Pruning Probability parameter, for the Wide Genetic Variation condition

	Pruning Probability Parameter Value								
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8
Population probability	0.004	0.03	0.11	-	0.49	0.22	0.11	0.03	0.004
Parameter value	0	0.01	0.025	0.05	0.05	0.1	0.5	0.75	1

Table S13. Lookup table linking the artificial genome to the Pruning Threshold parameter, for the Wide Genetic Variation condition

	Pruning Threshold Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	0.001	0.01	0.04	0.12	-	0.66	-	0.12	0.04	0.01	0.001

(%)											
Parameter value	0.1	0.2	0.3	0.4	0.5	0.5	0.5	0.75	1	1.25	1.5

Table S14. Lookup table linking the artificial genome to the Weight Decay parameter, for the Wide Genetic Variation condition

	Weight Decay Parameter Value										
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10
Population probability	-	-	-	-	0.38	0.25	0.21	0.12	0.04	0.01	0.001
Parameter value	0	0	0	0	0	1x10^{-7}	2x10^{-7}	9.8x10^{-7}	19.7x10^{-7}	98.4x10^{-7}	196.9x10^{-7}

Table S15. Lookup table linking the artificial genome to the Sparseness parameter, for the Wide Genetic Variation condition

	Sparseness Parameter Value												
Number of 1-valued alleles	0	1	2	3	4	5	6	7	8	9	10	11	12
Population probability	-	-	-	-	-	-	0.61	0.19	0.12	0.05	0.02	0.003	0.0002
Parameter value	0	0	0	0	0	0	0	0.05	0.1	0.2	0.3	0.4	0.5

A more constricted range of genetic variation was also considered for each computational parameter in the network, shown in Figure S10 in gray. This required fewer genes to encode, leading to a genome with only 60 genes (2 copies of 30). For one of the parameters, learning algorithm, there were only had two values in the original formulation; we restricted the range of variation by fixing the parameter to use cross-entropy, thus removing variation in this gene. On average, the parameters of the narrow condition had 40% of the range of variation of the wide condition. The lookup tables for the Narrow Genetic condition are shown below.

Table S16. Lookup table linking the artificial genome to the Hidden Unit parameter, for the Narrow Genetic Variation condition

	Hidden Unit Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	30	40	50	60	75

Table S17. Lookup table linking the artificial genome to the Temperature parameter, for the Narrow Genetic Variation condition

	Temperature Parameter Value						
Number of 1-valued alleles	0	1	2	3	4	5	6
Population probability	0.02	0.09	0.23	0.31	0.23	0.09	0.02
Parameter value	0.25	0.5	0.75	1	1.25	1.5	1.75

Table S18. Lookup table linking the artificial genome to the Noise parameter, for the Narrow Genetic Variation condition

	Temperature Parameter Value						
Number of 1-valued alleles	0	1	2	3	4	5	6
Population probability	0.02	0.09	0.23	0.31	0.23	0.09	0.02
Parameter value	0	0.1	0.25	0.5	0.75	1	2

Table S19. Lookup table linking the artificial genome to the Learning Rate parameter, for the Narrow Genetic Variation condition

	Learning Rate Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	0.05	0.075	0.1	0.125	0.15

Table S20. Lookup table linking the artificial genome to the Momentum parameter, for the Narrow Genetic Variation condition

	Momentum Parameter Value		
Number of 1-valued alleles	0	1	2
Population probability	0.25	0.50	0.25
Parameter value	0.1	0.2	0.3

Table S21. Lookup table linking the artificial genome to the Weight Variation parameter, for the Narrow Genetic Variation condition

	Weight Variation Parameter Value						
Number of 1-valued alleles	0	1	2	3	4	5	6
Population probability	0.02	0.09	0.23	0.31	0.23	0.09	0.02
Parameter value	0.05	0.1	0.25	0.5	0.75	1	1.5

Table S22. Lookup table linking the artificial genome to the Architecture parameter, for the Narrow Genetic Variation condition. (0 = 2-layer, 1 = 3-layer, 2 = fully-connected)

	Architecture Parameter Value		
Number of 1-valued alleles	0	1	2
Population probability	-	0.75	0.25
Parameter value	1	1	2

Table S23. Lookup table linking the artificial genome to the Learning Algorithm parameter, for the Narrow Genetic Variation condition. (0 = Euclidean distance error metric, 1 = cross-entropy error metric)

	Learning Algorithm Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	-	-	1.00	-	-
Parameter value	1	1	1	1	1

Table S24. Lookup table linking the artificial genome to the Nearest Neighbor Threshold parameter, for the Narrow Genetic Variation condition

	Nearest Neighbor Threshold Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	0.025	0.05	0.1	0.2	0.5

Table S25. Lookup table linking the artificial genome to the Pruning Onset parameter, for the Narrow Genetic Variation condition

	Pruning Onset Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	50	75	100	125	150

Table S26. Lookup table linking the artificial genome to the Pruning Probability parameter, for the Narrow Genetic Variation condition

	Pruning Probability Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	0.025	0.05	0.1	0.2	0.3

Table S27. Lookup table linking the artificial genome to the Pruning Threshold parameter, for the Narrow Genetic Variation condition

	Pruning Threshold Parameter Value						
Number of 1-valued alleles	0	1	2	3	4	5	6
Population probability	0.02	0.09	0.23	0.31	0.23	0.09	0.02
Parameter value	0.2	0.3	0.4	0.5	0.75	1	1.25

Table S28. Lookup table linking the artificial genome to the Weight Decay parameter, for the Narrow Genetic Variation condition

	Weight Decay Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	$98.0\text{x}10^{-7}$	$20.0\text{x}10^{-7}$	$10.0\text{x}10^{-7}$	$2.0\text{x}10^{-7}$	0

Table S29. Lookup table linking the artificial genome to the Sparseness parameter, for the Narrow Genetic Variation condition

	Sparseness Parameter Value				
Number of 1-valued alleles	0	1	2	3	4
Population probability	0.06	0.25	0.38	0.25	0.06
Parameter value	0.4	0.3	0.2	0.1	0

4. References

Beversdorf, D. Q., Narayanan, A., Hillier, A., & Hughes, J. D. (2007). Network model of decreased context utilization in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *37*, 1040– 1048.

Brant, A. M., Munakata, Y., Boomsma, D. I., DeFries, J. C., Haworth, C. M. A., Keller, M. C., Martin, N. G., McGue, M., Petrill, S. A., Plomin, R., Wadsworth, S. J., Wright, M. J., & Hewitt, J. K. (2013). The nature and nurture of high IQ: An extended sensitive period for intellectual development. *Psychol Sci*. 2013 Aug; 24(8): 1487–1495. Published online 2013 Jul 1. doi: 10.1177/0956797612473119

Cohen, I. L. (1998). Neural network analysis of learning in autism. In D. J. Stein & J. Ludik (Eds.), *Neural networks and psychopathology* (pp. 274–315). New York, NY: Cambridge University Press.

Hanscombe, K. B., Trzaskowski, M., Haworth, C. M. A., Davis, O. S. P., Dale, P. S., & Plomin, R. (2012). Socioeconomic status (SES) and children's intelligence (QI): In a UK-representative sample SES moderates the environmental, not genetic, effects on IQ. *PLoS ONE* 7(2): e30320. doi:10.1371/journal.pone.0030320

Harm, M. W. & Seidenberg, M. S. (1999). Phonology, reading acquisition, and dyslexia: Insights from connectionist models. *Psychological Review*, *106*, 491-528.

Harm, M. W., & Seidenberg, M. S. (2004). Computing the meanings of words in reading: Cooperative division of labor between visual and phonological processes. *Psychological Review*, *111*, 662-720.

Hinton, G. (1989). Connectionist learning procedures. *Artificial Intelligence*, *40*, 185–234.

Hoeffner, J. H. & McClelland, J. L. (1993). Can a perceptual processing deficit explain the impairment of inflectional morphology in developmental dysphasia? A computational investigation. In E.V. Clark (Ed), *Proceedings of the 25th Child language research forum*, (pp. 1-25). Stanford University Press.

Huttenlocher, P. R. (2002). *Neural plasticity: The effects of environment on the development of the cerebral cortex.* Cambridge, MA: Harvard University Press.

Joanisse, M. F., & Seidenberg, M. S. (1999). Impairments in verb morphology after brain injury: A connectionist model. *Proceedings of the National Academy of Sciences of the United States of America*, *96*, 7592-7597.

Joanisse, M. F. (2004). Specific language impairments in children: Phonology, semantics and the English past tense. *Current Directions in Psychological Science*, *13(4)*, 156-160.

Joanisse, M. F., Seidenberg, M. S. (2003). Phonology and syntax in Specific Language Impairments: Evidence from a connectionist model. *Brain and Language*, *86*, 40-56.

Karaminis, T. N., & Thomas, M. S. C. (2010). A cross-linguistic model of the acquisition of inflectional morphology in English and Modern Greek. In S. Ohlsson & R. Catrambone (Eds.), *Proceedings of 32nd Annual Conference of the Cognitive Science Society*, August 11-14, 2010, Portland, Oregon, USA.

Kovas, Y., & Plomin, R. (2006). Generalist genes: implications for the cognitive sciences. *Trends in Cognitive Sciences*, *10(5)*, 198-203.

Li, S.-C. & Lindenberger, U. (1999). Cross-level unification: A computational exploration of the link between deterioration of neurotransmitter systems and the dedifferentiation of cognitive abilities in old age, (pp. 103-146) in L.-G. Nilsson & H. Markowitsch (Eds.). *Cognitive neuroscience of memory*. Toronto: Hogrefe & Huber

McClelland, J. L. (1993) Toward a theory of information processing in graded, random, interactive networks. In D. E. Meyer & S. Kornblum (Eds.), *Attention and performance XIV: Synergies in experimental psychology, artificial intelligence and cognitive neuroscience* (pp. 655-688). Cambridge, MA: MIT Press.

McClelland, J. L. (2000). The basis of hyperspecificity in autism: A preliminary suggestion based on properties of neural nets. *Journal of Autism and Developmental Disorders*, *30*, 497–502

Munakata, Y., & McClelland, J. L. (2003). Connectionist models of development. *Developmental Science*, *6(4)*, 413-429.

Noble, K. G., Houston, S. M., Brito, N. H., Bartsch, H., Kan, E., Kuperman, J. M., Akshoomoff, N., Amaral, D. G., Bloss, C. S., Libiger, O., Schork, N. J., Murray, S. S., Casey, B. J., Chang, L., Ernst, T. M., Frazier, J. A., Gruen, J. R., Kennedy, D. N., Van Zijl, P., Mostofsky, S., Kaufmann, W. E., Kenet, T., Dale, A. M., Jernigan, T. L., & Sowell, E. R. (2015). Family income, parental education and brain structure in children and adolescents. *Nat Neurosci*. 2015 May;18(5):773-8. doi: 10.1038/nn.3983. Epub 2015 Mar 30.

Plaut, D. C. (1997). Structure and function in the lexical system: Insights from distributed models of word reading and lexical decision. *Language and Cognitive Processes*, *12*, 767-808.

Plaut, D. C., McClelland, J. L., Seidenberg, M. S., & Patterson, K. E. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review*, *103*, 56-115.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics (5th Edition)*. New York: Worth Publishers.

Plunkett, K. & Marchman, V. (1991). U-shaped learning and frequency effects in a multilayered perceptron: Implications for child language acquisition. *Cognition*, *38*, 1-60.

Plunkett, K. & Marchman, V. (1993). From rote learning to system building: acquiring verb morphology in children and connectionist nets. *Cognition*, *48*, 21-69.

Richardson, F. M., Forrester, N. A., Baughman, F. D., & Thomas, M. S. C. (2006b). Computational modeling of variability in the conservation Task. In *Proceedings of the 28th Annual Conference of the Cognitive Science Society* (p. 2010-2015), July 26-29, Vancouver, BC, Canada.

Richardson, F. M., Seghier, M. L., Leff, A. P., Thomas, M. S. C., & Price, C. J. (2011). Multiple routes from occipital to temporal cortices during reading. *Journal of Neuroscience*, *31(22)*, 8239-8247.

Richardson, F.M., Baughman, F.D., Forrester, N. A., & Thomas, M.S.C. (2006a). Computational modeling of variability in the balance scale task. *Proceedings of the 7th International Conference of Cognitive Modeling*.

Rumelhart, D. E., Hinton, G. E., & Williams, R. J. (1986). Learning internal representations by error propagation. In D. E. Rumelhart, J. L. McClelland and The PDP Research Group, *Parallel distributed processing: Explorations in the* *microstructure of cognition. Vol. 1: Foundations* (pp. 318-362). Cambridge, MA: MIT Press.

Sapolsky, R. (2005). *Biology and human behavior: The neurological origins of individuality (2nd Ed.)*. Chantilly, VA: The Teaching Company.

Seghier, M. L., Lee, H. L., Schofield, T., Ellis, C. L., & Price, C. (2008). Inter-subject variability in the use of two different neuronal networks for reading aloud familiar words. *Neuroimage*, *42(3-3)*, 1226-1236.

Simmons, D. R., McKay, L., McAleer, P., Toal, E., Robertson, A., & Pollick, F. E. (2007). Neural noise and autism spectrum disorders. *Perception*, *36(Suppl.)*, 119–120.

Strachan, T., & Read, P. (2003). *Human molecular genetics 3*. Garland Publishing.

Thomas, M. S. C. (2005). Characterising compensation. *Cortex*, *41(3)*, 434-442.

Thomas, M. S. C., Forrester, N. A., & Ronald, A. (2013). Modeling socio-economic status effects on language development. *Dev Psychol*. 2013 Dec;49(12):2325-43. doi: 10.1037/a0032301.

Thomas, M. S. C., Forrester, N. A. & Ronald, A. (2016), Multiscale modeling of gene–behavior associations in an artificial neural network model of cognitive development. *Cognitive Science*. doi: 10.1111/cogs.12230

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

Thomas, M. S. C. & Karmiloff-Smith, A. (2002). Are developmental disorders like cases of adult brain damage? Implications from connectionist modeling. *Behavioral and Brain Sciences*, *25(6)*, 727-788.

Thomas, M. S. C. & Karmiloff-Smith, A. (2003a). Connectionist models of development, developmental disorders and individual differences. In R. J.

Sternberg, J. Lautrey, & T. Lubart (Eds.), *Models of Intelligence: International Perspectives*, (p. 133-150). American Psychological Association.

Thomas, M. S. C. & Karmiloff-Smith, A. (2003b). Modeling language acquisition in atypical phenotypes. *Psychological Review*, *Vol. 110*, *No.4*, 647-682.

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. DOI:10.1044/2013_JSLHR-L-12-0254.

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., & McClelland, J. L. (2008). Connectionist models of cognition. In R. Sun (Ed.), *Cambridge handbook of computational cognitive modeling* (pp. 23-58). Cambridge: Cambridge University Press.

Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, *14(6)*, 623-628.

Westermann, G (1998) Emergent modularity and U-shaped learning in a constructivist neural network learning the English past tense. In M. A. Gernsbacher & S. J. Derry (Eds.), *Proceedings of the Twentieth Annual Conference of the Cognitive Science Society* (pp. 1130-1135). Hillsdale, NJ: Erlbaum.

Woollams, A. M., Joanisse, M., & Patterson, K. (2009). Past-tense generation from form versus meaning: Behavioral data and simulation evidence. *Journal of Memory and Language*, *61*, 55-76.

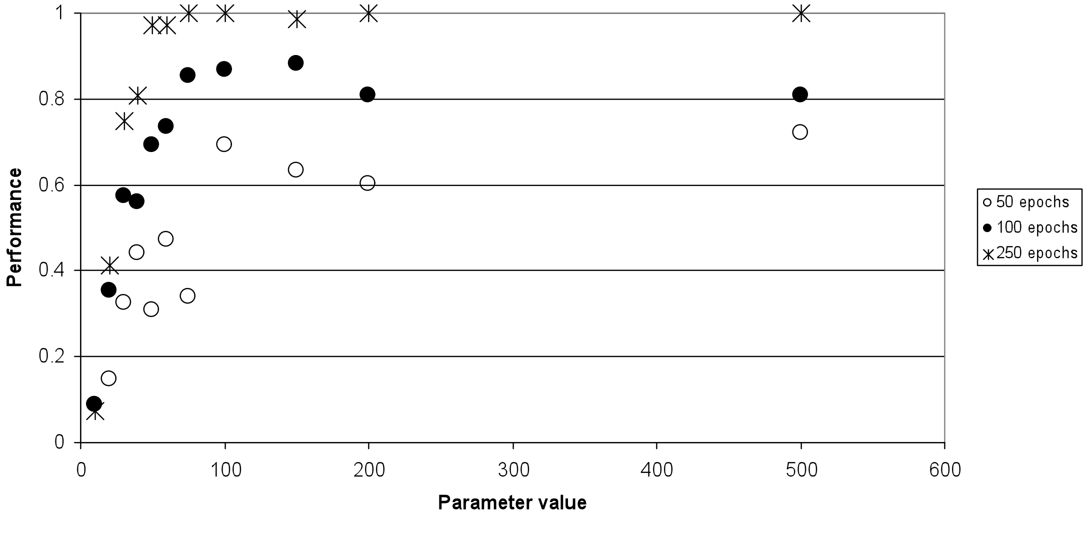
Zorzi, M., Houghton, G., & Butterworth, B. (1998). Two routes or one in reading aloud? A connectionist dual-process model. *Journal of Experimental Psychology: Human Perception and Performance*, *24*, 1131-1161.

Appendix A

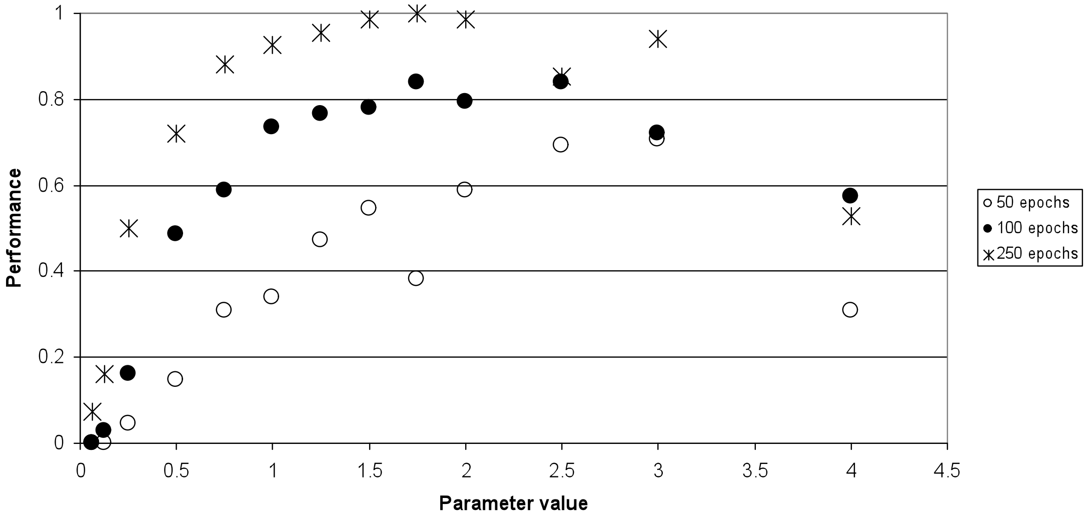
Parameter-behavior functions for the base past tense network

All other parameters were held at default values, while a single parameter was varied. Performance is reported for regular verbs on the past tense task. The shape of the function is displayed for three points in training, 50, 100, and 250 epochs. The default parameter values were: hidden units: 50; temperature: 1; noise: 0; learning rate: 0.01; momentum: 0.2; weight variance: 0.5; architecture: 3-layer; learning algorithm: back propagation error measure; nearest neighbor threshold: 0.1; pruning onset epoch: 50; pruning probability: 0.1; pruning threshold: 0.5; weight decay: 0.000019; sparseness: 90% connectivity.

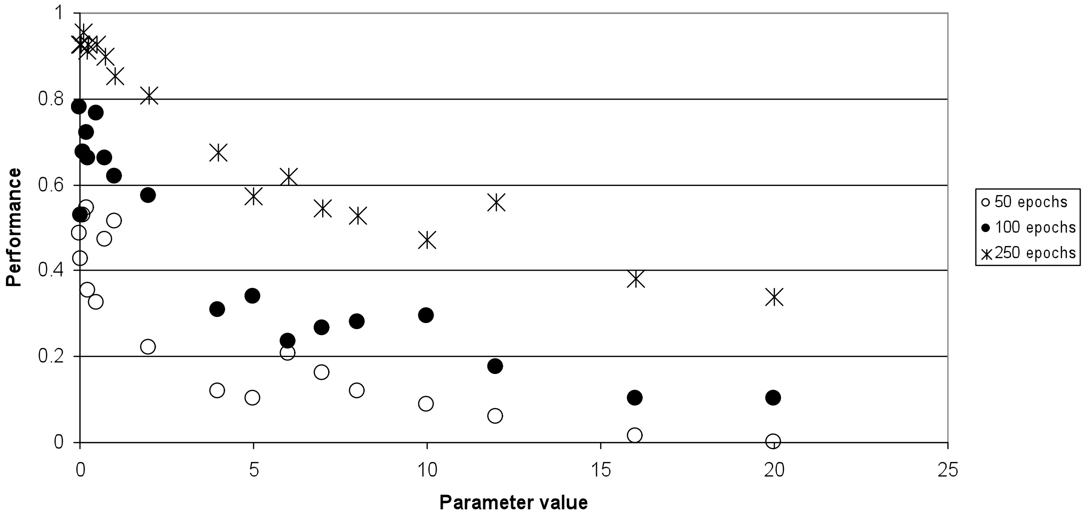
Hidden units:



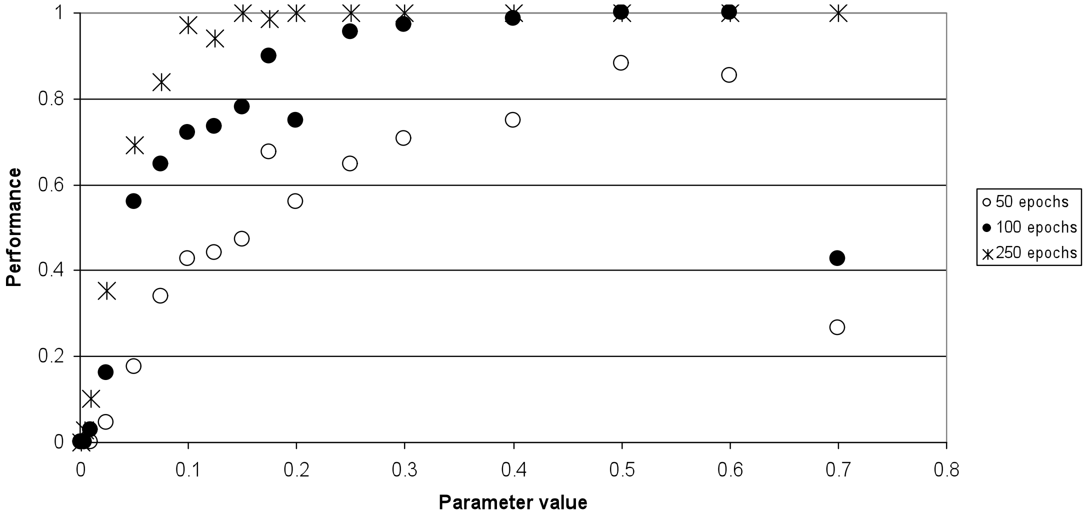
Unit threshold function (temperature)



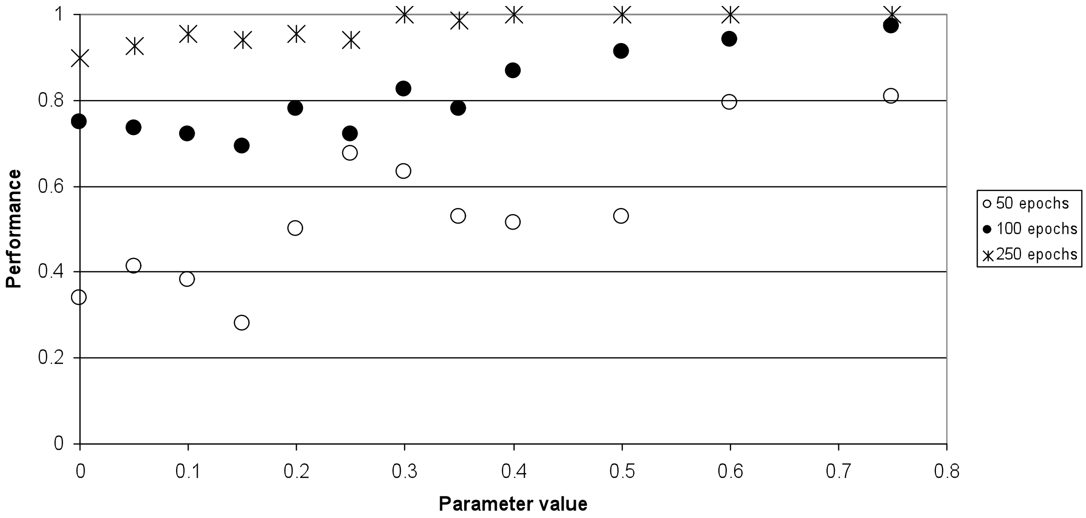
Processing noise:



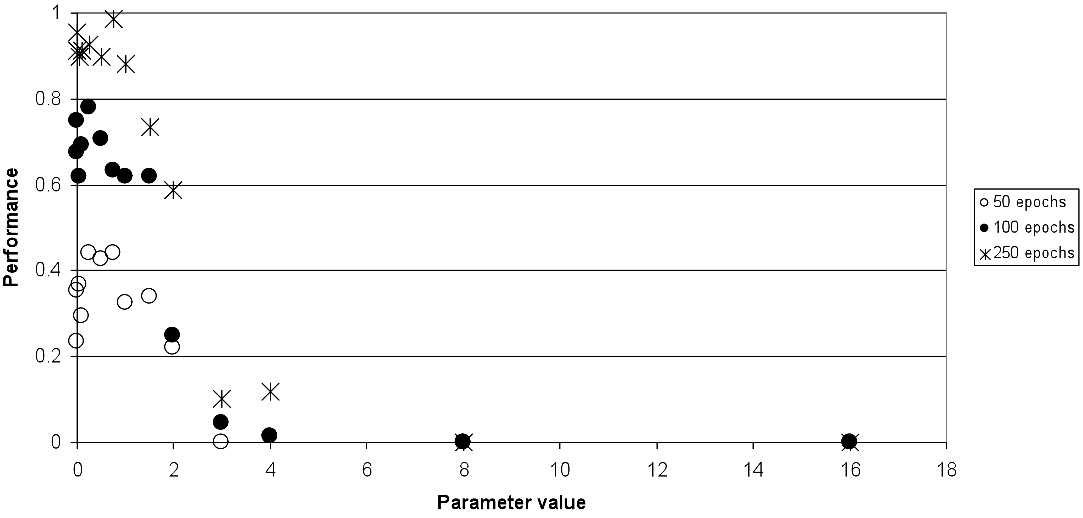
Learning rate:



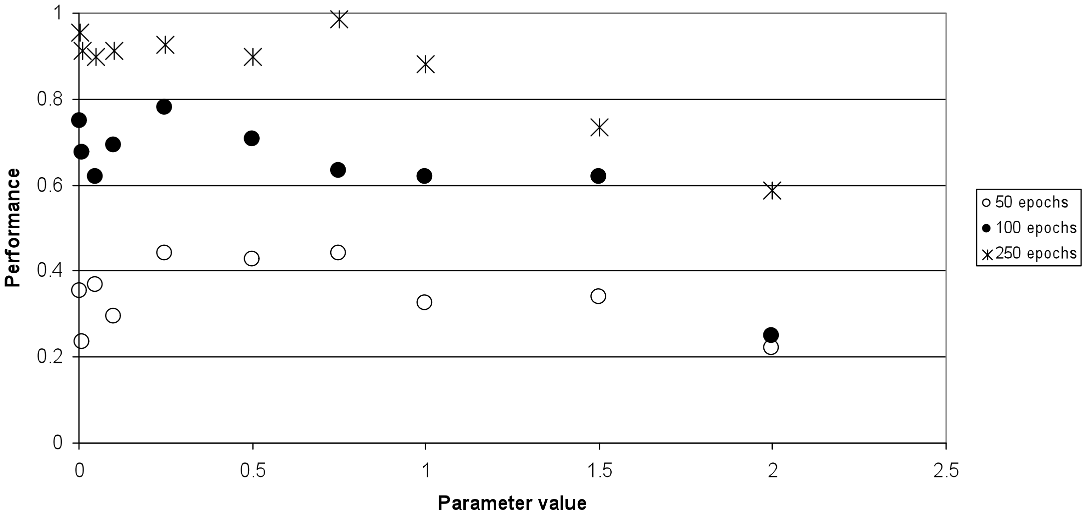
Momentum:



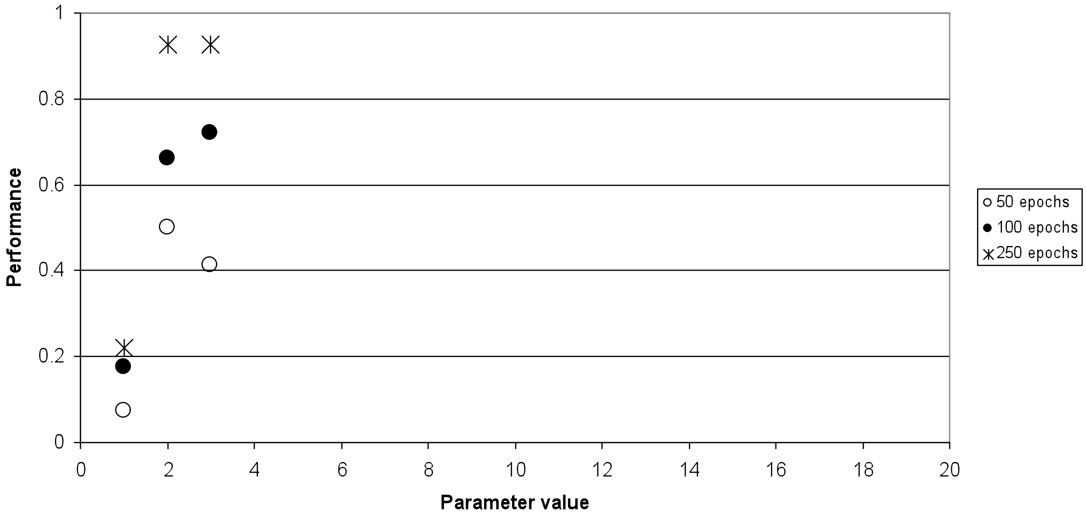
Initial weight variance



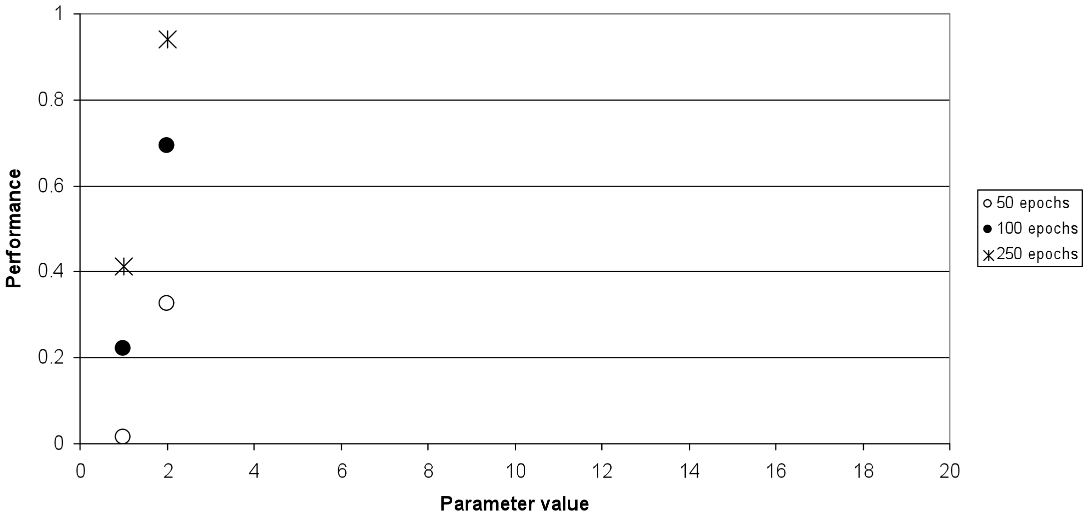
Initial weight variance (as above, focused on range 0 to 2)



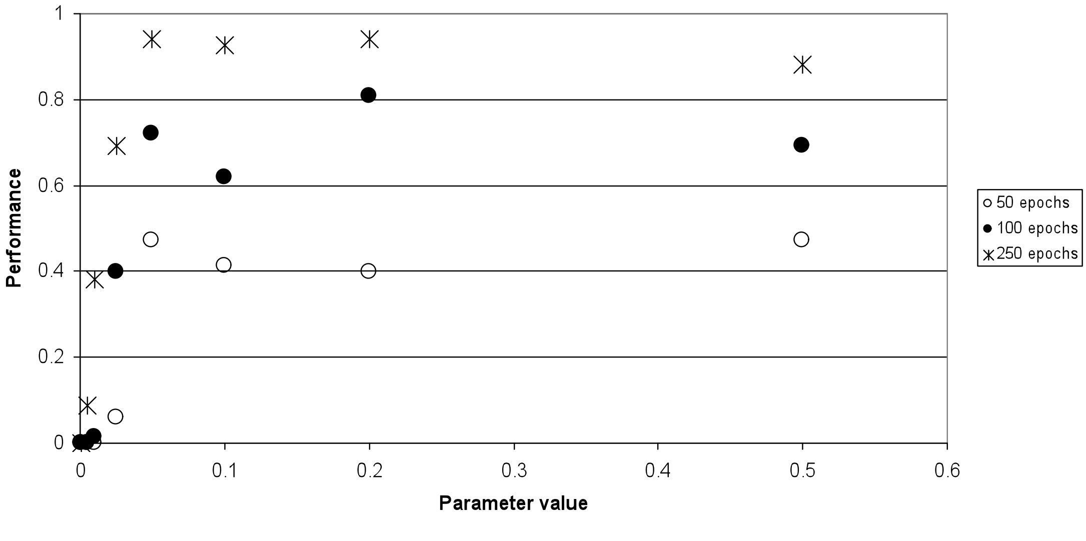
Architecture (1=2-layer; 2=3-layer; 3=fully connected):



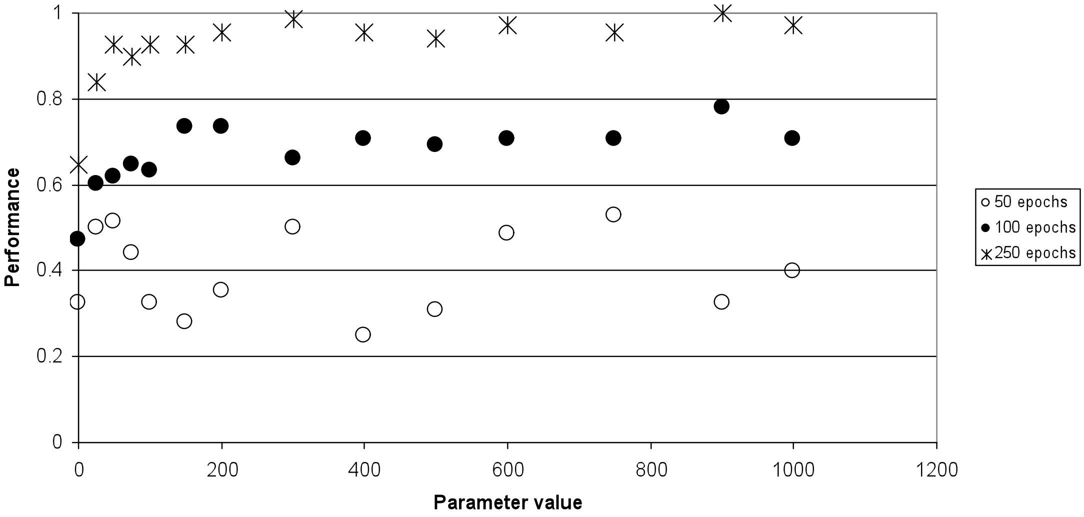
Learning algorithm error metric (1=Euclidean distance; 2=Cross-entropy):



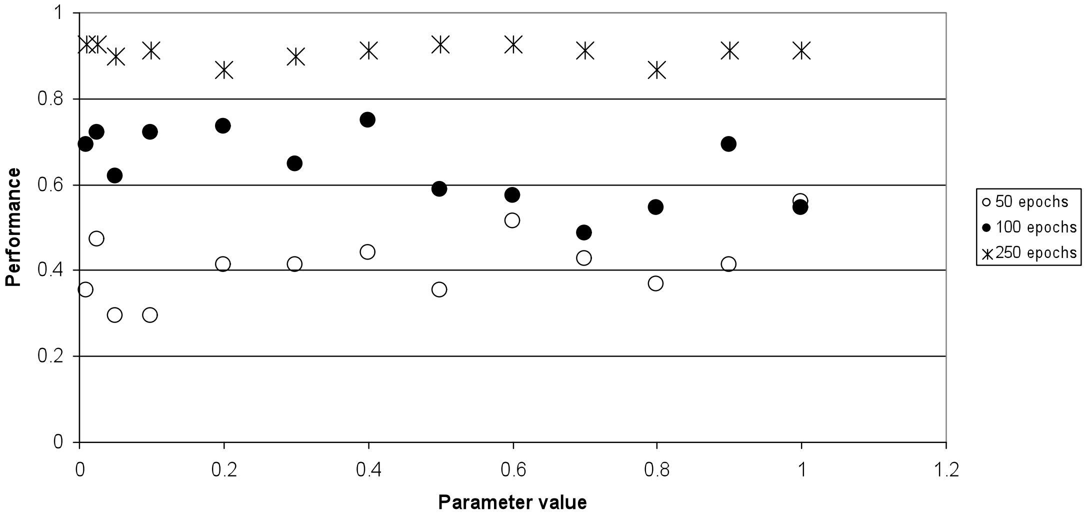
Nearest neighbor response threshold:



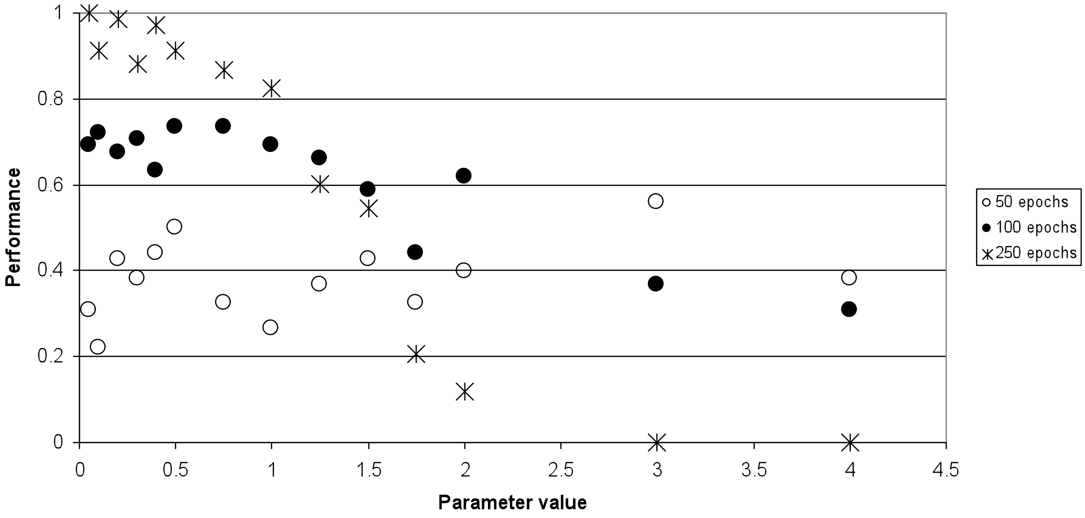
Pruning onset:



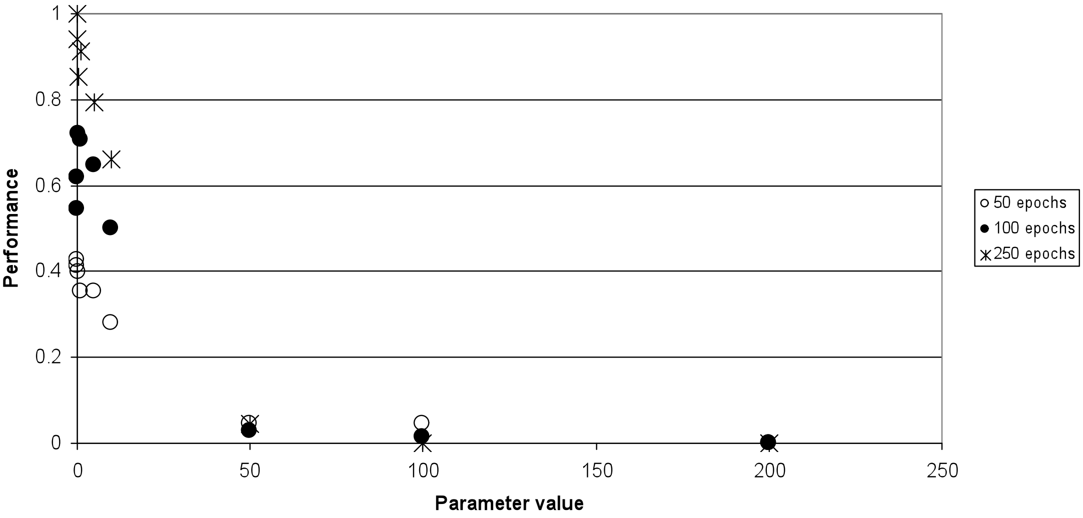
Pruning probability:



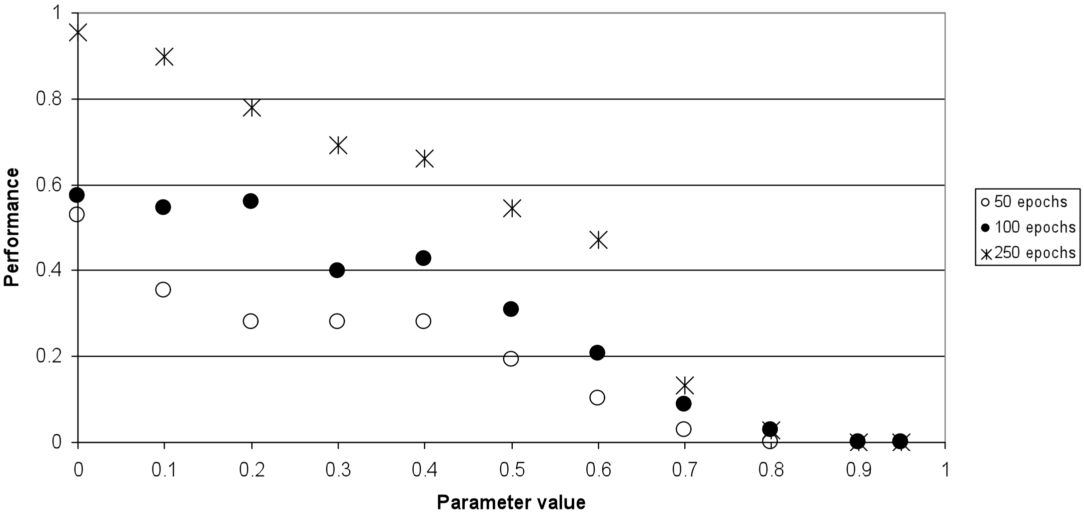
Pruning threshold:



Weight decay (total cumulative % of connectivity removed over full 1000 epochs of training):



Sparseness of initial connectivity (proportion removed):


Performance
Parameter value
○ 50 epochs
● 100 epochs
⋇ 250 epochs