

Atypical epigenesis

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Abstract

It is becoming increasingly clear that little in development is predetermined or permanently fixed. Rather, gene expression is activity dependent, and epigenesis is probabilistic. So, the study of genetic disorders needs to change from the still widely held view that developmental disorders can be accounted for in terms of intact versus impaired 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 increasing specialization and localization of 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 effects on other parts of the developing brain, even when 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. Atypical epigenesis may often involve a lack of specialization and localization of brain function over developmental time, even in cases of behavioural proficiency.

Introduction

In French, the word 'génétique' has two meanings: 'genetic' and 'developmental'. So, for instance, Piaget's term 'épistémologie génétique' was intended to cover *developmental* epistemology, in other words the progressive growth of knowledge over time (Piaget, 1952, 1971). The double meaning serves to remind us that 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. In other words, development really counts, both in healthy and atypical epigenesis. 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 for spatial cognition (Frangiskakis, Ewart, Morris, Mervis, Bertrand, Robinson, Klein, Ensing, Everett, Green, Proschel, Gutowski, Noble, Atkinson, Odelberg & Keating, 1996) or a gene or specific set of genes for language (Gopnik, 1990; Pinker, 1994, 1999; Wexler, 1966; but see Bates, 1979, 1984, 1994, for a very different position on language evolution) often stems from a focus on the structure of the *adult* brain and from cognitive dissociations found in neuropsychological patients whose brains were fully and normally developed prior to their brain trauma. The *developing* brain is very different. Cortical regions start out highly interconnected (Huttenlocher & de Courten, 1987; Huttenlocher & Dabholkar,

1997; Neville, 2006). Indeed, the neonate cortex is neither localized nor very specialized at birth (Goldman-Rakic, 1987; Johnson, 2001). This allows interaction with the environment to play a crucial role in gene expression and in the ultimate cognitive phenotype (Karmiloff-Smith, 1998; Kuhl, 2004; Majdan & Schatz, 2006; Meaney & Szyf, 2005). Progressive localization of cortical circuits, together with progressive restriction as to which inputs any particular brain circuit will process, i.e. gradual specialization of cortex (Johnson, 2001), and progressive modularization of function (Karmiloff-Smith, 1992) take many months or years before the child brain fully resembles that of the adult. Surprisingly little in development is strictly predetermined or permanently fixed. Rather, epigenesis is probabilistic (Gottlieb, this volume), and gene expression is activity-dependent (Majdan & Schatz, 2006). If this holds for normal development, how does it affect the study of atypical development, i.e. of infants, children and adults with genetic disorders?

Studying atypical epigenesis

It follows from the previous section that the brains of atypically developing infants should not be viewed as normal brains with parts intact and parts impaired, but brains that have *developed* differently throughout embryogenesis and postnatal development. Why, then, did the temptation to use the adult neuropsychological model of independently functioning modules for explaining genetic

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disorders in children hold its power throughout the 1990s? And why does it continue to permeate much of the developmental disorders literature in the new millennium (see critiques in Karmiloff-Smith, Scerif & Ansari, 2003; Thomas & Karmiloff-Smith, 2002)? In my view, it is because the explanatory framework is static and thus leads to simpler kinds of research strategy. Indeed, 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', i.e. 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, i.e. there is widespread activity across 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 modularized (Elman, Bates, Johnson, Karmiloff-Smith, Parisi & Plunkett, 1996; Karmiloff-Smith, 1992, 1998). Indeed, the simple dichotomy between domain-specific and domain-general mechanisms fails to capture both the dynamics of development and important differences in brain regions in terms of types of neuron, neuronal density, neuronal orientation, ratio of grey to white matter, neurochemical transmitters, etc., as well as the differential timing of their development.

Neonates don't 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 modularization over developmental time.

These considerations lead to a different type of research strategy for atypical development. So, for instance, the neuroconstructivist researcher will endeavour to trace

atypicalities back to their low-level origins in infancy. Furthermore, research within this approach expects to uncover not only serious deficits in a given domain, but also subtle impairments elsewhere in the system. In contrast to the modular strategy, this theoretical position leads to an in-depth analysis not only of impaired domains, but also of the cognitive and brain processes underlying the 'scores in the normal range' in other domains. In other words, as much effort is deployed in studying domains of behavioural proficiency (Karmiloff-Smith, 1992, 1998; Karmiloff-Smith, Thomas, Annaz, Humphreys, Ewing, Brace, van Duuren, Pike, Grice & Campbell, 2004; Thomas, Grant, Barham, Gsödl, Laing, Lakusta, Tyler, Grice, Paterson & Karmiloff-Smith, 2001), as those of deficit.

A concrete example of progressive specialization/localization in typical and atypical development

Face processing serves as a good illustration of domain relevance and of progressive specialization and localization of function in the normal case (Johnson, 2004). While processing faces during normal infancy, the cortex starts out by being very active over wide brain regions in both hemispheres. After 6 months, however, predominance of face processing gradually moves to the right hemisphere, which turns out to possess domain-relevant properties that lend themselves somewhat better to the configural processing of faces. By the end of the first year of life, the baby's brain not only shows a pattern of localization for face processing approaching that of adults, but also displays a restriction of the inputs processed by that particular brain circuit (de Haan, Humphreys & Johnson, 2002; Johnson, 2001, 2004). This progressive specialization of 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.

What about atypical development? Despite average IQs in the mid-1950s, adolescents and adults with Williams syndrome¹ (WS) have been shown by several laboratories worldwide to be very proficient on some standardized face processing tasks, such as the Benton

¹ Williams syndrome is a neurodevelopmental disorder, caused by a hemizygous deletion of some 28 genes on one copy of chromosome 7 (Donnai & Karmiloff-Smith, 2000).

(Bellugi, Wang & Jernigan, 1994; Bellugi, Lichtenberger, Jones, Lai & St George, 2000) and the Rivermead (Udwin & Yule, 1991). On such standardized tasks, the scores of the majority of individuals with WS fall within the normal range. This initially led to claims that a face-processing module in WS was 'intact' (Bellugi *et al.*, 1994). Since the seminal work of Bellugi and her colleagues, many other laboratories have studied face processing in WS. The general consensus (but see Tager-Flusberg, Pless-Skewer, Faja & Joseph, 2003, who continue to maintain that WS face processing is no different from that of healthy individuals) is that the behavioural proficiency in WS face processing is underpinned by different cognitive processes from controls (Annaz, 2006; Deruelle, Macini, Livet, Casse-Perrot & de Schonen, 1999; Karmiloff-Smith, 1997, 1998; Karmiloff-Smith *et al.*, 2004; Rossen, Jones, Wang & Klima, 1996). This was further corroborated by our ERP study comparing the brain processes of healthy controls versus adolescents and adults with WS when processing faces and cars (Grice, de Haan, Halit, Johnson, Csibra, Grant & Karmiloff-Smith, 2003), as well as in another study of cerebral integration (Grice, Spratling, Karmiloff-Smith, Halit, Csibra, de Haan & Johnson, 2001). The face processing findings highlighted the fact that, while healthy controls processed both human and monkey faces in a similar way, their brains treated cars very differently. By contrast, the brains of participants with WS displayed no differences between faces and cars. Moreover, unlike the healthy controls who showed a right hemisphere dominance for upright faces, the clinical group failed to display any difference in hemispheric activation (Karmiloff-Smith *et al.*, 2004). This highlights two facts about the deviant trajectory of WS face processing. First, there is a lack of specialization: individuals with WS show similar electrophysiological responses for both faces and cars, i.e. they have not progressively restricted the brain circuits responsible for face processing uniquely to face stimuli, but process all manner of visual stimuli in a similar way. Second, there is a lack of localization: healthy controls show stronger processing for faces in the right hemisphere, whereas the clinical population displayed equivalent bilateral activation. The lack of specialization and localization in WS face processing indicates that, despite enormous daily experience with faces, a face processing module fails to emerge over developmental time in this clinical population.

Does a lack of specialization and localization hold for all disorders?

Although the notion of a lack of specialization, localization and modularization of function turns out to be an

enlightening hypothesis for some disorders such as WS, obviously this need not hold for all disorders. Some might display a lack of specialization alongside brain localization that resembles that of healthy controls but with which they process other stimuli that normal groups do not. By contrast, for other clinical groups, the opposite might obtain: normal specialization but in a different brain circuit from healthy controls. This scenario may hold for children who have had early hemispherectomies, where one hemisphere has to take over the functions of the other.

This brings us to a further speculation: if specialization and localization 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. There is certainly an indication that this might hold, since the brains of normal infants prior to pruning show more widespread activity compared to older children's (Johnson, 2001). Another clue comes from brains of individuals with Fragile X syndrome, for instance, which have been shown to have abnormally high synaptic densities, i.e. they have undergone less pruning than is the normal case (Comery, Harris, Willems, Oostra, Irvin, Weiler & Greenough, 1997; Huttenlocher & de Courtene, 1987; Huttenlocher & Dabholkar, 1997). In other words, synaptic pruning leading to increasing specialization constitutes an essential part of normal development (Bourgeois & Rakic, 1993; Huttenlocher & de Courten, 1987; Huttenlocher & Dabholkar, 1997; Lund, Boothe & Lund, 1977), and is an important consideration when endeavouring to understand atypical development.

Timing is also likely to play a crucial developmental role (Johnson, Karmiloff-Smith, Pennington & Oliver, 2000; Karmiloff-Smith, 1998; Oliver, Johnson, Karmiloff-Smith & Pennington, 2000; Scerif & Karmiloff-Smith, 2005). If, for instance, pruning takes place too early, then the system may commit itself to specialization prior to sufficient confirmation, resulting in proficiency at processing only a few privileged types of input (Oliver *et al.*, 2000). By contrast, if pruning takes place too late or fails to occur, then activity in the cortex may be widespread in processing all types of input, thereby giving rise to problems in, for instance, multitasking (Mackinlay, Charman & Karmiloff-Smith, 2006). Such considerations lead us far from the boxology metaphor of intact and impaired modules.

Tracing full developmental trajectories

Given all the above arguments, it is clear that phenotypical differences in outcome must be traced back to their

origins in infancy (Karmiloff-Smith, 1998, 2006). Low-level processes in the visual, auditory and/or tactile modalities may cause the trajectory of normal development to deviate similarly across seemingly unrelated domains. For instance, I have argued (Karmiloff-Smith, 2006) that the difficulties witnessed in WS infants and toddlers with respect to planning visual saccades (Brown, Johnson, Paterson, Gilmore, Gsödl, Longhi & Karmiloff-Smith, 2003) may lead to atypical triadic interaction (Laing, Butterworth, Ansari, Gsödl, Longhi, Panagiotaki, Paterson & Karmiloff-Smith, 2002) and thence contribute, alongside other factors (Nazzi, Gopnik & Karmiloff-Smith, 2005; Nazzi & Karmiloff-Smith, 2002; Nazzi, Paterson & Karmiloff-Smith, 2003; Paterson, Brown, Gsödl, Johnson & Karmiloff-Smith, 1999), to the late onset of language in this clinical population. So, not only should we explore the language-relevant contributions to a delay in language, but we should also examine other domains like visual planning which may affect triadic interaction and thence the learning of vocabulary.

Unless we trace the complexities of full developmental trajectories (Annaz, 2006; Karmiloff-Smith *et al.*, 2004; Thomas *et al.*, 2001) and seek to capture the intricate timing and dynamics of very early development, we are, in my view, unlikely to understand the similarities and differences in the phenotypic outcomes of many developmental disorders.

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