Neuroconstructivism: understanding typical and atypical trajectories of development

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Abstract

In this article, we give an overview of neuroconstructivism as a theory of cognitive development. Neuroconstructivism seeks to integrate a Piagetian perspective, that development constitutes a progressive elaboration in the complexity of mental representations via experience-dependent processes, with emerging findings on the nature of functional brain development. It is therefore premised on the view that theories of cognition should be constrained by the properties of the substrate in which cognition is implemented. We identify the origins of neuroconstructivist approaches, and summarise the core tenets of the theory with respect to typical and atypical development. We then consider three aspects of neuroconstructivism. First we address in more detail the idea that theories of cognition should be constrained by evidence from brain function. Second, we consider some of the methodological advances made to improve the analysis of developmental trajectories, particularly with respect to developmental disorders. Third, we give examples of the use of computational approaches to understand mechanisms of development, including connectionist modelling and dynamical systems theory. We finish by considering some of the challenges that lie ahead for neuroconstructivism.

Keywords

Neuroconstructivism, cognitive development, atypical development, individual differences, trajectory analysis, computational modelling

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Neuroconstructivism is a relatively recent theory of cognitive development that seeks to integrate a Piagetian perspective – that development constitutes a progressive elaboration in the complexity of mental representations via experience-dependent processes – with emerging findings on the nature of functional brain development. As such, it is premised on the view that theories of cognition should be constrained by the properties of the substrate in which the cognitive system is implemented. While much of developmental psychology has identified the abilities that infants and children exhibit at different ages, neuroconstructivism is concerned with understanding the nature of the mechanisms that allow transition between these different states, and the extent of their reliance on interaction with the environment. It therefore integrates research from different levels of analysis to model the multidimensional dynamics of development, including findings from cognitive studies, computational modelling, neuroimaging, and developmental and evolutionary biology. Its principal focus is on trajectories of development, both identifying and describing them empirically, and understanding the biological and environmental constraints that produce variations in trajectories, such as those observed in typical and atypical development.

In this article, we consider the origins of neuroconstructivism approaches, and the core proposals at the heart of the theory. We then consider three aspects of neuroconstructivism. First we address in more detail the idea that theories of cognition should be constrained by evidence from brain function. Second, we consider some of the recent methodological advances designed to improve the analysis of developmental trajectories, particularly with respect to

developmental disorders. Third, we give examples of the use of computational approaches to understand mechanisms of development, including connectionist modelling and dynamical systems theory. We finish by considering some of the challenges that lie ahead for neuroconstructivism.

Background

The emergence of neuroconstructivism in the 1990s was driven in part by new methods. These included advances in behavioural studies of infants, yielding a greater understanding of (limits on) infant knowledge and abilities; advances in neuroimaging techniques allowing insights into functional brain development, including such methods as structural and functional magnetic resonance imaging, electroencephalography, magnetoencephalography, and near-infrared spectroscopy; and advances in computational and robotic models that enable the testing of specific hypotheses regarding the mechanisms that drive developmental change. The term 'neuroconstructivism' was used by several authors, including Karmiloff-Smith (1998, 2006), and Quartz and Sejnowksi (1997), before the theoretical framework was articulated more fully by Mareschal and colleagues (Mareschal, Johnson, Sirois, Spratling, Thomas & Westermann, 2007; Mareschal, Sirois, Westermann & Johnson, 2007; Sirois et al., 2008; Westermann et al., 2007; Thomas et al., 2008; Thomas, 2000).

Theoretically, the emergence of neuroconstructivism was driven by two factors. The first was a desire to reconcile nativist and empiricist approaches to development by characterising the mechanisms by which biological and environmental factors interact, which underpinned the motivation for integrating data from disciplines at multiple levels of description. The second

factor was to respond to developmental theories that were strongly influenced by patterns of functional specialisation or modularity observed in the adult state. One might caricature such adult-state-inspired theories as 'componential' and 'uni-directional', to the extent that the cognitive system is viewed as built from independent pre-specialised components. Such theories include *deterministic epigenesis*, where genes are viewed as directly responsible for cognitive and brain outcomes; and *innate modularity*, where the function of cognitive components is specified prior to experience-dependent developmental processes, and development merely establish the content of cognitive mechanisms. In contrast, neuroconstructivism favours *probabilistic epigenesis* (see Gottlieb, 2002, 2007), which posits bi-direction interactions between genes, brain mechanisms, and the environment; and *emergent modularity*, where the cognitive and brain specialisations observed in adulthood are the product of domain-relevant computational biases combined with an experience-dependent developmental process rather than a precursor to development (Karmiloff-Smith, 1992, 1998; Johnson, 2000, 2001). The overarching principle of neuroconstructivism is context-dependence rather than independence, at each level of description. The action of genes takes place in the context of other gene expression, the operation of neurons takes place in the context of other neural activity, the development of brain regions takes place in the context of other brain regions, the brain develops in the context of the body, and the body operates in the context of a physical and social environment.

A brief overview of the tenets of neuroconstructivism

Mareschal et al. (2007) summarised the main tenets of neuroconstructivism in terms of a core principle, *context dependence*, three general mechanisms of *competition*, *cooperation* and *chronotopy*, two developmental processes of *proactivity* and *progressive specialisation*, and a single outcome of *partial representations*. In addition, they identified a set of developmental constraints operating at each level of description (see also Sirois et al., 2008; Westermann et al., 2007; Westermann, Thomas & Karmiloff-Smith, 2010). We define each of these aspects in turn.

At the heart of the theory is the idea that development concerns the elaboration of neural patterns of activation that result from experience. Change to knowledge comprises the emergence of new representations, realised by the process of neural elaboration. The emergence and development of representations is influenced by the principle of *context dependence.* The neural structures that give rise to mental representations are highly dependent on contexts that include the cellular, inter-brain regions, the whole brain, the body, and the social environment.

The mechanisms that drive development include *competition*, *cooperation* and *chronotopy*. Representational complexity is the outcome of these processes as cellular regions and networks gradually become more fine-tuned and resemble their adult function. Competition leads to the specialisation of components in a system. Its purpose is to allow for stable, minimal representations. Cooperation is a mechanism involved in the integration of multiple contributors to a function. Its purpose is to allow for overall efficiency through the coordination of interrelated functions. Competition and cooperation build a system that is minimal but involves a degree of redundancy that makes it

robust to damage. Chronotopy refers to the temporal aspect of development: events occur at a point in time that is defined by a temporal context, such as sequences of gene expression, or adaptive plasticity occurring at different times in different parts of the developing system.

Development is underpinned by two processes. *Proactivity* captures the idea that the child is an active agent in their development. The emergence of more complex representations is influenced by the child's interactions with their environment. The child selects information from the environment, rather than being a passive recipient. *Progressive specialisation* captures the idea that the events that the child experiences constrain how the child adapts to them, which also impacts on how the child adapts to future events (Piaget, 1954). At the brain level, neural systems become more specialised with development, tuning their function to particular domains depending on experience. For example, within vision, dedicated systems for face recognition and written word recognition are experience-dependent specialisations of an initially more general object recognition system. Some neural circuits, once wired, may be hard to alter, either as a result of reduced intrinsic plasticity or learning-driven stabilisation of the system (Thomas & Johnson, 2008).

These mechanisms and processes result in a developmental trajectory that at each point in time is determined by the immediate demands of the environment instead of necessarily converging towards an adult goal state. This local adaptation can often be achieved by small adaptations of the existing mental representations, resulting in *partial representations*, (e.g., for objects), that are fragmented and distributed across a range of brain regions. A given behaviour in respect of the object or situation would not require activation of all

aspects of the representation, only those that are task or situation relevant. Therefore neuroconstructivism posits that mental representations of concepts are not necessarily, nor indeed often, *full* representations (as full knowledge of some object or event is rarely possible). Instead, representations are distributed, and partial. Neural elaboration thus leads to the emergence of more complex, partial representations.

Neuroconstructivism views development as an adaptation to multiple interacting constraints, and individual differences in development as the result of variation in the degree to which constraints are present for a given child. Such influences may act to enhance and enrich development or to restrain. *Developmental constraints* are identified in terms of genes, encellment, embrainment, embodiment, and ensocialment.

At the level of *genes*, genetic activity is viewed as exerting a strong influence on development. This is not according to the more traditional view in which genes operate in a direct, cause-effect way on developmental outcomes. Rather, within the context of activity that occurs at the genetic level, neuroconstructivism sees development as dependent on multiple bi-directional interactions between genes, behaviour and the environment. Accordingly, neuroconstructivism emphasises probabilistic epigenesis (Gottlieb, 2007) – the likelihood, and extent to which genes are active in the expression of protein release is as a result of internal and external signals.

Encellment refers to the idea that at the neural level, the development of cells is influenced by their interactions with other cells in their environment. These cells develop to form networks through progressive specialisation, both as a consequence of spontaneous, internally generated neural activity, and as a

result of feedback following external behaviour. At the cellular level, neural activation leads to change, or elaboration to the underlying neural networks, and elaboration of these same networks offers the capacity for supporting the emergence of progressively more complex mental representations. In this context, experience alters the neural networks that support representations of the experience.

Embrainment refers to the idea that the development of the brain is constrained by the development of brain regions. In contrast to modular views of development, neuroconstructivism stresses the interactive nature of brain regions during development. Brain regions become progressively more tuned in their functions as a result of experience-dependent processes. Different brain regions become more strongly connected to each other in reflection of their history of being co-activated to deliver behaviours that are important and frequent for the child.

Neuroconstructivism views the body as an important constraint (in some instances a filter) for the developing brain, as it is through the body that the child is proactive in exploring their environment. This is the constraint of *embodiment*. For example, our sensory organs highly constrain the representations that are possible (e.g., humans do not have the apparatus to see the full spectrum of light). Some constraints persist but other constraints change over development. For instance, during the first few months of life human infants are particularly limited in their visual acuity and motor control. This constraint early in development restricts the experiences that are possible for the infant, thus limiting changes in representational complexity. As young children develop greater mobility, the range of experiences they are capable of having increases.

The child's increasing interactions with their environment in turn leads to progressive elaboration at the neural level and as a result affords greater complexity in their representations of the world around them.

The constraint of *ensocialment* refers to the idea that social experiences play an important role in shaping the development of the embodied mind. The type and quality of experiences that the child receives and information that he or she seeks out from the social environment will constrain the emergence of neural elaboration. Such experiences are initially with caregivers but as the child gets older, interactions between the child and their peers are an increasingly important social constraint.

Lastly, neuroconstructivism emphasises that developmental events in the brain must be construed within the wider framework of evolutionary developmental biology. An adaptive framework informs the functions that will be established during brain development. What has evolution designed the system to do? What are the neural constraints fashioned into the structure of the brain that allow the individual to achieve that goal when the child is raised in a normal environment? With respect to, the important domain of education, how can such constraints respond to environments that are evolutionary novel, cultural inventions such as literacy and numeracy?

This is a brief characterisation of the concepts at the heart of neuroconstructivism, stated at a fairly abstract level, but their original derivation was with respect to concrete examples of developmental phenomena (Mareschal et al., 2007; Sirois et al., 2008; Westermann et al., 2007). The interested reader can follow these arguments as they unfold in the concrete example domains of

early visual perception, infant habituation, object recognition, phonological development, and the role of face recognition in social cognition.

Consistency between levels of description and its impact on theories of cognition

A central aspect of the neuroconstructivist approach is its commitment that theories of cognition must be consistent with theories of brain function, and in turn, theories of cognitive development with those of functional brain development. This does not make the framework reductionist, and it is therefore important to clarify why not. Again, in this regard, the neuroconstructivist approach can be seen as responding to the prevailing theoretical approaches of the 1980s and 1990s. Traditional theories of cognition were developed based on behavioural studies and task analyses, independent of a consideration of how cognition would be implemented in the brain. Partly, it because at the time, less was known about the way in which the brain processes information. But partly, this was because cognitive psychology was influenced by the computational metaphor of mind. This metaphor stated that the mind could be viewed as a desktop (or von Neumann) computer, in which a distinction is made between the software a computer is running and the hardware on which the software is implemented. Software can be implemented on many different types of hardware (so long as they are 'Turing equivalent'), motivating the view that software can be considered independently of hardware. Under the traditional computer metaphor of mind, it is the job of the psychologist to characterise the software that the brain is running, and the job of the brain scientist to characterise how that software is implemented in the substrate of the brain.

This view was made more explicit by the vision scientist David Marr (1982), who distinguished between three levels of description within computation. The *computational level* specifies the problem that the cognitive system faces in any given domain, without saying how the problem is to be solved. The *algorithmic level* specifies the way in which the problem can be solved. The *implementational level* identifies the physical substrate or mechanism by which the computation will be performed. Each level was viewed as a realisation of the level before it. Theories of cognition concern themselves with the algorithmic level. Marr argued that a process can be described and analysed independently on these three different levels. Cognition, in other words, can be analysed independent of its implementation in the brain.

There are two flaws in this argument. First, over time, the structure of the brain is influenced by its activity. That is, the changing brain constrains the possible mental representations (neural activation patterns), but at the same time through the mechanisms of experience-dependent brain development, neural activity itself changes the underlying brain structures. Thus far from being independent, the software of the mind changes the hardware of the brain. Second, although mathematical arguments exist to show that the same software can be implemented on different hardware (so long as the software involves computable functions and the hardware is Turing equivalent), in practice, the properties of the hardware make certain software easier to run in real time. Given that the essence of cognition is that it must be adaptive (that is, it must deliver behaviour quickly enough to keep the individual alive and deliver its goals), it is likely that the software of the mind is of a form that is easy and quick to implement on the substrate of the brain. What this suggests is that cognitive

theory, so long influenced by the (desktop) computational metaphor of the mind, needs to be refashioned to be consistent with the types of computation that the brain finds easy to implement (Thomas & McClelland, 2008). This refashioning is slowly taking place, and it is at the heart of the enterprise of neuroconstructivism as a theory of cognitive development.

To highlight what this refashioning might entail, we must first briefly describe the implementation constraints of a desktop computer. What computations does a desktop computer find easy to do? Such computers are equally able to process any content with the same abstract structure (and hence, ideal for processing logic!). In these systems, abstraction is easy. Computation relies on domain-general processing mechanisms (the central processing unit, or CPU, carries out the computations on all types of content). The movement of information is fast and easy, between long-term memory (the hard disk), working memory (random access memory or RAM) and CPU. The power of the computer is limited by the speed of the CPU, the speed of information transfer, and the size of the working memory and hard disk. The intelligence of the system is limited by the software, if time pressure is not important (that is, new software will run on an old computer, but very slowly!)

What do the implementation constraints of the brain look like? We don't yet have a complete answer, but some themes are emerging. Crucially, knowledge is part of the structure of the system: it is encoded in the strength of the connections between neurons. As such, knowledge cannot be easily moved around the system. The system cannot easily perform abstraction. Instead computational circuits are content-specific and, indeed, the brain prefers to retain information in sensori-motor codes. Processing itself is context-sensitive,

similarity based, frequency sensitive, and predictive. There are no domain-general processing mechanisms, instead mechanisms are domain-specific. Information processing is intrinsically local, and global control of processes must be implemented via separate bespoke systems (for example, integrative systems with widespread connectivity, or neurotransmitter systems with diffuse spatial influence). Plasticity is modulated by current organismic goals (emotions), and resources for plasticity reduce with age. All of these implementation constraints contrast with those of the desktop computer. (See O'Reilly, Hazy & Herd, in press; O'Reilly, Herd & Pauli, 2010, for further recent views of the brain's cognitive architecture; Price & Friston, 2005, for arguments that cognitive constructs need to be refashioned to fit with the constraints of brain mechanisms).

Let us consider one of these implementation constraints in more detail (Thomas & McClelland, 2008). If we start from the premise that knowledge will be very difficult to move about in our information processing system because it is built into the structure of the brain, what kind of cognitive architecture do we end up with? There are four main themes.

First, we need to distinguish between two different ways in which knowledge can be encoded: active and latent representations (Munakata & McClelland, 2003). Latent knowledge corresponds to the information stored in the connection weights from accumulated experience. By contrast, active knowledge is information contained in the current activation states of the system. Clearly, the two are related because the activation states are constrained by the connection weights. But, particularly in recurrent networks with cycling activation, there can be subtle differences. Active states contain a trace of recent

events (how things are at the moment), whereas latent knowledge represents a history of experience (how things tend to be). Differences in the ability to maintain the active states (e.g., in the strength of recurrent circuits) can produce errors in behaviour where the system lapses into more typical ways of behaving (Munakata, 1998; Morton & Munakata, 2002).

Second, if information does need to be moved around the system, for example, from a more instance-based (episodic) system to a more general (semantic) system, this will require special structures and special (potentially time-consuming) processes. Thus McClelland, McNaughton, and O'Reilly (1995) proposed a dialogue between separate stores in the hippocampus and neocortex to gradually transfer knowledge from episodic to semantic memory.

Third, information will be processed in the same substrate where it is stored. Therefore, long-term memories will be active structures and will perform computations on content. An external strategic control system plays the role of differentially activating the knowledge in this long-term system that is relevant to the current context and goals. In anatomical terms, this distinction broadly corresponds to frontal/anterior (strategic control) and posterior (long-term) cortex. In computational terms, the control system has properties that are domain-specific to that role.

The design means, somewhat counter intuitively, that the control system has no content. Rather, the control system contains placeholders that serve to activate different regions of the long-term system. The control system may contain plans (sequences of placeholders) and it may be involved in learning abstract concepts (using a placeholder to temporarily co-activate previously unrelated portions of long-term knowledge while Hebbian learning builds an

association between them); but it does not contain content in the sense of a domain-general working memory. The study of frontal systems then becomes an exploration of the activation dynamics of these placeholders and their involvement in learning (see, e.g., work by Davelaar & Usher, 2002; Usher & McClelland, 2001).

Fourth, the focus on brain implementation constraints changes how we should think about the very concept of domain generality in processing systems. It is unlikely that there are any domain-general processing systems in the brain that serve as a "Jack-of- all-trades," that is, that can shift between representing the content of multiple domains. However, there may be domain-general systems that are involved in modulating many disparate processes without taking on the content of those systems, what we might call a system with "a finger in every pie." Meanwhile, short-term or working memory (as exemplified by the active representations contained in the recurrent loop of a network) is likely to exist as a devolved panoply of discrete systems, each with its own content-specific loop.

In sum, in its focus on the brain, neuroconstructivism is not reductionist but has the aim of altering cognitive theory. Cognitive theory will need to shift in emphasis from domain-general mechanisms (such as working memory) to domain-specific mechanisms, and from abstraction to sensori-motor codes as the preferred form of representation. A key dimension of how cognition operates will be the activation (inhibition) of task relevant (irrelevant) domain-specific structures according to context and goals. Developmental theory, in turn, will entail characterising the emergence of these domain-specific systems, the tuning of the dynamics of control, and changes in the influence of context and goals.

Typical and atypical development

As we have seen, neuroconstructivism focuses on the constraints that operate at multiple levels to shape trajectories of development. Variations in those constraints can lead to altered trajectories, explaining individual differences and atypical development. For the neuroconstructivist, developmental disorders in particular serve as an illustration of the way in which constraints at the genetic, neural, physical and social levels of description operate to shape cognitive development. And therefore, the study of such disorders is crucial to shed light on the nature of these constraints. Perhaps the most informative will be cases where cognitive profiles are uneven, with particular patterns of strengths and weaknesses. Where verbal and non-verbal abilities develop out of step, or there is an apparently differential impairment in the development of skills like reading or face recognition despite exposure to a normal environment, the atypical constraints must in some way be relevant to some or all of the cognitive processes underlying these skills.

The view that atypical development can, like typical development, be characterised as an adaptation to multiple interacting constraints that shapes the trajectory stands in contrast to theories that assume disorders arise from isolated failures of particular functional modules to develop. Modular accounts of developmental disorders illustrate the kind of componential, unidirectional theories that originally triggered the emergence of neuroconstructivism (Karmiloff-Smith, 1998, 2009; Thomas & Karmiloff-Smith, 2002; Thomas, Purser & Richardson, 2013). Modular explanations were characteristic of early investigations of several disorders. For example, autism was initially viewed in

terms of the failure of an innate, dedicated theory-of-mind module to develop (Frith, Morton, & Leslie, 1991); and specific language impairment in terms of selective damage to a genetically pre-specified syntactic module (van der Lely, 2005).

However, there are good reasons to think development is key in producing atypical cognitive profiles, because these profiles do not necessarily retain a consistent shape across development. For example, Paterson, Brown, Gsödl, Johnson, and Karmiloff-Smith (1999) explored the language and number abilities of toddlers with the genetic disorders of Down syndrome and Williams syndrome, both of which cause characteristic patterns of cognitive strengths and weaknesses along with learning disability. These authors found a different relative pattern of cognitive strengths and weaknesses to that observed in adults with the same disorders. Their findings that the profile in early childhood was not a miniature version of the adult profile, thus implicating the developmental process as a contributory factor. That is, the nature of the uneven cognitive profile depended on the age at which it was measured.

Several of the core ideas of neuroconstructivism are emphasised by the study of atypical development (Westermann, Thomas & Karmiloff-Smith, 2010). For example, in some cases localisation and specialisation of cortical areas appear atypical. Adults with Williams syndrome exhibit face recognition skills in the normal range but electrophysiological studies examining event-related potentials revealed different neural activity compared to typical controls (Grice et al., 2001). Neuroimaging data have suggested differences in the constraints of chronotopy, in terms of the changes in connectivity (and associated plasticity) over time in disorders such as autism and Down syndrome (e.g., Becker et al.,

1986; Chugani et al., 1999). Differences in input encoding have been proposed to have cascading effects on the context in which other cognitive abilities are acquired, for example in autism, specific language impairment, and dyslexia. It is possible that in these disorders, the consequence of atypical similarity structure in the input representations results in a processing deficit much higher up in a hierarchy of representational systems. Differences in embodiment may also impact on the trajectory of development. For example, Sieratzki and Woll (1998) proposed that in children with spinal muscular atrophy—a disorder that reduces early mobility—language development might be accelerated as a compensatory way for the young child to control his/her environment. Lastly, an atypical child co-specifies an atypical social environment, for example, in the expectations and reactions of parents and peers, which has also been observed to influence these children's development (e.g., Cardoso-Martins, Mervis & Mervis, 1985).

Of course, when we place an emphasis on development as a trajectory, and atypical development as an atypically constrained trajectory, it becomes increasingly important on the one hand to have a rich vocabulary with which to describe different trajectories, and on the other, to be able to formulate and test precise hypotheses concerning what is different about the constraints and mechanisms of change in a given disorder. Respectively, this has led to advances in methodological techniques and computational modelling of development.

Methodologies to analyse developmental trajectories

A focus on change over time has led to new methods that allow us to describe, analyse, and compare the developmental trajectories followed by different cognitive systems. This is especially the case when we wish to study variations in

the trajectories found in typically or atypically developing children. The cognitive profile associated with any developmental disorder does not emerge full-blown at birth but develops gradually and sometimes in transformative ways with age. This can only be studied by following atypical profiles over time. New methods have been designed for just this purpose (e.g., Knowland & Thomas, 2011; Thomas et al., 2009; Westermann, Thomas & Karmiloff-Smith, 2010).

The use of trajectories to study cognitive variation contrasts with methods that compare static snapshots of development to assess differences (e.g., Hodapp, Burack, & Zigler, 1990; Leonard, 1998). For example, when researchers investigate behavioural deficits in individuals with developmental disorders, a common methodology is to use a matching approach. The research asks, does the disorder group show behaviour appropriate for its mean age? To answer this question, the disorder group is matched with two separate typically developing control groups, one match based on chronological age and a second match based on mental age, (the latter derived from a standardised test relevant to the cognitive domain). If the disorder group shows an impairment compared with the chronological-age-matched group but not with the mental-age-matched group, individuals with the disorder are considered to exhibit 'developmental delay' on this ability. If, however, the disorder group shows an impairment compared with both control groups, then the disorder group is considered to exhibit developmental 'deviance' or 'atypicality'.

This matching approach ultimately dispenses with age as an explicit factor by virtue of its design. Necessarily, this restricts its ability to describe change over developmental time. An alternative analytical methodology is based on the idea of trajectories or growth models (Annaz et al., 2009; Jarrold & Brock,

2004; Rice, 2004; Singer Harris, Bellugi, Bates, Jones, & Rossen, 1997; Thomas et al., 2001, 2006; Thomas, Purser & van Herwegen, 2011). In this alternative approach, the objective is first to construct a function linking performance with age on a specific experimental task and then to assess whether this function differs between the typically developing group and the disorder group. The use of trajectories in the study of development has its origin in growth curve modelling (see, e.g., Chapman, Hesketh, & Kistler, 2002; Rice, 2004; Rice et al., 2005; Singer Harris et al., 1997; Thelen & Smith, 1994; van Geert, 1991) and in the wider consideration of the shape of change in development (Elman et al., 1996; Karmiloff-Smith, 1998).

The aim of the trajectory approach within the study of atypical development is twofold. First, it seeks to construct a function linking performance with age for a specific experimental task. Separate functions are constructed for the typically developing group and for the disorder group, and the functions are then compared. Second, it aims to shed light on the causal interactions between cognitive components across development. To do so, it establishes the developmental relations between different experimental tasks, assessing the extent to which performance on one task predicts performance on another task over time. Once more, the developmental relations found in the disorder group can be compared against those observed in a typically developing group. Trajectories may be constructed in three ways: (1) they may be constructed on the basis of data collected at a single point in time, in a cross-sectional sample of individuals varying in age and/or ability; (2) they may be constructed on the basis of data collected at multiple points in time, tracing longitudinally changes in individuals usually of the same age; or (3) they may

combine both methods, with individuals who vary in age followed over two or more measurement points. In most cases, analyses employ linear or non-linear regression methods, for example comparing the gradients and intercepts of best-fit regression lines between groups (Thomas et al., 2009).

The trajectory methodology is not without its drawbacks. It makes several demands of behavioural measures. It relies on the use of experimental tasks that yield sensitivity across the age and ability range of the children under study; that avoid floor and ceiling effects where possible; and that have conceptual coherence with the domain under investigation. Conceptual coherence means that the behaviour must tap the same underlying cognitive processes at different ages and ability levels. It is worth noting that the first of these criteria, task sensitivity across a wide age range, may be one of the hardest to fulfil. This is particularly the case in domains that are characterised by early development, where measures may exhibit ceiling effects at a point when other domains are still showing marked behavioural change over time. In the domain of language, for example, speech development reaches ceiling levels of accuracy much earlier than vocabulary or syntax. This can compromise our ability to assess developmental relations between abilities that plateau at different ages. Currently, one significant challenge facing the study of cognitive development is to calibrate measurement systems to afford age-level sensitivity while at the same time retaining conceptual coherence over large spans of time. There are few theoretically interesting behavioural measures that tap development over a very wide age range. Sometimes researchers are tempted to rely on subtests from standardised test batteries (IQ tests), since these are often constructed with a wide age range in mind but, despite being psychometrically sound measures,

standardised tests are frequently very blunt measures of the development of individual cognitive processes and do not guarantee conceptual coherence.

One alternative is to appeal to more sensitive dependent measures such as reaction times. Although reaction times can be noisy, they continue to exhibit developmental change when accuracy levels are at ceiling. A second alternative is to use implicit rather than explicit measures of performance to assess underlying cognitive processes. Implicit measures are online, time-sensitive assessments of behaviour in which the participants are usually unaware of the experimental variables under manipulation, such as the frequency of words in a speeded language comprehension task (Karmiloff-Smith et al., 1998).

Whether or not the neuroconstructivist framework is the appropriate one to conceptualise cognitive variation, the use of trajectories as empirical primitives is a descriptively powerful approach, since it can distinguish between multiple ways that development can differ. For example, trajectories may differ in their onset, in their rate, in their shape, in their monotonicity – whether they consistently increase over time or go up and down – and the point and level at which performance asymptotes. Figure 1 demonstrates the richer vocabulary available to describe the notion of 'delayed' development when a trajectories approach is adopted. An accurate and detailed characterisation of empirical patterns of change is a necessary precursor to formulating causal accounts of developmental variations.

<Insert Figure 1 around here>

Computational modelling as a key methodology supporting neuroconstructivism

We have seen how the neuroconstructivist view shifts the emphasis from compiling a series of abilities present at different ages toward the dynamics of the development. Because neuroconstructivism aims to understand how development is influenced via bi-directional interactions between constraints across multiple levels (i.e., from the expression of genes on protein release, to changes in connectivity between neurons, differences in intra and interregional interactions, and of changes to all these as a result of physical and social interactions), a strong emphasis is placed on understanding how these may be realised at the neurocomputational level. The combination of construing development as the outcome of local changes in response to multiple interacting constraints, and linking neural and cognitive development produces an approach that lends itself to specification through computational modelling. One of the virtues of implemented models is that they allow us to simulate the consequences of changes to a complex system in which behaviour is generated by the on-going interaction of many components. Such outcomes are not always predictable using analytical means, and are therefore called 'emergent properties'.

Two of the most influential computational or formal approaches in respect of modelling cognitive development are connectionist modelling and dynamical systems theory (Elman, Bates, Johnson, Karmiloff- Smith, Parisi, & Plunkett, 1996; Quinlan, 2003; Mareschal, Sirois, Westermann, & Johnson, 2007; Spencer, Thomas, & McClelland, 2009). *Connectionist models* are computational systems loosely based on the principles of neural information processing. As

such they are positioned at a level of description above biological neural networks, but aim to explain behaviour on the basis of the same style of computations as the brain (see Thomas & McClelland, 2008, for discussion of their relation to recent Bayesian approaches to cognition). Connectionist models have the ability to learn from data by progressively altering the strengths of the connections in their networks, and are therefore relevant for explaining the mechanisms underlying behavioural change in cognitive development. In *dynamical systems theory*, individual growth functions are specified to stipulate the trajectory of development in components of the system, while interactions between multiple components allow for the consideration of complex dynamics of change over time. In this section, we consider examples from each methodology that have been use to study the causes of variations in developmental trajectories within the neuroconstructivist approach.

Connectionist or artificial neural network models are well suited to study development within the neuroconstructivist framework, because the learning trajectory in a model is the outcome of local adaptations to interacting constraints. Changes in behaviour are the result of experience-dependent alterations in the network that result from its interaction with a structured learning environment. In contrast to child development, however, in a model these constraints are precisely known and can be manipulated by the modeller to observe changes to the developmental trajectory and the learning outcome. A model has intrinsic constraints such as the number of artificial neurons, the pattern of connections between units within a network and the way in which external or environmental inputs are encoded for processing. Other constraints include those pertaining to plasticity, such as the function and parameters of the

connection weight change rule, and to the learning environment, such as the type, frequency and order of the stimuli to which the model is exposed. Insights from developmental cognitive neuroscience have also been incorporated into connectionist modelling by allowing for experience-dependent structural development (that is, changes in the architecture of the neural networks), and the gradual integration of network sub-components (Westermann, Sirois, Shultz, & Mareschal, 2006; Mareschal, Sirois, Westermann & Johnson, 2007), adding further constraints to the developmental model.

One recent computational approach has been to simulate developmental processes in large populations of children and to include intrinsic (neurocomputational) and extrinsic (environmental) factors that interact to produce variability in developmental trajectories across the whole population. This approach was employed to consider variations in trajectories of language development. It provides a framework to consider the origins of population-wide individual differences. For example, the bottom tail of a normal distribution of performance can be defined as 'delayed', but genetic mutations can also be introduced as a new manipulation to the learning properties of the system occurring in a subset of individuals. The population modelling approach has been applied to consider the causes of delay in typical populations (Thomas & Knowland, 2014), as well as variability in developmental disorders (Thomas, Knowland & Karmiloff-Smith, 2011a, 2011b), and environmental effects on language acquisition (Thomas, Forrester & Ronald, 2013). Such mechanistic frameworks are necessary to move the concept of developmental variation beyond a description of observed behavioural trajectories to an explanation of their origins.

Importantly, such an approach encourages us to view individual differences as variations in developmental trajectories. Traditionally, there has been a separation between the study of the development of the 'average' child, linking cognition to increasing age, and the study of individual differences between children or adults at a given age, taking cross-sections of the population (see, e.g., Gross (2010) for a recent introductory psychology text where development and individual differences are considered in separate chapters). Figure 2 depicts data from a large number of connectionist networks simulating acquisition of a notional cognitive domain in a population of children. It demonstrates how the development of the 'average' child (Fig.2a) in fact summarises a host of different trajectories (Fig.2b). While variation can be studied by cross-sections taken at different ages (Fig.2c), and indeed neurocomputational parameters can be identified that predict individual differences in these cross-sectional analyses (Fig.2d), the cross-sections are a single and potentially misleading perspective. There are no mechanisms for individual differences separate from the developmental process. There are just variations in trajectories reflecting a dynamic developmental processing taking place under differing constraints. Ultimately, the neuroconstructivist approach may lead to dissolution of the artificial divide between development and individual differences.

<Insert Figure 2 around here>

One advantage of modelling is that it can lead to novel hypotheses. For example, one model utilised the population-based approach to generate a new

hypothesis that autism is caused by disruptions in connectivity occurring during synaptic pruning (Thomas, Knowland & Karmiloff-Smith, 2011a). During development, the brain initially produces exuberant connectivity, which is subsequently pruned back in childhood. This gives the brain greater plasticity in early development, to adapt to the environment in which it finds itself, while saving on metabolic resources later in development. However, if the pruning process is too aggressive, rather than just removing spare computational resources, it can comprise the neurocomputational properties of the system or even lead to regression in behaviour.

Notably, Thomas, Knowland and Karmiloff-Smith (2011a) found that the cause of the disorder in their networks (over-aggressive synaptic pruning) interacted with other dimensions that varied in the general population, such as the amount of computational resources, the rate of learning, and the richness of the learning environment to which the individual was exposed. These risk and protective factors led to a probabilistic relationship between the (in the model, known) cause of the disorder and its manifestation in behavioural deficits. Moreover, the authors demonstrated how a direct cause of one disorder (e.g., slow development) could be a risk factor for another (e.g., slow development makes the effects of aggressive synaptic pruning worse). This would explain why there should be shared causal factors (such as gene variants) between different disorders: the shared factor indexes the cause of one disorder and the elevated risk (but not direct cause) or another. Bishop (2006) recently advocated that researchers move to an explanatory framework of developmental disorders based on risk and protective factors, rather than necessary and sufficient conditions. The population modelling approach is consonant with this shift to

viewing causal factors as probabilistic against a background of variability, as well as the view that the developmental process itself is a key part of the explanation of developmental disorders (Karmiloff-Smith, 1998).

Turning to dynamical systems modelling, one theoretical question to which models have been applied is the origin of uneven cognitive profiles within development. Whilst uneven profiles of cognitive abilities are often found during childhood, current debates concern precisely how deficits emerge and the true extent of specificity of a deficit in a developmental disorder. For acquired deficits following brain damage, since there is some localisation of brain function, it makes sense that uneven cognitive profiles can occur due to focal damage to particular components of the cognitive system. However, within a developmental framework, the components of the cognitive system interact with each other over time. One might therefore expect early damage to individual components to lead either to deficit spread (as other components relying on the damaged component do not receive the developmental input which they require) or to compensation (as other components adapt to provide an alternative means to deliver the required function, perhaps altering their typical functioning). Either would reduce the specificity of observed cognitive deficits. Whether deficit spread or compensation would be predicted to occur depends, presumably, on the computational properties of each component and the overall cognitive architecture.

Baughman and Thomas (2008) used dynamical systems modelling to investigate this question (see also Thomas, Baughman, Karaminis & Addyman, 2012). They simulated development in different types of cognitive architecture that were constructed from multiple interacting components. Development in

each component was specified by a nonlinear growth function specified by three parameters, onset, rate, and final level of functioning (the growth curve asymptote). Figure 3 illustrates a developmental growth curve and the types of variation in developmental trajectory that can be produced by changes to each of these parameters within a single component. The cognitive architecture was specified by a matrix summarising interactions between the set of components over developmental time. The cognitive architectures compared distributed, modular, hemispheric, central processor, and hierarchical designs. Baughman and Thomas examined how early damage to a single component led to consequent impairments across developmental time. In some cases, the initial damage was followed by compensation from surrounding components. In other cases, causal interactions between components across development caused the impairment to spread throughout the system. Several factors determined the exact pattern, including the architecture, the location of the early damage within that architecture with respect to connectivity, and the nature of the initial impairment. In particular, this formal model demonstrated three results. The density of connectivity at the point of damage, as well as positioning in hierarchical systems, were influential in determining both spread and compensation effects following an initially more restricted deficit. Second, the number of processes that interacted to drive development in a given cognitive component affected compensation but not spread of the deficit. Third, damage to growth curve asymptotes (the developmental equivalent of the capacity of a cognitive process) was more serious for outcomes than damage to its rate (equivalent to the plasticity of the component).

Overall, the dynamical systems model highlighted the importance of understanding causal connectivity in explaining the origin of uneven cognitive profiles. Computational simulations of this kind are required to reconcile views of the apparently specific nature of some behavioural impairments, in disorders such as developmental dyslexia, specific language impairment, or developmental prosopagnosia, with those views that posit the highly distributed nature of cognition.

<Insert Figure 3 around here>

Future directions

Thus far, we have seen how neuroconstructivism is a developmental theory that seeks to characterise the developmental processes as the confluence of constraints operating at multiple levels of description, and in particular seeks consistency between theories of cognitive development and functional brain development; and we have seen how neuroconstructivism has led both to methodological advances and to an increasing use of computational modelling to specify the operation of developmental mechanisms. In this section, we consider some of the challenges facing neuroconstructivism in the immediate future.

First, the requirement that cognitive theories are constrained by theories of brain function is of course contingent on having an understanding of that function, and cognitive neuroscience is still far from complete. One of the main challenges is to understand how the multiple neural networks of the brain integrate to produce its global function. Sporns (2014) recently summarised the

contribution of the mathematical approach of network science to understanding the huge amount of data now emerging from imaging studies of brain structure and function. Network science defines nodes or elements and the strength of the relationships between them. It can be used to analyse either structural connectivity data from brain imaging studies, or functional data such as the correlated activity between different brain regions. This descriptive analytic approach has pointed towards the importance of distinguishing between network hubs – highly connected nodes that are centrally placed in the network's global topology or layout – and network communities or modules, which are regions that exhibit coherent changes in activation that in turn point to high internal functional connectivity. Such coherent fluctuations in regional functional activations are in fact often measured when the participant is 'at rest' in the brain scanner rather than performing a prescribed task, and so are referred to as 'resting-state' networks.

According to Sporns (2014), recent findings emerging from this literature include that (1) resting-state networks strongly resemble the sets of regions that are co-activated across a wide range of cognitive and behavioural tasks. This is consistent with idea that resting-state networks reflect a history of co-activation and common recruitment during task-related activity. In other words, in an echo of the micro-phenomenon of Hebbian learning at the neural level, large-scale brain networks that frequently 'fire together' tend to 'wire together'; (2) unimodal cortices, such as visual or motor cortex, tend to have consistent module membership early or late in learning of a given skill, while the module membership of multimodal association areas is more likely to shift between modules. This suggests a mode of functional organisation that combines a stable

unimodal core with a more variable multimodal periphery. Such an organisation would be a requisite for modulating task and learning dependent interactions among domain-specific network modules; (3) network hubs, with a high degree of connectivity, tend to be highly connected to each other. This suggests that integration between regions in the brain is subserved by structures dedicated for this role. It chimes with the implementation view that we saw earlier: neural processing requires domain-specific mechanisms, even where the domain is integration, an apparently general function.

The methods of network science encourage optimism that the wealth of data emerging from brain imaging can be leveraged into a deeper understanding of the principles of global brain function. However, for a neuroconstructivist, there are still significant limitations in current findings. Results mainly stem from the study of adults, and the methods are still to be robustly applied to the study of development, so that we may understand how network hubs and modules alter over development. As we have seen, neuroconstructivism argues for the progressive specialisation of representations.

With respect to atypical development, there is an additional challenge of understanding which differences in the apparent structure or function of the brain in a developmental disorder have information-processing consequences for the development of cognition. The difficulty here is that while atypical functioning at the cognitive level seems to correlate with atypical activation patterns in the brain, atypical activation patterns in the brain do not guarantee atypical cognitive functioning. For example, 2–5 per cent of typically developing individuals have right-lateralised language systems (Bates & Roe, 2001). Yet these individuals are not marked out as having atypical cognitive-level language

systems. Women can demonstrate more bilateral patterns of brain activation in language tasks than men (e.g., Shaywitz et al., 1995). Indeed, sex steroid hormones have been shown to modulate a wide range of brain processes including neurogenesis, cell migration, growth of the neuronal soma, dendritic growth, differentiation and synapse formation, synapse elimination, neuronal atrophy and apoptosis, neuropeptide expression, the expression of neurotransmitter receptors and neuronal excitability (Cameron, 2001). Yet cognitive psychology does not (at present) posit qualitatively different functional structures for the language system in the two genders, let alone different overall cognitive architectures. Such differences in brain function are put down to the multiple realisability of cognitive architectures in neural structures, whereby the same cognitive level computations can be implemented in different ways in the wetware available. The negotiation between these two ideas – brain constraints that alter cognitive architecture versus multiple realisability of cognitive architectures – remains to be worked through.

With respect to computational methods, attempts to build models that incorporate constraints from multiple levels of description have much further to progress, and advances are only beginning to become possible via increases in available computational power. For example, one type of empirical data that increasingly influences researchers within cognitive development is statistical associations between levels of description, such as gene variants that correlate with individual differences in behaviour, or structural and functional properties of the brain that correlate with behaviour across individuals or within individuals over time. However, it is a significant challenge to construct causal accounts of development that span levels of description and thereby unify the

correlations by appeal to explanatory mechanism. This is particularly so for gene-behaviour associations, because so many levels of description can be specified in between (Johnston & Lickliter, 2009). Genetic effects are cellular but must be linked to behaviour via neural circuits and global brain function, and the contribution of some genetic activity to individual differences in behaviour occurs via an extended developmental process.

A recent response to this challenge is the use of *multi-scale* computational models. This approach originated in systems biology, where the availability of more powerful computers has enabled the coupling of complex models across multiple spatial and temporal scales and for multiple physical processes (Southern et al., 2008). Dammann and Follett (2011) argued that multi-scale computational models may be equally applicable to developmental cognitive neuroscience. In the context of developmental disability, they identified *in silico* approaches as complementary to *in vivo* and *in vitro* studies in teasing apart the complicated inter-relationships between etiological exposures and pathological mechanisms on developmental outcomes. Thomas, Forrester and Ronald (submitted) recently employed multi-scale computational modelling to investigate gene-behaviour associations, and in particular, the extent to which reliable associations from the low level of genes to the high level of behaviour can shed light on the causal processes that take place at the intervening cognitive level of description. Since the causal mechanisms operating at all levels were well understood in their model, Thomas et al. were able to evaluate whether cross-level associations gave an accurate picture of causal processes. They concluded that the principal ways that gene-behaviour statistical associations

could inform theories of cognitive development was with respect to the effect size, specificity, and timing of the statistical associations.

In conclusion, neuroconstructivism is a developmental model that integrates elements of Piagetian theory with the findings of modern developmental cognitive neuroscience, while resisting the temptations of reductionism that a focus on the brain sometimes involves. With the advances in neuroscientific methods, neuroconstructivism is likely to have a rich future for understanding cognitive development, if it can successfully address the challenge of integrating data from multiple levels of description.

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Figure Captions

Figure 1. The trajectory methodological approach moves beyond a simple descriptive partition between 'delay' and 'deviance' with respect to atypical development. The use of trajectories distinguishes at least seven ways that a disorder group can statistically differ from a control group in the functions that link performance and age (or mental age). The plot shows five of these in comparison to typical development: delayed onset, slower rate, delayed onset and slower rate, non-linearity, and premature asymptote. In addition, trajectories may be flat (zero gradient), or there may be no systematic relationship between age and performance in the disorder group. (y-axis is arbitrary). See Thomas et al. (2009) for further details.

Figure 2. Computational modelling results from connectionist simulations of population development. (a) Population mean development for 1000 simulated individuals; (b) individual trajectories; (c) frequency distributions showing individual differences at three time points shown on (b); (d) predictors of individual differences from a linear multiple regression model at the three time points, based on the values of neurocomputational parameters in each network and the quality of the environment with which that network interacted. The three statistical regression models each explained around 40% of the population variance in performance. Although the differing neurocomputational parameters of each network and its training environment were the known cause of the variations in the simulated developmental trajectories, 60% of the variance

remained unexplained by the linear statistical model. This was because (1) the relationships between parameters and performance were not in fact linear; (2) the computational parameters interacted with each other in their effects; and (3) there were stochastic factors, such as the initial random state of each network.

Figure 3. Computational modelling results using dynamic systems theory to specify trajectories of development. Panels show variability in trajectories that follow when deficits are applied to parameters that influence developmental growth curves within a fully distributed architecture, that is, fully connectivity between component processes. Here, deficits are applied to parameters of a single process (labelled Direct) and trajectories depict different resulting levels of spread of damage and compensation (labelled Indirect). Dashed lines represents the average typical trajectory within a population. Grey lines represent the consequence of the deficit on the trajectory of the single process, and the effect of that damage on other connected processes (i.e., spread and compensation). Respectively, these show the effects of: (a) severe reduction to the Asymptote of a single process; (b) moderate level of reduction to the rate of Growth of a single process; (c) severe reduction to the Starting state of a single process; (d) combined deficit of a severe reduction to the rate of Growth and the Starting state of a single process.

Figures

Figure 1

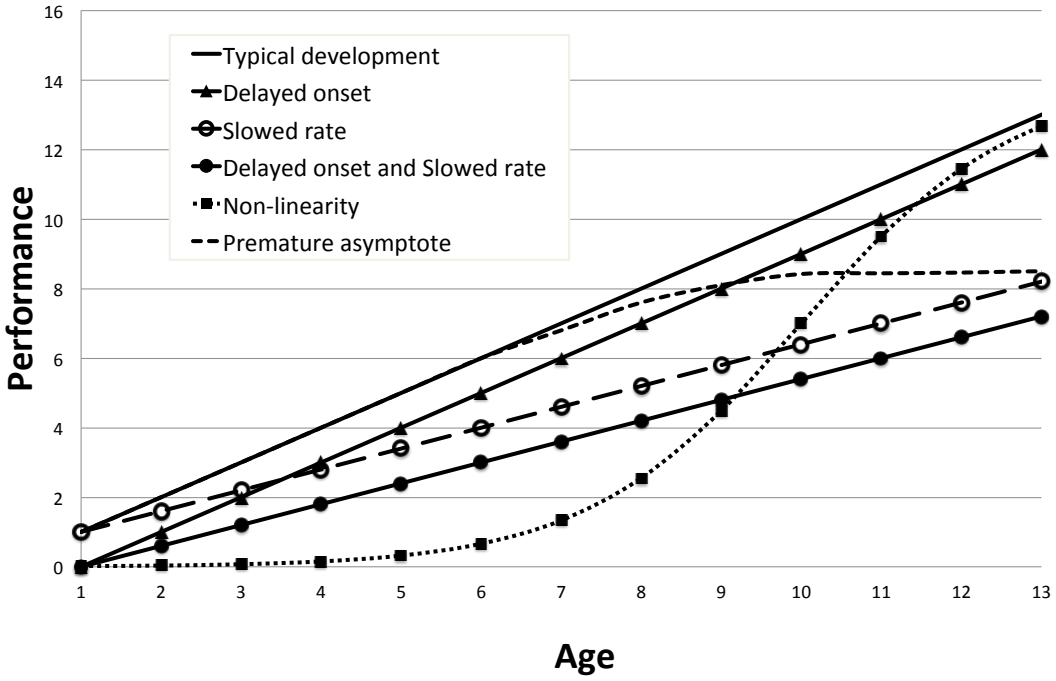
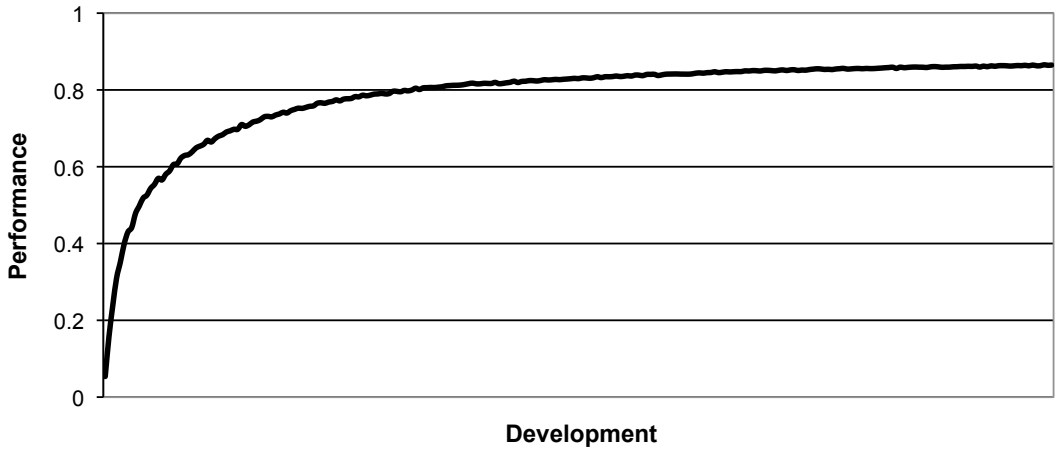
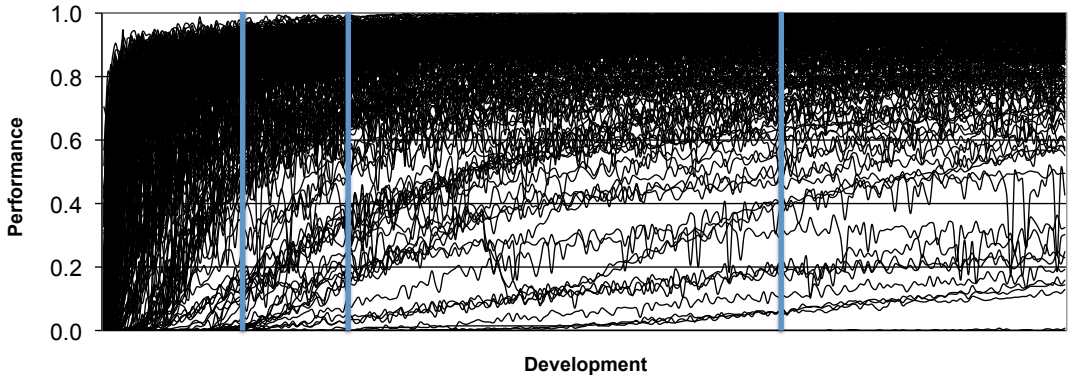


Figure 2.

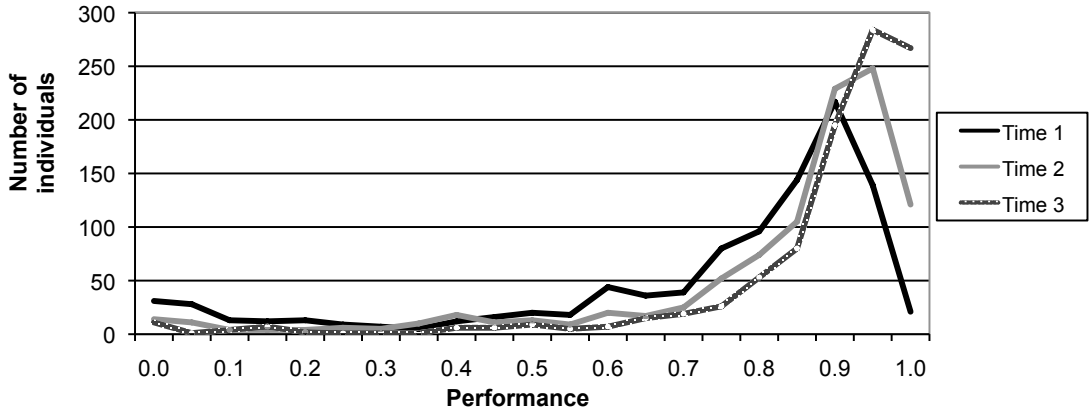
(a)


1
0.8
0.6
0.4
0.2
0
Performance
Development

(b)


1.0
0.8
0.6
0.4
0.2
0.0
Performance
Development

(c)


Number of individuals
300
250
200
150
100
50
0
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
1.0
Performance
Time 1
Time 2
Time 3

(d)

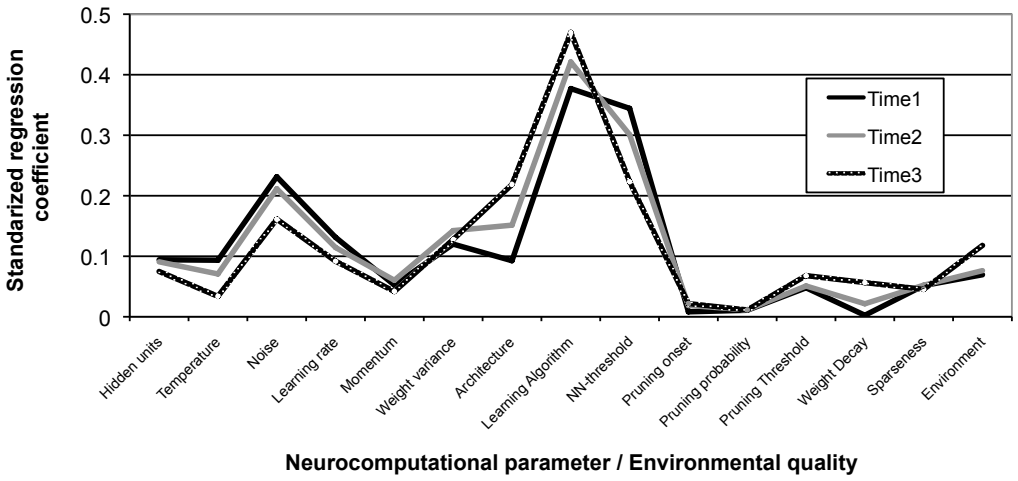
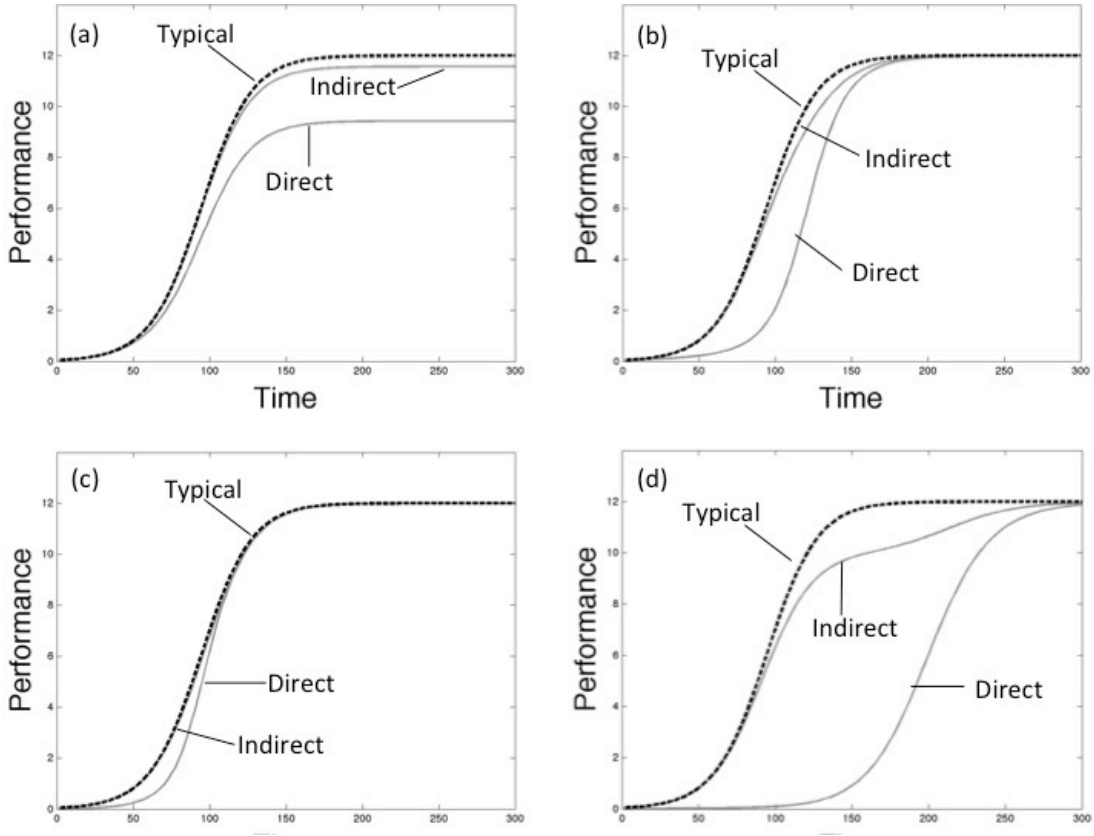

Standarized regression coefficient
0.5
0.4
0.3
0.2
0.1
0
Hidden units
Temperature
Noise
Learning rate
Momentum
Weight variance
Architecture
Learning Algorithm
NN-threshold
Pruning onset
Pruning probability
Pruning Threshold
Weight Decay
Sparseness
Environment
Time1
Time2
Time3
Neurocomputational parameter / Environmental quality

Figure 3.


(a)
Typical
Indirect
Direct
Performance
Time
(b)
Typical
Indirect
Direct
Performance
Time
(c)
Typical
Direct
Indirect
Performance
(d)
Typical
Indirect
Direct
Performance