Neuroconstructivism

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Words: (main text) ~ 9,472

To appear in: Goswami, U. (Ed). *The Handbook of Cognitive Development*, *2nd ed.* Oxford: Blackwell.

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1. Introduction

In this chapter, we outline the neuroconstructivist framework for studying cognitive development. Neuroconstructivism builds on the Piagetian view that development constitutes a progressive elaboration in the complexity of mental representations via experience-dependent processes. However, Neuroconstructivism is also informed by recent theories of functional brain development, under the view that the character of cognition will be shaped by the physical system that implements it. First, we begin by outlining the main premises of the neuroconstructivist framework. Second, we describe one of the emerging methodologies on which Neuroconstructivism relies – the modelling of development in complex neurocomputational systems. Third, we turn to consider atypical development, and the way it can shed light on the constraints shaping the typical developmental process. Fourth, we describe a new empirical methodology that has been designed to analyse the primary data on which Neuroconstructivism relies: developmental trajectories. Finally, we review recent findings on genetic influences on brain development, and indicate how these may shape our conceptions of cognition.

2. The Neuroconstructivist Framework

Perhaps surprisingly the bulk of existing research in developmental psychology is not strictly developmental at all. Instead it is concerned with static snapshots of the abilities of infants and children at different ages. For example, we know that in language development, six month old infants can discriminate between all speech sounds, but by 12 months of age they have lost the ability to discriminate between non-native sounds (Werker & Tees, 1984). In object categorization we know that 3-4 month old infants are capable of forming perceptual categories on the basis of animal

pictures, but only by 10 months are they able to encode the correlations between object features to constrain categories (Younger & Cohen, 1986). And Piaget showed that children younger than around 7 years, but not at 10 years, lack the concept of conservation, that is, they do not understand that the physical characteristics of an object or substance remain the same even when its appearance changes (Piaget, 1955). However, the perhaps biggest challenge facing developmental psychologists is to link these individual observations into a developmental trajectory and to explain the causes of developmental change that allow the child to progress from one set of abilities to another, more complex one. A recent attempt to provide such a developmental framework is Neuroconstructivism (Mareschal, Johnson, Sirois, Spratling, Thomas, & Westermann, 2007a; Westermann, Mareschal, Johnson, Sirois, Spratling, & Thomas, 2007).

The neuroconstructivist approach characterizes development as a trajectory that is shaped by multiple interacting biological and environmental constraints. The central aspect of understanding cognitive development in this framework is the explanation of how these constraints affect the development of the neural networks of the brain that give rise to progressively more complex mental representations. Brain and cognitive development are linked by characterizing mental representations as neural activation patterns that are realized in the developing neural network of the brain. By considering constraints at all levels from the gene to the social environment, Neuroconstructivism draws on, and integrates, different views of brain and cognitive development such as (1) probabilistic epigenesis which emphasizes the interactions between experience and gene expression (Gottlieb, 2007) (2) neural constructivism which focuses on the experience-dependent elaboration of neural networks in the brain (Purves, 1994; Quartz & Sejnowski, 1997), (3) the ‘interactive specialization’

view of brain development which focuses on the mutually constraining interactions of different brain regions in shaping the developing brain (Johnson, 2001), (4) embodiment views that emphasize the importance of the body in cognitive development and processing (Clark, 1999; Smith, 2005), (5) Piaget's constructivist approach to development that stresses the pro-active acquisition of knowledge by the child, and (6) approaches highlighting the role of the evolving social environment for the developing child.

The neuroconstructivist approach has in part been motivated by advances in infancy research that allow for the investigation of brain and cognitive development in parallel (Johnson, 1997; Nelson & Luciana, 2001). First, in the past fifteen years our ability to investigate the developing brain has progressed dramatically through the application of sophisticated imaging methods such as fMRI, ERP, MEG and NIRS to infancy research. Second, new experimental methods such as preferential looking, head turn paradigms and eye tracking have been developed and refined to study the abilities of even very young infants in a range of behavioural domains. Third, computational modelling has enabled the development and testing of brain-inspired models of cognitive behaviour in which the effect of changed constraints on cognitive outcomes can be investigated. And finally, great progress has been made in characterizing gene-environment interactions in development.

Acknowledging the close link between brain and cognitive development has important implications pertaining to the study of cognitive development. Perhaps most importantly, Neuroconstructivism rejects the widely accepted separation of levels of description proposed by Marr (Marr, 1982). Marr argued that a process can be described and analyzed independently on three different levels: the computational, algorithmic and implementational levels. This widely accepted approach was inspired

by the computer metaphor of the mind which separates between the 'software' of mental processes and the underlying 'hardware' of the brain, and it argued that the nature of mental processes could be studied without regard to the nature of its implementation. However, the neuroconstructivist approach is incompatible with this assumption. This is because 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. In the language of the computer metaphor, the hardware constrains the software, but the software changes the underlying hardware. This interdependency between levels makes it clear that hardware and software cannot be studied independently from one another. It also means that, despite highlighting the importance of brain development for cognitive development, Neuroconstructivism does not advocate a reductionist viewpoint in which cognitive change should be explained solely on the basis of neural adaptation. Instead, Neuroconstructivism argues for consistency between levels of description and an acknowledgement that processes described best at one level can change those at a different level and vice versa.

A second implication of the neuroconstructivist viewpoint is that development, adult processing and age-related decline can in principle be accounted for within a single framework by characterizing the variations in constraints that operate at different stages of life. Likewise, in the neuroconstructivist framework atypical development can be explained as arising from a set of altered constraints that push development off its normal track (Karmiloff-Smith, 1998). We will return to this point below.

3. Sources of Constraints in Neuroconstructivist Development

In this section we describe the different levels of constraints that shape development and we define a common set of developmental mechanisms and principles that operate across all levels.

Genes. In the past decade the view of a genetic blueprint for development has been radically changed. This traditional view postulated a one-directional chain from gene (DNA) to RNA transcription to protein structures. Development was seen as the progressive unfolding of the information in the genome. In contrast, more recent work has found many instances of gene-environment interactions, recognizing that the expression of genes is often subject to environmental and behavioural influences (Lickliter & Honeycutt, 2003; Rutter, 2007). This probabilistic epigenetic view of development (Gottlieb, 2007) emphasizes that gene expression is not strictly pre-programmed but is regulated by signals from the internal and external environment, and that development is therefore subject to bi-directional interactions between genes, neural activity, and the physical and social environments of the developing child. For example, a longitudinal study of the effect of life stress on depression (Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, McClay, Mill, Martin, Braithwaite, & Poulton, 2003) revealed that although genetic factors affected the susceptibility to depressive symptoms, this effect was modulated by stressful life experiences earlier in life. Another recent study (Wiebe, Espy, Stopp, Respass, Stewart, Jameson, Gilbert, & Huggenvik, 2009) reported interactions between genotype and prenatal exposure to smoking in preschoolers on tasks requiring executive control: those children with a particular genotype performed poorly in these tests only if they also had been exposed prenatally to tobacco. With reference to the nature-nurture debate these results

therefore suggest that development proceeds through interactions between genes and the environment that are so closely linked that an attempt to quantify either contribution makes no sense (Karmiloff-Smith, 2006). We return to epigenetic approaches to explaining atypical development below.

Encellment (Neural constructivism). The development of a neuron is constrained by its cellular environment throughout development. Even at early stages of foetal development the way in which an individual cell develops is influenced by molecular interactions with its neighbouring cells. At later stages in development, neural activity, generated spontaneously or through sensory stimulation, can affect the functional and structural development of neural networks in various ways (Quartz, 1999; Butz, Wörgötter, & van Ooyen, 2009). Neural activity can guide the outgrowth and retraction of neural axons and dendrites, leading to addition or loss of synaptic connections between neurons and to synaptic rewiring, modifying the connection patterns between neurons. These mechanisms can act rapidly with parallel progressive and regressive events (Hua & Smith, 2004). Together they lead to the experience-dependent elaboration and stabilization of functional neural networks (Quartz & Sejnowski, 1997). Evidence for the role of experience on neural development has come, among others, from studies in which rats were reared in environments of different complexities (Rosenzweig & Bennett, 1996), and this work has led to a wider research effort to identify the neural consequences of environmental enrichment (van Praag, Kempermann, & Gage, 2000; Sale, Berardi, & Maffei, 2009). In these studies it was reliably shown that the brains of rats growing up in stimulating environments with other rats, toys and opportunities for physical exercise, had markedly increased cortical weight and thickness, more dendritic arborisation, and a

higher number and size of synapses. Furthermore, these animals showed increased hippocampal synaptogenesis and less age-related cell death. These structural changes went hand in hand with increased cognitive function, improved learning and memory, and reduced age-related cognitive decline. Some of the observed changes were associated with altered gene expression, pointing further towards a role of gene-environment interactions in experience-dependent brain development.

From a neuroconstructivist perspective these mechanisms are important because they indicate that experiences can alter the neural networks that are in place to support the processing of these experiences. The nature of mental representations, realized through neural activation patterns, is constrained by the structure of the neural networks supporting them. The fact that these activation patterns can in turn themselves modulate the structure of these networks provides a mechanism by which progressively more complex representations can be built onto simpler ones by the gradual adaptation of the constraints (neural structures) to the experiences (neural activation patterns).

Embrainment (Interactive specialization). As individual neurons are linked to other neurons affecting their development, so entire functional brain regions develop in a network with other regions through a process of interactive specialization (Johnson, 2001). This view of brain development is different from the more traditional modular view which focuses on the development of encapsulated functional brain regions in isolation. It is supported by functional neuroimaging research showing that the functional specialization of brain regions is highly context sensitive and depends on interactions with other brain regions through feedback processes and top-down modulation (Friston & Price, 2001). This process becomes most evident in brain

organization in people who lack one sensory modality. For example, in individuals who have been blind from an early age, the brain area that is the primary visual cortex in seeing people is recruited for the tactile modality instead, i.e., Braille reading (Sadato, Pascual-Leone, Grafman, Ibañez, Deiber, Dold, & Hallett, 1996). Interfering with normal processing in this area through transcranial magnetic stimulation (TMS) affects tactile identification of Braille letters in the blind, but not in seeing people who instead display impaired visual processing when stimulated in this area (Cohen, Celnik, Pascual-Leone, Corwell, Falz, Dambrosia, Honda, Sadato, Gerloff, Catalá, & Hallett, 1997). It therefore appears that the functional development of cortical regions is strongly constrained by available sensory inputs and that the final organization of the cortex is an outcome of interactive processes such as competition for space.

Embodiment. The fact that the brain is embedded in a body has a profound impact on the constraints on cognitive development. On the one hand the body develops in parallel with cognitive abilities and serves to change the information available to the child. In this way the developing body can serve as an information filter to the brain: for example, during the first months of life visual acuity is low, leading to less detailed visual input than in the mature visual system. Likewise, the infant's ability to manipulate her environment develops progressively as she moves from lying to sitting, reaching and grasping, crawling and walking, allowing her to actively generate new inputs to her sensory systems with increased sophistication. It has been speculated that the gradual increase in complexity of sensory inputs might be beneficial to the developing child (Turkewitz & Kenny, 1985; Newport, 1990). According to this 'Less is More' hypothesis, initially only the coarser aspects of the environment are processed and more detail is added gradually, supporting the development of

progressively more complex mental representations while protecting the immature mind from being overloaded with irrelevant detail too early.

On the other hand, the body also serves to constrain the mental computations necessary to solve a problem. For example, the structure of the skeleton, muscles, tendons and ligaments together with continuous proprioceptive feedback affords only certain movement trajectories in reaching for an object, thus greatly simplifying the computations that are necessary to execute that movement.

The embodiment view highlights that pro-active exploration and manipulation of the environment are an essential part of cognitive development. The child does not passively absorb information but actively generates and selects the information from which to learn. This view also suggests that the classic view of cognition – the mind receiving rich representations of the external world, operating off-line on these representations and generating outputs, neglects real-time interactions and dynamical loops between body, brain and environment (Kleim, Vij, Ballard, & Greenough, 1997).

Ensocialment. The final constraint in the neuroconstructivist framework is the social environment in which a child develops. For example, it has long been acknowledged that the contingent timing of interactions between a mother and child can have a profound effect on the development of secure attachment, the expression of emotions as well as social and cognitive development (reviewed in Harrist & Waugh, 2002). By contrast, an atypical social environment, for example early traumatic experiences such as death of a parent, maternal depression, child abuse or neglect, can have severe adverse effects on the neural and behavioural development of the infant (Murray, 1992; Kaufman, Plotsky, Nemeroff, & Charney, 2000; Cirulli, Berry, & Alleva, 2003).

Common Principles

The neuroconstructivist framework identifies a number of common principles and mechanisms that operate across all levels of analysis and shape the development of neural structures and thus, mental representations. The main principle is *context dependence*. On all levels, the constraints that shape the developing neural system establish a context that affects the specific outcome of development. This is true for the cellular environment of the developing neuron, for interacting brain regions, and for the specific details of the biological and social environment of the child. A specific context is realized through the processes of *competition, cooperation*, *chronotopy* and *pro-activity*. Competition leads to the specialization of components in a system, allowing for the development of more complex representations. Likewise, cooperation leads to the integration between sub-components and for existing knowledge to be re-used at higher levels. 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 relies on *pro-activity* in selecting information from the environment.

Together, these mechanisms lead to the *progressive specialization* of the learning system. Some neural circuits, once wired, may be hard to alter. Likewise, cognitive function becomes more entrenched and committed to a specific function, possibly becoming less sensitive to inputs outside its range (Scott, Pascalis, & Nelson, 2007).

These constraints and mechanisms result in a learning trajectory that at each point in time is determined by the immediate demands of the environment instead of

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. Such distributed, modality-specific representations have recently become the focus of investigation in adults (Pulvermüller, 2001; Barsalou, Simmons, Barbey, & Wilson, 2003).

3. Neuroconstructivism and Computational Modelling

Characterizing development as the outcome of local changes in response to multiple interacting constraints, and linking neural and cognitive development, lends itself to specification through computational modelling, particularly the connectionist approach to modelling cognitive development (Elman, Bates, Johnson, Karmiloff-Smith, Parisi, & Plunkett, 1996; Quinlan, 2003; Mareschal, Sirois, Westermann, & Johnson, 2007b; Spencer, Thomas, & McClelland, in press). Connectionist models are computational systems loosely based on the principles of neural information processing. As such they are placed on 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. Moreover, connectionist models have the ability to learn from data and are therefore relevant for explaining the mechanisms underlying behavioural change in cognitive development.

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Figure 1 around here

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A connectionist model consists of a large number of interconnected units that are idealized simplifications of biological neurons (although it should be noted that modellers do not assume that an artificial neuron in any sense stands for a biological neuron). Typically, each unit receives excitatory or inhibitory inputs from other neurons through weighted connections, sums up this activation and, if this activation exceeds a threshold, becomes active itself. Often these units are arranged in layers (Figure 1). In many models activation thus flows from an input layer that receives input from the environment, to internal layers of the network and on to an output layer that generates a response that is visible to the environment. There are different manners in which connectionist models learn, but learning nearly always proceeds by adjusting the strengths of the connections between the units. One of the most common learning principles is *backpropagation of error* (Rumelhart, Hinton, & Williams, 1986). In this *supervised* learning paradigm, activation flows through a layered network in response to an input, resulting in a pattern of activation over the units of the output layer. In supervised learning there is a teaching signal corresponding to the desired output for a specific input. This teaching signal can be construed as explicit feedback from a parent, or as the child comparing a prediction with an actual subsequent experience. The difference between the output that is generated by the network and the desired output is computed as the network error. This error is then used to strengthen or weaken the connection weights in order to change the flow of activation in such a way that on presentation of the same input, the network output will match the desired output more closely. Since a network is usually trained on a large number of data and each weight change is very small, there is pressure on the model to develop a weight pattern that produces the correct output for all inputs. This

pressure leads the model to extract generalizations from the data, which often allows it to produce meaningful outputs for previously unseen stimuli.

Another commonly used type of connectionist model is based on *unsupervised* learning. In this paradigm, output units are often arranged in the form of a map (such as in the *Kohonen feature map* (Kohonen, 1982), and the model learns to cluster input stimuli on this map on the basis of their similarities. In these models there is no teaching signal because the model's task is to make sense of the input data merely based on the structure of these data. Unsupervised models are attractive because they tend to form topographic maps like those found in many parts of the cortex. In self-organizing topographic feature maps, the similarity relationships from a high-dimensional environment (such as the visual world) are preserved on the two-dimensional mapping in that similar items occupy nearby positions on the map. Their closeness to cortical maps has led some researchers to claim a higher biological plausibility for unsupervised than for supervised models (e.g., Li, 2003). However, as clearly not all learning is unsupervised, both supervised and unsupervised models have their place in the modelling of cognitive development.

The validity of a computational model of development, that is, its ability to explain the mechanisms underlying cognitive change, can be assessed in different ways. Where developmental change is assessed in laboratory studies, the model can likewise be exposed to experimental situations in which stimuli are presented in a controlled fashion. An example of this approach has been the modelling of the development of infant categorization between 4 and 10 months of age. In experimental studies using the preferential looking paradigm, Younger and Cohen (1986) found that 4-month old infants were able form categories on the basis of the perceptual features of cartoon animals. When 10-month olds were shown the same

animal pictures they categorized them in different ways, indicating that they, but not the 4-month olds, were sensitive to the correlational structure between object features. This transition from feature-based to correlation-based category formation was modelled in a connectionist system that was exposed to encodings of the same stimuli that the infants had seen (Westermann & Mareschal, 2004). By gradually changing the function by which network units integrate their incoming activations the model displayed a developmental trajectory that at one point mimicked the 4-month olds' behaviour, and at a later point, the 10-month olds' behaviour. The change in the network function leading to this behavioural change was interpreted as infants developing the progressive ability to form more precise internal representations of objects in their environment on the basis of experience-dependent neural tuning during their first year of life (see Thomas, 2004, for discussion).

In cases where development is assessed outside the laboratory, such as in language development, a model can be exposed to data that reflects a child's experience in the real world. For example, several connectionist models have been used to investigate the mechanisms underlying children's learning of the English past tense (e.g., Rumelhart & McClelland, 1986; Westermann, 1998; Plunkett & Juola, 1999). These models are usually trained on a set of verbs that reflects the frequency of occurrence in spoken (and sometimes written) language. In this case, the characteristic error patterns observed in children of different ages are compared with the performance of the model at different stages of learning.

Finally, the validity of a model can be assessed by generating predictions that can then be tested against children's performance. As noted above connectionist models often generalize to previously unseen stimuli in meaningful ways (for example, to new objects in categorization studies or nonsense words in past tense

learning), and these generalizations can be assessed against the behaviour of children tested on the same stimuli.

Connectionist models are an ideal tool to study development within the neuroconstructivist framework, because the learning trajectory in a model is likewise the outcome of local adaptations to interacting constraints. 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 units, the pattern of connections between units and the way in which environmental inputs are encoded for processing; plasticity constraints such as the function and parameters of the weight update rule; and environmental constraints such as the type, frequency and order of the stimuli presented to the model. More recently, insights from developmental cognitive neuroscience have been incorporated into connectionist modelling by allowing for experience-dependent structural development and the gradual integration of network sub-components (Westermann, Sirois, Shultz, & Mareschal, 2006; Mareschal et al., 2007b), adding further constraints to the developmental model.

As we will discuss below, manipulating these constraints is particularly well suited to exploring the causes and consequences of atypical development.

4. Neuroconstructivism and Developmental Disorders

Developmental disorders can shed light on the way in which constraints at the genetic, neural, physical and social levels of description operate to shape cognitive development. Several questions come to the fore in considering what happens when a child's development does not proceed as expected. It is important to establish the role

that the developmental process itself plays in producing the behavioural impairments that are observed in, for example, the older child with autism or language impairment. It is also important to consider the extent to which emerging impairments are influenced by the interactivity of brain systems or by disruption to the timing and order in which developmental events usually unfold. Finally, we must consider how the child's social context can serve to attenuate or exaggerate deficits.

Variability is a pervasive feature of cognitive development, both in terms of intelligence in typically developing children and in the possibilities of development impairments. Disorders can have several causes. They can stem from genetic abnormalities, such as in Down syndrome (DS), Williams syndrome (WS), and Fragile X. They can be identified on the basis of behavioural impairments, such as in autism, Specific Language Impairment (SLI), Attention Deficit Hyperactivity Disorder (ADHD), or dyslexia. In the case of behaviourally defined disorders, genetic influence is frequently suspected as these conditions can run in families, but the genetic basis is not fully understood. Finally, disorders can be caused by atypical environments, either biochemical, such as mothers taking drugs during pregnancy, or psychological, such as cases of deprivation or abuse.

Notably, some developmental disorders can exhibit uneven cognitive profiles. For example, there may be particular problems in language but less so in nonverbal areas (e.g., SLI). Some abilities can appear relatively stronger against a background of low IQ (e.g., face recognition in WS). To understand disorders, we must explain both how development can be generally poor, perhaps occurring more slowly than usually, perhaps terminating at low levels of ability, and also how abilities can be impaired to different extents (Thomas, Purser & Richardson, in press).

Within the neuroconstructivist framework, developmental disorders can be understood through altered constraints that push the developmental trajectory off its normal track. Atypical development can, like typical development, be characterised as an adaptation to multiple interacting constraints, only that in this case the constraints are different. These atypical constraints then lead to different (sub-optimal) outcomes possibly through a deflection in the process of representation construction. This explanation of atypical development stands in contrast to theories that assume that disorders arise from isolated failures of particular functional modules to develop (see Karmiloff-Smith, 1998, 2008, and Thomas, Purser & Richardson, in press, for discussion). Modular explanations were characteristic of early investigations of several disorders: autism was initially viewed in terms of the failure of an innate, dedicated theory-of-mind module to develop (Frith, Morton, & Leslie, 1991); and SLI in terms of selective damage to a genetically pre-specified syntactic module (van der Lely, 2005).

Empirical evidence supports the role of development in producing atypical cognitive profiles, because these profiles do not necessarily retain a consistent shape across development. For example, when Paterson, Brown, Gsödl, Johnson, and Karmiloff-Smith (1999) explored the language and number abilities of toddlers with DS and WS, they found a different relative pattern to that observed in adults with these disorders. The profile in early childhood was not a miniature version of the adult profile.

The neuroconstructivist approach places the developmental process at the heart of explanations of developmental deficits (Karmiloff-Smith, 1998). Empirically, the framework encourages researchers to focus on trajectories of development, rather than static snapshots of behaviour at different ages in comparison to typically

developing children matched for chronological or mental age. The theoretical emphasis is that the disordered system is still developing but it does not possess the information or neurocomputational constraints that enable it to acquire a domain. Notably, in some circumstances, atypical underlying cognitive processes may be sufficient to generate normal levels of behaviour on particular tasks, for example, as demonstrated by research on face recognition in children with autism and WS (Annaz et al., 2009; Karmiloff-Smith et al. 2004). In other cases, the atypical constraints may even produce better than typical performance for a given behavioural task, such as in some aspects of perception in autism (Mottron, Belleville, & Menard, 1999; Shah & Frith, 1983). Such possibilities make it clear how a neuroconstructivist developmental framework differs from viewing disorders as if they were normal systems with broken parts. Nevertheless, a modular view of developmental disorders still persists amongst some researchers. Thus Temple and Clahsen (2002, p.770) argue that "there remains no empirical evidence in any developmental disorder that the ultimate functional architecture has fundamentally different organisation from normal, rather than merely lacking or having reduced development of components of normal functional architecture."

Several of the core ideas of Neuroconstructivism are emphasized by the study of atypical development. For example, in some cases localisation and specialization of cortical areas appear atypical (Karmiloff-Smith, 2008). Adults with WS exhibit face recognition skills in the normal range but examination of ERPs revealed different neural activity compared to typical controls (e.g., 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 DS (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 (e.g., in autism, SLI, and dyslexia). Alterations in the level of abstraction achieved in forming internal representations, or in the dimensions of similarity that those representations encode, can play a material role in the ability of other brain systems to employ this information to drive other processes. It is possible that in autism, SLI, and dyslexia, for example, 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 to specify what is different about the constraints and mechanisms of change in a given disorder. Here again, computational modelling offers a very useful tool.

5. Modelling Atypical Development

Constructing a computational model of development involves making a range of decisions. These include the nature of the input and output representations corresponding to the target cognitive domain, the regime of training experiences, the

specific architecture and learning algorithm, and a set of free parameters. These are concrete realisations of the constraints that act on or shape the normal developmental trajectory (Mareschal & Thomas, 2007; Spencer, Thomas & McClelland, 2009). Because the constraints can be systematically varied and the effects of such variation on performance investigated in detail, models provide a mechanistic means to explore candidate ways in which developmental impairments can arise.

From a formal learning perspective, alterations to the model's constraints can produce a number of effects. They may change the nature of the *hypothesis space* that can be reached (i.e., the knowledge that can be stored); they can change the nature of the *search* of an existing hypothesis space (i.e., how information from the environment can be used to acquire this knowledge); they can change the *inductive bias* which the system uses to generalise its knowledge to novel situations; or they can change the set of *training examples*, either in the system's autonomous, self-guided sampling of the environment or when the environment is itself impoverished.

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. These outcomes are not always predictable using analytical means (and are therefore called 'emergent properties'). One issue to which models have been applied is the consequence of multiple on-going interactions across development between the components that make up a whole cognitive system. Baughman and Thomas (2008) used *dynamical systems modelling* to simulate development in different types of cognitive architecture that were constructed from multiple interacting components. These architectures included distributed, modular, hemispheric, central processor, and hierarchical designs. Baughman and Thomas examined how early damage to a single component led to consequent impairments

over development. 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 through 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. The model highlighted the importance of understanding causal connectivity in explaining the origin of uneven cognitive profiles.

One ongoing debate in the field of development disorders is their relation to acquired disorders following brain damage. Is a child with SLI similar in any way to the adult with acquired aphasia? Modelling generated insights into this question by investigating the consequences of damaging a learning system in its initial state (analogous to a developmental disorder) compared to damaging a system in its trained state (analogous to an adult acquired deficit). Using a backpropagation connectionist model of development, Thomas and Karmiloff-Smith (2002) demonstrated that some types of damage hurt the system more in the 'adult' state (e.g., severing network connections) while others hurt the system more in the 'infant' state (e.g., adding noise to processing). The adult system tolerates noise because it already possesses an accurate representation of the knowledge, but loss of network structure leads to a decrement in performance since connections contain established knowledge. By contrast, the infant system tolerates loss of connections because it can reorganise remaining resources to acquire the knowledge, but is impaired by noisy processing since this blurs the knowledge that the system has to learn. Empirical evidence supports the importance of a good representation of the input during language acquisition. When McDonald (1997) analysed the conditions for successful and unsuccessful language acquisition across a range of populations (including early and

late first language learners, early and late second language learners, individuals with DS, WS and SLI), the results indicated that good representations of speech sounds (or components of signs for sign language) were key in predicting the successful acquisition of a language. This included acquisition of higher level aspects such as syntax.

Models can also be used to establish whether one empirically observed feature of a disorder can serve as a causal explanation for other observed features via the development process. Triesch, Teuscher, Deák and Carlson (2006) proposed a computational model of the emergence of gaze following skills in infant-caregiver interactions. Triesch et al. constructed their model to test the idea that the emergence of gaze following may be explained in terms of the infant's gradual discovery that monitoring the caregiver's direction of gaze is predictive of where rewarding objects will be located in the environment. Triesch et al. based their model of gaze following on a biologically plausible reward-driven mechanism called Temporal Difference learning, which is a type of *reinforcement learning*. Reinforcement learning is a way of training computational models where certain outcomes are associated with rewards. In the current context, the model learned a sequence of actions that lead to a reward. The infant was construed as an agent situated in an environment. The agent generated actions based on what it perceived from the environment, and then potentially received a reward for its action, along with updated information of the new state of the environment. In the Triesch et al. model, the environment depicted a range of locations containing either the caregiver, an interesting object, or nothing. If the infant looked at the caregiver, information would also be available on the direction of the caregiver's gaze (i.e., whether the caregiver was looking at the infant or at some location in the environment). Rewards were available to the infant for fixating an

object or the caregiver, but rewards reduced over time as the infant became bored. A schematic of the model is shown in Figure 2.

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The model demonstrated three results. First, through rewards gained during exploration of the simulated environment, the model successfully acquired gaze following behaviour. Second, when the intrinsic reward value of observing faces was lowered to simulate autism (e.g., Annaz et al., 2009; Dawson et al., 1998) or raised to simulate Williams syndrome (e.g., Bellugi et al., 2000; Jones et al., 2000), the result in both cases was an atypical developmental trajectory, with the emergence of gaze following absent or substantially delayed. Empirically, deficits in shared attention (mutual gaze to a common object) are observed in both developmental disorders (Laing et al., 2002; Osterling & Dawson, 1994). Third, the implemented model could be used to predict possible deficits in other disorders. For example, it has been proposed that ADHD may in part stem from deficits in the reward-learning system (Williams & Dayan, 2005; Williams & Taylor, 2004). Richardson and Thomas (2006) demonstrated that appropriate parameter changes applied to the Triesch et al.'s model to simulate ADHD also produced impairments in the development of early gaze behaviour. If the genetic influence on ADHD (e.g., Banaschewski et al., 2005) means that precursors to the childhood behavioural symptoms can also be observed in infancy, then the Richardson and Thomas simulation predicts that atypical gaze following may be such a precursor.

The gaze-following model underscores a key theoretical point at the heart of Neuroconstructivism. Disorders that appear very different in their adult states may in

fact be traced back to infant systems that share much in common, but differ in certain low-level neurocomputational properties (see Mareschal et al., 2007). It is development itself – together with the characteristics of the system that is undergoing development – that produces divergent behavioural profiles.

6. Recent developments in methodology: the use of Trajectory Analysis

The neuroconstructivist focus on change over time generates a need for methods that allow us to describe, analyse, and compare the 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. New methods have been designed for just this purpose (see, e.g., Thomas et al., 2009).

The use of trajectories to study cognitive variation contrasts with a static 'snapshot' approach to measuring differences. 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 (CA) and a second match based on mental age (MA) derived from a relevant standardized test. If the disorder group shows an impairment compared with the CA-matched group but not with the MA-matched group, individuals with the disorder are considered to exhibit developmental delay on this ability. If, by contrast, the disorder group shows an impairment compared with both control groups, then the disorder group is considered to exhibit developmental deviance or atypicality (see, e.g., Hodapp, Burack, & Zigler, 1990; Leonard, 1998).

The matching approach dispenses with age as an explicit factor by virtue of its design, but 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; Annaz, Karmiloff-Smith, & Thomas, 2008; Jarrold & Brock, 2004; Karmiloff-Smith, 1998; Karmiloff-Smith et al., 2004; Rice, 2004; Rice, Warren, & Betz, 2005; Singer Harris, Bellugi, Bates, Jones, & Rossen, 1997; Thomas et al., 2001, 2006, 2009). In this alternative approach, the aim is 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). In the context of disorder research, the impetus to move from matching to trajectory-based studies was a motivation to place development at the heart of explanations of developmental deficits, since as we have argued, the phenotype associated with any neurodevelopmental disorder does not emerge full-blown at birth but, rather, develops gradually and sometimes in transformative ways with age. This can only be studied by following atypical profiles over time.

Focusing on the example of disorder research, the aim of the trajectory methodology approach 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: (a) 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; (b) 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 (c) 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).[1]

The trajectory methodology 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 age 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 characterized 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

[1] An introduction to these methods can be found at http://www.psyc.bbk.ac.uk/research/DNL/stats/Thomas_trajectories.html

earlier than vocabulary or syntax. This can compromise our ability to assess developmental relations between abilities that plateau at different ages. Currently, one of the biggest challenges 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 currently 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. However, despite being psychometrically sound measures, standardised tests are frequently very blunt measures of the development of individual cognitive processes. One alternative is to appeal to more sensitive dependent measures such as reaction time. 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 or imageability of words in a speeded recognition task (Karmiloff-Smith et al., 1998).

Lastly, it is important to stress that irrespective of the correct theoretical explanation of a given disorder, trajectories are descriptively powerful because they 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. An accurate and detailed characterization of

empirical patterns of change is a necessary precursor to formulating causal accounts of developmental impairments.

7. **Recent developments in the genetic bases of atypical development**

Much work has been done to uncover the genes contributing to various developmental disorders. For some, e.g., autism, SLI and dyslexia, behavioural genetics has identified multiple genes of small effect as contributing to the phenotypic outcome (Plomin et al., 2003). In others, such as Williams syndrome, Down syndrome and Fragile X for which molecular genetics has already identified the gene or set of genes playing a role in the phenotypic outcome, efforts are placed on uncovering the function(s) of individual genes. These functions are rarely if ever at the cognitive level, although animal models are sometimes interpreted to suggest this. An example of this approach is spatial cognition in Williams syndrome. Here, members of a family who had a tiny deletion (ELN and LIMK1) within the WS critical region (WSCR) displayed spatial deficits similar to those found in WS. This was taken to indicate that the LIMK1 gene was a major contributor to spatial cognition (Frangaskakis et al., 1996).

LIMK1 konockout mice likewise revealed spatial deficits in the Morris Maze (Meng et al., 2002), providing further apparent evidence for an important role of LIMK1 in spatial processing. Although subsequent research on other LIMK1 patients revealed no spatial deficits, thereby challenging this view (Tassabehji et al., 1999; Karmiloff-Smith et al., 2003; Gray et al., 2006), this misses the neuroconstructivist point. It is not only the final effects of a gene's downstream pathway on cognitive-level outcomes that matters, but also LIMK1 expression over developmental time, thus to examine its basic-level functions during embryogenesis and postnatal development.

Indeed, LIMK1 is involved in dendritic spine growth and synaptic regulation across the brain, and not expressed solely in parietal cortex to form a spatial cognition module.

While animal models are useful for testing hypotheses about human disorders, obviously we must compare like with like at the cognitive level. The LIMK1 knockout mice were tested in the Morris Water Maze (Meng et al., 2002), a task that necessitated the mouse updating the representation of its position in space each time it moved. By contrast, the human spatial tasks had participants seated stationary at a table representing relations between objects. Therefore, while one problem involves egocentric space, the other involves allocentric space. This discrepancy has recently been remedied by designing human tasks that resemble the Water Maze (a pool filled with balls for children to search for a tin full of surprises) or mouse designs which resemble the human tasks, with the aim of bringing the cognitive demands of tasks in line across species comparisons. Obviously, it will be crucial to study both species across developmental time.

Although rare, partial deletion patients are useful in narrowing down the contributions of certain genes to phenotypic outcomes. Several patients with differing sized deletions within the WSCR have been identified. This allows us not only to examine basic functions, but also to analyse downstream and longer-term effects on aspects of cognition (Karmiloff-Smith, Grant, Ewing et al, 2003; Tassabehji, Hammond, Karmiloff-Smith et al., 2005). For example, one patient, HR, has only 3 of the 28 WS genes not deleted, yet she displays subtle differences with the WS fullblown phenotype (less of an overly friendly personality profile, somewhat less impaired intellectually, neither the gait nor the monotonous tone of those with classic WS). Cases like these enable us to hone in on the contributions of specific genes and

their interactions with others genes to the phenotypic outcome. Here again, development plays a crucial role. HR examined at 28 months had scores matching CA controls on general cognitive abilities. By 42 months, however, her performance was close to age-matched children with WS, and by 60 months her cognitive profile was identical to that of WS, although she remains different in personality and facial morphology. So, when making genotype/phenotype correlations, it is critical to take developmental time into account.

Would it be simpler to study a disorder caused by a single gene mutation (FragileX syndrome-FXS) rather than the 28 genes deleted in Williams syndrome? This question would only make sense if genes coded directly for cognitive-level outcomes. In reality, genotype/phenotype correlations in FXS are just as complex as in other syndromes. FXS is caused by an expansion of the CGG repeat at the beginning of the FMR-1 gene on the X chromosome. Healthy individuals have 7-~60 repeats with 30 repeats at the FMR-1 gene site. In most affected individuals, significant expansion of repeats (>200) results in hypermethylation and silencing of the FMR1 gene, a lack of messenger RNA and a diminution of the the FMR1 gene's protein product (Verkerk et al., 1991).

Realising that the FMR1 gene is involved in brain-wide processes such as synaptic regulation, the complexities of the cognitive outcome from a single gene make sense: problems with attention, language, number, and spatial cognition (Cornish, Scerif & Karmiloff-Smith, 2007).

Note that different genetic mutations may result in similar phenotypic outcomes. For example, although autism spectrum disorder (ASD) is considered by some to present with the opposite profile from WS, in fact they display numerous phenotypic similarities, such as atypical pointing, triadic attention, sustained and

selective attention, deficits in identifying complex emotional expressions, problems with pragmatics of language, auditory memory and theory-of-mind deficits, and a focus on features at the expense of global configuration. This suggests that multiple genes contribute to outcomes in both ASD and WS. Clearly the likelihood of one gene/one outcome is exceedingly small.

The importance of tracing gene expression over time became particularly clear with respect to the FOXP2 gene, originally claimed to be directly involved in speech and language deficits (Gopnik & Crago, 1991; Pinker, 2001). A British family (KE) had yielded several generations of children with speech and language impairments. When affected family members were discovered to have a FOXP2 mutation on chromosome 7 (Lai, et al., 2003), some hailed this as the gene contributing to human language evolution (Pinker, 2001; Whiten, 2007). But in-depth molecular analyses in humans (Groszer et al., 2008), chimpanzees (Enard et al., 2002) and birds showed that the function of this gene was widespread and contributed to the rapid coordination of sequential processing and its timing. FOXP2 is expressed more during learning than during other periods of development (Haesler et al., 2004**),** and its expression becomes increasingly confined to motor regions (Lai, Gerrelli, Monaco, Fisher & Copp, 2003). Why, in the human case, the mutation affects speech/language more than other domains is because speech/language is the domain in which the rapid coordination of sequential processing and its timing is critical. But FOXP2 is not specific to that domain. It also affects other domains, albeit more subtly. Indeed, it was shown that the KE family also had problems with imitating non-linguistic oral articulation, with fine motor control and with the perception/production of rhythm (Alcock et al., 2000), suggesting a domain-general effect of differing impact.

Note that Neuroconstructivism does not rule out domain-specificity; it argues

that it cannot be taken for granted when one domain is more impaired than another (Karmiloff-Smith, 1998). Rather, developmental trajectories and cross-domain interactions must always be explored. Unlike the Nativist perspective, Neuroconstructivism offers a truly developmental approach that focuses on change and emergent outcomes. Genes do not act in isolation in a predetermined way. The profiles of downstream genes to which FOXP2 binds suggest roles in a wide range of general, not domain-specific, functions including morphogenesis, neuronal development, axon guidance, synaptic plasticity and neurotransmission (Teramisu & White, 2007). This differs from theorizing at the level of cognitive modules and points to the multi-level complexities of genotype/phenotype relations in understanding human development in any domain.

In general, researchers must always recall that development really counts. For example, were one to discover, as is the case with WS adult brains, that parietal cortex is proportionally small, it cannot be automatically assumed that this causes their problems with spatial cognition and number. A question that must always be raised is whether parietal cortex started out smaller in proportion to other cortical areas or whether parietal cortex *became* small over time because of atypical processing in that region. Only a truly developmental approach can address such questions.

In our view, developmental disorders are explicable at a very different level from high-level cognitive modules; rather phenotypic outcomes are probably due to perturbations in far more basic processes early in development, such as a lack of/over-exuberant pruning, of differences in synaptogenesis, in the density/type of neurons, in differing firing thresholds, in poor signal to noise ratios, or generally in terms of atypical timing across developing systems. Rather than invoking a start state of innately-specified modules handed down by Evolution, the neuroconstructivist

approach argues for increased plasticity for learning (Finlay, 2007), i.e., for a limited number of domain-relevant biases, which *become* domain-specific over developmental time via their competitive interaction with each other when attempting to process environmental inputs (Johnson, 2001; Karmiloff-lSmith, 1998). In other words, Neuroconstructivism maintains that if the adult brain contains modules, then these *emerge developmentally* during the ontogenetic process of gradual localisation/ specialisation of function, i.e., progressive modularisation (Elman et al., 1996; Johnson, 2001; Karmiloff-Smith, 1992, 1998). In this sense, it is probable that domain-specific outcomes enabled by gene-environment interactions may not even be possible without the gradual process of development over time.

8. Conclusion

In this chapter we have described Neuroconstructivism as a new framework for understanding and explaining cognitive development, with cognition defined as based on patterns of neural activity that constitute mental representations. The main tenet of this approach is that development is a trajectory that is shaped by constraints at different levels of the organism, from genes to the social environment. Importantly there are also tight interactive loops between these levels: for example, neural activity affects the structural development of the brain's neural networks, partially mediated through the activity-dependent expression of genes. The structure of the network in turn constrains the possible patterns of activity. Neural activity leads to behaviour by which the physical and social environment can be manipulated, leading to new experiences and thus, new patterns of neural activity.

It is not necessary for an explanation of development to be useful that all changes and interactions are fully characterized: for example, in many cases it will not

be necessary to specify the genetic mechanisms by which neural activation is translated into experience-dependent neural plasticity. What is important, however, is to consider the implications of the dynamic nature of these constraints and their interactions. Ignoring them (or not knowing about them) has led researchers to develop theories of development in which a genetic blueprint leads to a pre-programmed maturation of encapsulated modules with innate functionality. On the opposite extreme, radical empiricist views would have argued for an 'anything goes' view of development under total plasticity. Neuroconstructivism rejects both views and instead it follows the Piagetian constructivist notion of pro-active interactions between the individual and the environment in which a strongly constrained developing system comes to optimally adapt to these constraints, be they 'typical' constraints in typical development or altered constraints in atypical development. Investigating the nature of these constraints and their role in shaping the developmental trajectory is at the heart of the neuroconstructivist endeavour.

Acknowledgements

This work was funded by ESRC grant RES-000-22-3394, EC grant 0209088 (NEST) and UK MRC grant G0300188. Thanks to Frank Baughman for his help in the preparation of Figure 2.

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Figure captions

Figure 1. A typical connectionist model with three layers. The input layer receives stimulation from the environment. The resulting activation of the input units is propagated to hidden and output units through weighted interconnections. The output layer produces a response visible in the environment. Different grey scales indicate the activation levels of the units.

Figure 2: Schematic of Triesch, Teuscher, Deák, and Carlson's (2006) computational model of the development of gaze-following behaviour, based on Reinforcement Learning.

Figures

Figure 1.

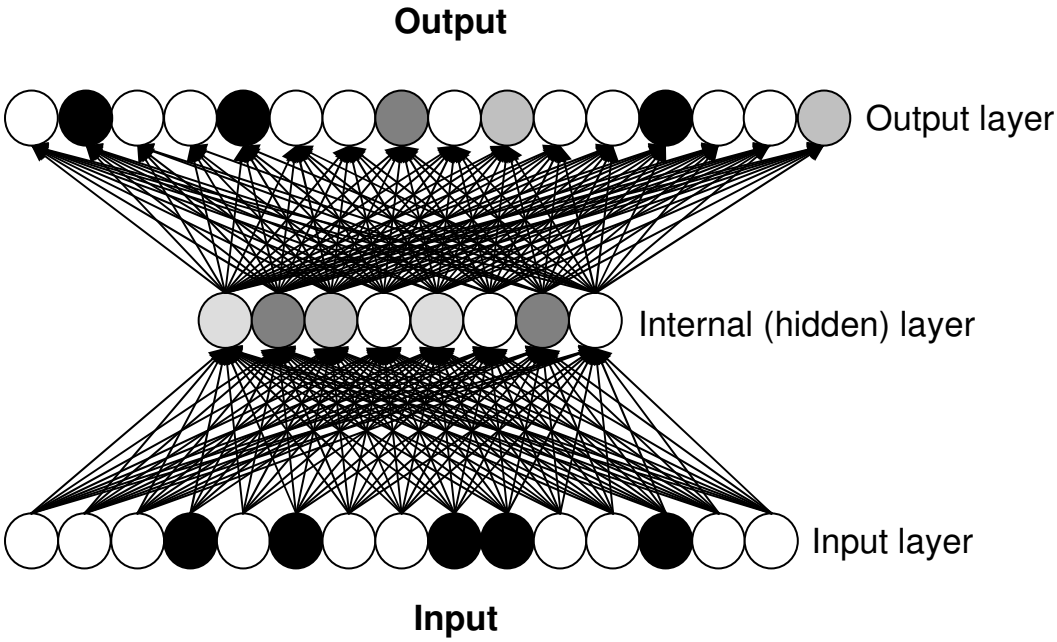
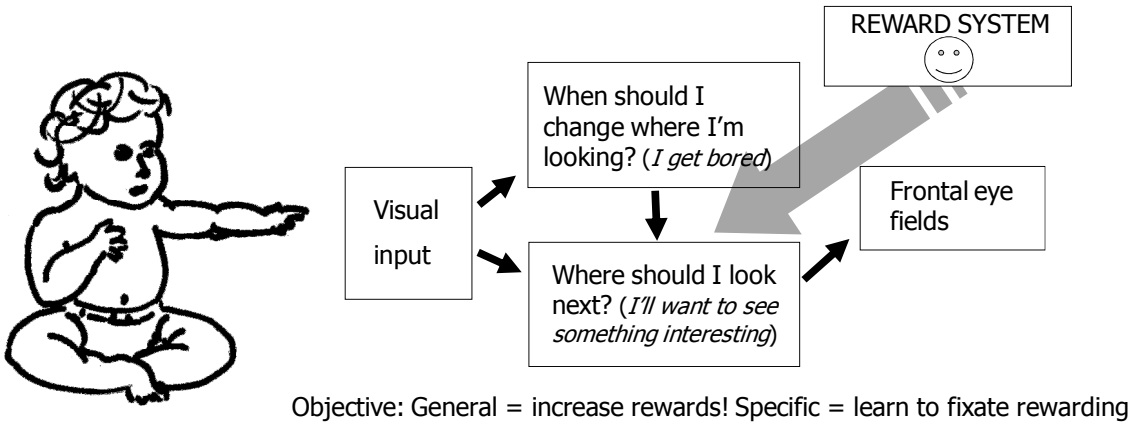


Figure 2



Objective: General = increase rewards! Specific = learn to fixate rewarding objects (make use of where mummy is looking?)