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**State-of-Science Review: SR-D13
Trajectories of Development and Learning Difficulties**

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Summary

It is becoming increasingly clear that little in development is pre-determined or permanently fixed. So, the study of children's learning difficulties needs to change from the still widely-held view that developmental disorders can be accounted for in terms of intact *versus* impaired brain modules, to one which takes serious account of the fact that the infant cortex passes from an initial state of high regional interconnectivity to a subsequent state of progressively increasing specialisation and localisation of brain function. With such early interconnectivity in mind, developmental neuroscientists must consider the possibility that an early deficit in one part of the brain may have subtle (or large) effects on other parts of the developing brain, even when subsequent behavioural scores fall 'in the normal range'. In studying developmental disorders, it is thus crucial to examine not only domains of clear-cut deficit, but also domains of behavioural proficiency. It is also vital to trace developmental disorders of higher-level cognition back to their infancy origins in low-level processing mechanisms. The trajectories approach leads to new syndrome-specific intervention and teaching strategies.

1. Introduction

Most aspects of human life – from gene expression, to brain structure/function, to cognitive processes, to overt behaviour – are, by their very essence, the result of development over time. Yet, in their excitement to use the Human Genome Project to uncover the functions of specific genes, researchers have often ignored how the gradual process of ontogenetic development affects gene expression.

The view that there might be a gene pre-specifying the structure of spatial cognition (Frankiskakis et al., 1996) or a gene or specific set of genes for language (Gopnik, 1990; Pinker, 1994; 1999; Wexler, 1996) often stems from a focus on the *adult* brain and from cognitive dissociations found in neuropsychological patients whose brains had developed normally prior to their brain trauma.

The *developing* brain is very different. Cortical regions start out highly interconnected (Huttenlocher and de Courten, 1987; Huttenlocher and Dabholkar, 1997; Neville, 2006), with the neonate cortex neither localised nor specialised at birth (Goldman-Rakic, 1987; Johnson, 2001). This allows interaction with the physical and social environment to play a crucial role in gene expression and in the ultimate cognitive phenotype (Karmiloff-Smith, 1998; Kuhl, 2004; Majdan and Schatz, 2006; Meaney and Szyf, 2005). Progressive localisation of cortical circuits, together with progressive restriction as to which inputs any particular brain circuit will process, i.e. gradual specialisation of cortex (Johnson, 2001), and progressive modularisation of function (Karmiloff-Smith, 1992) take many months or years before the child brain fully resembles that of the adult.

Thus, little in development is pre-determined or permanently fixed. Rather, epigenesis is probabilistic (Gottlieb, 2007), and gene expression is activity-dependent (Majdan and Schatz, 2006). If this holds for normal development, how does probabilistic epigenesis and activity-dependent gene expression affect the study of brain development and learning difficulties in atypical development, that is, of infants, children and adults with genetic disorders?

2. How theoretical biases affect research strategies

It follows from the introduction that the brains of atypically developing infants should not be viewed as normal brains with some parts intact and some impaired, but rather as brains that have *developed* differently

throughout embryogenesis and postnatal development. If one views the healthy child brain as composed of pre-specified, independently functioning modules, and the atypical brain as a neat juxtaposition of intact and impaired modules, then the brain/mind can be theoretically represented by a number of segregated boxes, one or more of which will have a line crossed through if impaired.

This kind of model implies that any damaged module has no effect on any of the other, purported independently functioning, 'intact' modules. Such a research strategy, therefore, frequently leads to a merely cursory check of the purported 'intact' parts of the brain, to confirm that individuals' scores fall 'in the normal range', placing thereafter a major emphasis on the impaired part(s). Within such a strategy and its underlying theory, claims reporting *relative* findings (domain X is worse than domain Y, but both are impaired relative to chronologically matched controls) easily slip to *absolute* statements: "Domain X is impaired; domain Y is intact".

A very different strategy arises if the researcher considers the initial state of the cortex to be composed of many interconnected parts, and that initially brain regions are not yet domain specific but 'domain relevant'. That is, they have certain neuronal and neurochemical properties that are somewhat more suitable for processing certain kinds of inputs over others. Initially, all regions compete to process inputs. There is widespread activity across the cortex for processing all incoming inputs. However, with time and sufficient experience, certain domain-relevant circuits win out and subsequently *become* domain specific and gradually modularised (Elman et al., 1996; Karmiloff-Smith, 1992; 1998). Neonates do not start out with innately specified modules that are either intact or impaired; rather, domain-specificity *emerges* from domain-relevant predispositions and competition, together with a subsequent gradual process of modularisation over developmental time.

These considerations lead to a different type of research strategy for atypical development, whereby the neuroconstructivist researcher will trace atypicalities back to their low-level origins in infancy, focusing on cross-domain timing. Furthermore, this approach expects to uncover subtle impairments across the system. In other words, as much effort is deployed in studying domains of behavioural proficiency (Karmiloff-Smith, 1992; 1998; Karmiloff-Smith et al., 2004; Thomas et al., 2001), as those of deficit.

Future research should keep in mind the whole cognitive system and whole brain interactions, rather than only focused domains and specific brain areas, and should study both deficits and proficiencies.

3. Factors affecting atypical development

While genetic mutations themselves will place constraints on learning, these are not the only factors that interact in producing atypical developmental trajectories. It would, for instance, be wrong to consider the environment as a static entity with which atypical brains interact. Interestingly, the moment that parents are told that their baby presents with a syndrome, parental behaviour changes. Quite unconsciously, parents hold and treat their atypical infant differently from their typically developing infant and place different constraints on their learning. For example, unlike the case of a typically developing child who is allowed to explore his environment, when the atypical baby starts to crawl, parents tend not to let them move far away.

Another example comes from language learning: whereas the typically developing child is allowed to over-generalise (e.g. temporarily use 'dog' for several different animals) in the parents' secure belief that this is part of normal learning, parents of atypically developing children tend to sanction over-generalisation and correct their child immediately, in their fear that their child may never learn the correct words (Mervis and Bertrand, 1997). This affects subsequent categorisation abilities.

My personal experience, over years of dealing with parents and their children with genetic mutations, is that the rare parents who treat their atypical child as if he or she were normal are the ones whose children turn out to have the best prognosis.

Very early parental training during the first weeks of life would encourage parents of atypical children to do their utmost to treat them as if they were normal in terms of social interaction and exploring the environment.

4. Importance of full developmental trajectories

In the past, the main method for understanding developmental disorders used matching. This could be group matching or individual subject matching, and could be done on the basis of full IQ, Verbal IQ or Performance IQ, or Mental Age in specific domains. We have recently argued that such matching is theoretically laden and that the choice of matching methodology can radically change the interpretation of the differences between clinical groups and controls. Noteworthy is the fact that those such as the disciples of Chomsky, who deny any relationship between language and cognition, control for general intelligence, implying thereby that intelligence does play a role in language!

We have, therefore, argued for a method of tracing full, task-specific developmental trajectories (Annaz, 2006; Annaz et al., 2008; Ansarai and Karmiloff-Smith, 2002; Karmiloff-Smith, 1998; Karmiloff-Smith et al., 2004; Thomas et al., 2001; submitted). In our view, an understanding of mechanism will be furthered by the richer descriptive vocabulary provided by the trajectories approach (for example, distinguishing three different types of delay that are conflated in the matching approach: delayed onset; slowed rate; and delayed onset + slowed development).

Moreover, an optimal design for studying developmental disorders is, in our view, to combine initial cross-sectional trajectory designs on large numbers of participants at each consecutive age, with longitudinal follow-up of a smaller number of participants across the same age range.

But where do we start such trajectories? In other words, when does cognitive development begin? In the past many would have argued that cognition only begins with the onset of language production around 18 to 24 months. Nowadays, it is generally recognised that linguistico-cognitive development begins at birth, with the visual, auditory and tactile systems rapidly gaining knowledge through exploration of the physical and social worlds. However, linguistico-cognitive development actually begins during the final trimester of intrauterine life (Karmiloff-Smith, 1995). Using controlled experiments and measurements of changes in heart rate or leg kicking, foetuses have been shown to discriminate different sounds, different languages, different forms of music, and different voices (Hepper, 1989; 1991), and to remember what they heard *in utero* once they are born. Yet, currently, we know nothing about prenatal discriminatory abilities and memory in atypically developing foetuses such as those with Down syndrome (DS).

A study of women carrying DS babies to term, measuring during the final trimester foetal sensitivities to a variety of auditory stimuli would assess whether the serious delays witnessed in subsequent DS development start *in utero* or only after birth.

After birth, it is critical for research to focus, not on snapshots of development at specific ages, but on the full developmental trajectory from birth to adulthood. In previous research we have shown that the profile of proficiencies and learning deficits at one stage, say, infancy, can be very different from the profile in, for example, adulthood (Karmiloff-Smith, 1998; Paterson et al., 1999).

Moreover, it is vital for understanding higher-level cognitive phenotypes to understand how they originated in low-level processing mechanisms early on in development. For example, a deficit in visual saccadic planning in Williams syndrome (WS) (Brown et al., 2003) may lead to atypical triadic interaction (Laing et al., 2002) and thence contribute, alongside other factors (Nazzi, et al., 2005; Nazzi and Karmiloff-Smith, 2002; Nazzi et al., 2003; Paterson et al., 1999), to the late onset of language in this clinical population.

So, not only should we explore the communication-relevant contributions to a delay in language, but also examine other domains such as visual saccadic planning which may affect triadic interaction and thence the learning of vocabulary. This delay in vocabulary will subsequently affect the acquisition of syntax, the onset of which, in the normal case, requires a critical mass of vocabulary items (Bates, 1994). Thus, something that might at first blush seem irrelevant at one level to the acquisition of syntax, namely, saccadic eye movement planning, may have been critical at an earlier stage of development for subsequent language acquisition.

Interestingly, infants and toddlers with DS are not impaired in their planning of saccadic eye movements, yet they are just as delayed as infants and toddlers with WS in their vocabulary (Paterson et al., 1999). Thus, the reason for the similar delay and similar overall IQ must be different across these two syndromes, pointing to the importance of tracing full, cross-syndrome, developmental trajectories.

Timing is clearly a vital factor in development. In children with Specific Language Impairment, for instance, a problem with processing rapid sequential transitions early in development may be overcome later in development. But its importance resides in the early period (Karmiloff-Smith, 1998; Benasich and Spitz, 1999), i.e. at the very time when it was necessary for interactions with other aspects of development. Such reasoning about the importance of timing leads to policy implications.

The developmental trajectory approach recommends that intervention should target low-level processes at the outset of development in infancy and not await the overt problems that emerge and become overly consolidated in school-aged children.

5. International comparisons

In my personal view, whereas research into atypical development in the USA is more frequent than in the UK, the work in the UK is deeper. For example, in studies of genotype/phenotype correlations, US studies only use standardised tests (Bellugi et al., 1994; 2000), whereas UK studies add to the standardised measures hypothesis-driven experimental tasks (see Karmiloff-Smith et al., 2004). By contrast, the formation of family support groups in the USA seems to be more successful than in the UK and other European countries. This seems to stem from a more positive attitude shown by US parents towards their atypical offspring, which links us back to the possibility, discussed earlier, of offering parental training during the first weeks of life for parents of atypical children. In France, by contrast, family support groups are far less successful, partly due to the high esteem that intellect holds in that society and the embarrassment that parents feel when their child has learning difficulties. Finally, what parents want from research on learning difficulties often differs from the focus of researchers, the former being more applied.

More interaction between parental groups representing children with learning difficulties and the education/health authorities would be useful in addressing this.

6. Future of the study of learning difficulties in the UK

Although some developmental disorders, such as Fragile X Syndrome (FXS), are inherited and families receive genetic counselling once one child is born with the condition, many other developmental disorders are sporadic in nature and due to chance mutations during meiosis or subsequent embryogenesis. Moreover, with the huge advances in neonatal care, more children with genetic mutations will survive. This underlines the importance of placing research emphasis on very early development if we are to help such children shift their atypical trajectory closer to the normal one.

Furthermore, the greater survival rates of children with serious genetic disorders are likely to place an ever-increasing burden on health and education services. However, at present it seems that funds for education tend to increase at an age when brain plasticity starts to decrease (although some degree of plasticity is of course maintained even in adulthood).

Many children with learning difficulties also bear the burden of a dysmorphic face, a flat intonation in their speech output and/or an awkward gait. This can lead to automatically treating them as different or less able, as well as to bullying in the school environment, particularly where special units are situated in mainstream schools, and thus to social exclusion. However, placing children in separate, special needs schools is also not a solution, because teaching and expectations frequently reflect the lowest common denominator across the children. I believe that the trajectories approach will lead to more syndrome-specific teaching styles.

More research funds should be earmarked for studies of very early cognitive and brain development. Tracing these over developmental time may be useful in developing syndrome-specific intervention and teaching strategies that start in early infancy and toddlerhood.

7. Brain changes in atypical development

Does the atypical brain show increasing hemispheric specialisation, as in the normal case? Or does processing in atypical brains with learning difficulties tend to remain bilateral? Take, for example, face processing. In the normal case (de Haan et al., 2002; Johnson, 2001; 2004), the progressive specialisation of brain function, enabled by the gradual pruning of non-relevant connections in the brain, continues to refine itself throughout childhood and the early adolescent years. In short, children are not born with a dedicated face processing module. Rather, this *emerges* progressively from experience and the competition across different cortical regions, until the most domain-relevant wins out.

But this is not necessarily the case in the atypical brain (see Karmiloff-Smith, 2007 for a concrete example of lack of localisation and specialisation of function in face processing in WS). If specialisation and localisation fail to occur in certain genetic disorders, then the brains of such individuals, even in adulthood, should show *more* widespread activity than is the case for healthy controls. For example, the brains of individuals with Fragile X syndrome have abnormally high synaptic densities, having undergone less pruning than in the normal case (Comery et al., 1997; Huttenlocher and de Courten, 1987; Huttenlocher and Dabholkar, 1997). In other words, synaptic pruning constitutes an essential part of normal development (Bourgeois and Rakic, 1993; Huttenlocher and de Courten, 1987; Huttenlocher and Dabholkar, 1997; Lund, et al., 1977), and is an important consideration when endeavouring to understand learning difficulties in atypical development.

Longitudinal studies would allow a greater understanding of the way in which the atypical brain progressively changes over developmental time.

8. Concluding thought

I have stressed throughout how a small perturbation in low-level processes very early in development can result in large developmental deficits in higher-level cognition later in development. This points naturally to very early intervention at the outset of development. Unless we trace the complexities of full developmental trajectories and seek to capture the intricate timing and cross-domain dynamics of very early development, we are, in my view, unlikely to understand the similarities and differences in the phenotypic outcomes of different children with learning difficulties.

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