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Do more intelligent brains retain heightened plasticity for longer in development? A computational investigation

Michael S.C. Thomas*

Developmental Neurocognition Lab, Centre for Brain & Cognitive Development, Birkbeck, University of London, United Kingdom

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ABSTRACT

Twin studies indicate that the heritability of general cognitive ability – the genetic contribution to individual differences – increases with age. Brant et al. (2013) reported that this increase in heritability occurs earlier in development for low ability children than high ability children. Allied with structural brain imaging results that indicate faster thickening and thinning of cortex for high ability children (Shaw et al., 2006), Brant and colleagues argued higher cognitive ability represents an *extended sensitive period* for brain development. However, they admitted no coherent mechanistic account can currently reconcile the key empirical data. Here, computational methods are employed to demonstrate the empirical data can be reconciled without recourse to variations in sensitive periods. These methods utilized population-based artificial neural network models of cognitive development. In the model, ability-related variations stemmed from the timing of the increases in the non-linearity of computational processes, causing dizygotic twins to diverge in their behavior. These occurred in a population where: (a) ability was determined by the combined small contributions of many neurocomputational factors, and (b) individual differences in ability were largely genetically constrained. The model's explanation of developmental increases in heritability contrasts with proposals that these increases represent emerging gene-environment correlations (Haworth et al., 2010). The article advocates simulating inherited individual differences within an explicitly developmental framework.

1. Introduction

1.1. Cognitive ability and brain structure

Structural properties of the brain have been found to correlate with individual differences in cognitive ability. For example, there is a correlation of brain size to general cognitive ability[1] of between 0.1 and 0.3 (McDaniel, 2005). When Ritchie et al. (2015) used a range of measures from structural brain imaging to predict general cognitive ability in adults, they found that brain volume explained 12% of the variance, cortical thickness another 5%, and all measures together up to 21% of the variance. It has been proposed that the timing and magnitude of developmental changes in structural indices, such as cortical thickness or cortical surface area, are the more important predictors of general cognitive ability than brain structure per se (Schnack et al., 2015). For example, in a longitudinal study tracing development from young childhood into early adulthood, Shaw et al. (2006) reported that a superior intelligence (121–149 IQ points) was associated with faster and more prolonged thickening of cortex in childhood and faster thinning in adolescence, with correlations between structure and ability at any time point falling between 0 and 0.1. Thickening and thinning effects differed between brain regions, being most noticeable in frontal and temporal regions. However, in this study, only the superior intelligence group showed reliable differences, with high (109–120 IQ points) and average (83–108 IQ points) IQ groups overlapping sufficiently to be statistically indistinguishable.

The relationship between general cognitive ability and brain structure is re-enforced by the finding that both are highly heritable (e.g., Thompson et al., 2001; Plomin and Spinath, 2004), where heritability is defined as the proportion of phenotypic variability explained by genetic similarity. Moreover, bivariate analyses reveal that the heritability of ability and structure is explained by partially overlapping genes (e.g., Posthuma et al., 2002). This finding extends

Abbreviations: IQ, intelligence quotient; MZ, monozygotic; DZ, dizygotic; ANN, artificial neural network; SES, socio-economic status.

* Correspondence address: Department of Psychological Sciences, Birkbeck, University of London, Malet Street, Bloomsbury, London WC1E 7HX, United Kingdom.
E-mail address: m.thomas@bbk.ac.uk

[1] Some authors use the terms 'ability' and 'intelligence' interchangeably. Others make a distinction whereby 'cognitive ability' is used to refer to the mental processes and mechanisms required to execute tasks, while 'intelligence' is used to describe a score on a test that relates an individual's cognitive ability to that of the general population. Here, instead of 'intelligence', we mostly use the term 'general cognitive ability'.

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to the rate of change of structural properties such as cortical thickness (Brans et al., 2010). The observation that general cognitive ability is related to dynamic properties of cortical maturation led Shaw et al. (2006) to propose that 'the prolonged phase of prefrontal cortical gain in the most intelligent might afford an even more extended "critical" period for the development of high-level cognitive cortical circuits' (p. 678), that is, a period of heightened sensitivity to variation in environmental influences.

However, Brant et al. (2013) noted that extended structural brain development does not necessarily imply longer sensitivity to environmental influences. That is, the brain data provide no direct evidence for individual differences in the length of a sensitive period associated with cognitive ability (see Thomas and Johnson, 2008; for discussion of the notion of sensitive periods in brain development). Brant et al. (2013) sought out more direct evidence by taking advantage of the finding that the heritability of general cognitive ability increases linearly with age (Haworth et al., 2010). The common explanation for this pattern is that the aged-related increase marks an emerging gene-environment correlation, which then exaggerates the influence of the initial genetic differences on performance (Briley and Tucker-Drob, 2013; Haworth et al., 2010). As individuals become more autonomous with increasing age, they seek out environments that match their genetic profiles. For example, brighter children may seek out more stimulating environments. Environmental influences then cause an exaggeration of initial genetic differences. Haworth et al. described the gene-environment correlation thus: 'as children grow up, they increasingly select, modify and even create their own experiences in part based on their genetic predispositions' (2010, p. 1112).

Brant et al. (2013) reasoned that if higher cognitive ability corresponds to an extended sensitive period in brain development, individuals with higher ability should remain sensitive to environmental variation for longer. Greater influence of environmental variation translates to lesser influence of genetic variation. The prediction was therefore that in higher ability individuals, the rise in heritability should occur later. Combining data from over 10,000 monozygotic (MZ) and dizygotic (DZ) twin pairs in a cross-sectional study, and around 400 MZ and DZ pairs in a longitudinal study, they found support for this hypothesis (see Fig. 1a). High and low ability groups showed similar heritability in childhood (4–12 years). By adolescence (13–18 years), the low ability group now showed an increase in heritability, while the high ability group continued to show the same lower level observed in childhood. In adulthood (18+ years), both groups now showed similarly high heritability.

There are, however, some difficulties with the picture. There is little understanding of the low-level mechanisms linked to neural processing that underpin the macro cortical changes in indices such as thickness or surface area; or, indeed, whether the cortical changes reflect intrinsic genetic processes or the influence of environmental variables such as socio-economic status (SES) (Shaw et al., 2006; Noble et al., 2015). Shaw et al. (2006) suggested that cortical thickening might correspond to experience-dependent molding of the architecture of cortical columns along with dendritic spine and axonal remodeling, while the thinning observed in adolescence might reflect the refining of neural circuits via use-dependent selective elimination of synapses. But hypotheses of this form largely rely on animal models, and the link to the development of high-level cognitive behavior is not demonstrated. Indeed, there is disagreement about which are the key structural indices and how they relate to function, with absolute cortical volume, cortical thickness, and cortical surface area all implicated, but showing different developmental relationships to cognitive ability (see, e.g., Noble et al., 2015).

The consequence of an absent mechanistic account is illustrated by Brant et al.'s (Brant et al. 2013) admission that they were unable to derive a coherent causal account of their findings. The hypothesis that protracted development is beneficial for the acquisition of higher and uniquely human cognitive functions as measured by intelligence quotient (IQ) does not suffice, because individuals with an eventual higher IQ tend to score higher in tests from early in development (Columbo and Frick, 1999; Deary et al., 2000)—not just in adolescence, when the benefit of protracted sensitivity would become apparent. Neither does the association of longer environmental sensitivity in high IQ fit with the common explanation of the age-related increase in the heritability of general cognitive ability, as an emerging gene-environment correlation. As Brant et al. 2013 put it, one would need to 'posit, counter-intuitively, that higher-IQ individuals seek out environments concordant with their genetic propensities *later* in development than do lower-IQ individuals' (2013, p.1493, italics added). Brant et al. (2013) concluded that the reason for developmental increases in the heritability of IQ remains unclear.

1.2. Population-level computational modeling of development

Computational modeling provides a method to clarify theoretical proposals via implementation, to unify empirical data with respect to common mechanisms, and to generate novel predictions. Its main disadvantage involves the simplifications required for implementation. Artificial neural networks (ANNs) have been used widely in the modeling of cognitive development (e.g., Spencer et al., 2009; Thomas and McClelland, 2008). Recently, these models have been used to investigate associations between levels of description, including those between genes, brain structure, brain activation, and behavior (Thomas et al., 2016). While much simplified and focusing on development within a single computational mechanism, the formalism of the ANN that the authors employed had several useful properties for this purpose. The model comprised an associative network with distributed processing across a network of simple integrate-and-fire processing units; behavior was acquired via an experience-dependent developmental process, which involved interaction with a structured and variable learning environment and gradual alterations in network connectivity strengths; and the developmental trajectory and final representational states of each network were constrained by parameters with analogues in neurocomputation, such as the activation function of the neurons, the number of neurons, and the connection density.

Three aspects of the Thomas et al. (2016) model make it useful for addressing the current empirical data. First, the model simulated cognitive development in populations of individuals, where variability in trajectories arose from intrinsic neurocomputational sources or extrinsic environmental sources (see also, Thomas and Knowland, 2014; for the application of this method to modeling sub-types of language delay; Thomas et al., 2013, for its application to modeling socio-economic status effects on language development). Second, the model included an artificial genome that specified the neurocomputational properties of the ANN. This allows modeling of genetic similarity between individuals, including creating identical and non-identical twin pairs. Twin study designs can then be simulated, which are the principal method to measure the heritability of individual differences. Third, the output of model can be viewed as generating behavior, while changes in structural properties of the ANNs, such as their connectivity, can be viewed as potentially informative of mechanisms contributing to structural changes in the brain. As indicated above, there is no consensus on the low-level neural mechanisms responsible for macro changes in brain structure. The ANN included two potentially relevant properties, one analogous to thinning (the decay and pruning of unused connectivity) and one analogous to thickening (the accumulation of connectivity strength), which were both central to the developmental process. Individual differences in these properties can provide candidate hypotheses for factors that contribute to