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The tortuous route from genes to behavior: A neuroconstructivist approach

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In their excitement at using the human genome project to uncover the functions of specific genes, researchers have often ignored one fundamental factor: the gradual process of ontogenetic development. The view that there might be a gene for spatial cognition or language has emanated from a focus on the structure of the adult brain in neuropsychological patients whose brains were fully and normally developed until their brain insult. The *developing* brain is very different. It starts out highly interconnected across regions and is neither localized nor specialized at birth, allowing interaction with the environment to play an important role in gene expression and the ultimate cognitive phenotype. This article takes a neuroconstructivist perspective, arguing that domain-specific end states can stem from more domain-general start states, that associations may turn out to be as informative as dissociations, and that genetic mutations that alter the trajectory of ontogenetic development can inform nature/nurture debates.

The nature *or* nurture controversy is, one hopes, obsolete, because even the staunchest nativist and the most domain-general empiricist agree that development involves contributions from both genes and environment. However, first, because we lack a testable theory of the precise way in which gene expression and environment interact, and second, because of entrenched philosophical views about what it means to be human, the debate remains as to whether it is nature or nurture that plays the greater role in constraining the developing brain. For some, consistent regularities in the physical and social environments to which children are exposed play a critical role, whereas for others the environment acts merely as a trigger for the functioning of our prespecified biological endowment. For the latter position, data from adult neuropsychological patients and from studies of developmental disorders of genetic origin are often used to motivate strong claims about the evolution of the neonate brain in terms of innate cognitive modules, the contents of which are argued to be specified in our genetic makeup (Barkow, Cosmides, & Tooby, 1992; Duchaine, Cosmides, & Tooby, 2001; Pinker, 1997). The present article takes a neuroconstructivist perspective, arguing that domain-specific end states can stem from more domain-general start states, that associations may turn out to be as informative as dissociations, and that genetic mutations that alter the trajectory of ontogenetic development can inform debates about the nature of the human cognitive architecture.

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Some Definitions

Before we proceed, it may be helpful to clarify how I will be using the terms *innate* and *module*. The term *innate* has been employed in many different ways. A useful division can be made between architectural constraints, chronotopic (temporal) constraints, and representational constraints (Elman et al., 1996). No one denies that there are architectural and chronotopic constraints governing the outcome of human cognition. The question is whether representational innateness exists. In other words, does the neonate brain come equipped with innate knowledge (representations with content) of, say, the abstract structure of human language or the coordinates of spatial cognition, prior to any experience? Pinker (1994), for example, has claimed that children are born expecting language to contain nouns and verbs. Such a claim must call on representational innateness. My criticism of innate knowledge in this article is concerned with representational constraints that would seem to require a prespecified pattern of synaptic connectivity within the cortical microcircuitry of a specific neural system, whereas here I shall argue that such microconnectivity *emerges* from the gradual process of ontogenetic development (Karmiloff-Smith, 1992; Karmiloff-Smith, Plunkett, Johnson, Elman, & Bates, 1998).

My criticism here of modularity concerns the concept of *module* in the Fodorian sense (Fodor, 1983). Fodor specified a number of criteria that must hold for something to count as a module: Modules are hardwired (not assembled from more primitive processes), of fixed neural architecture, domain specific, fast, autonomous, mandatory (higher level cognitive processes cannot curtail their operation), and stimulus driven, give rise to shallow out-

puts, are insensitive to central cognitive goals, process only proprietary inputs, and most important, are informationally encapsulated. It is the *co-occurrence* of all these properties that constitutes, for Fodor, a module (see the critical discussion in Karmiloff-Smith, 1992). In my view, once one weakens these co-occurring criteria, as many authors have done, the notion of a module loses its theoretical power. Two questions arise. Can specific genes be invoked to map directly onto cognitive-level modules, and are cognitive-level modules prespecified or do they emerge gradually as the result of a process of modularization over developmental time?

Are Developmental Disorders Examples of Intact and Impaired Modules?

Insults to particular brain regions from accidents or stroke are claimed to have provided some of the most convincing evidence that the adult brain has modular structure (Rapp, 2001). Agrammatic patients, for example, are claimed to present with severe impairments in the grammatical structures of their language but have otherwise normal vocabulary and cognition (Grodzinsky, 2000). Other patients with different brain lesions seem to present with normal grammar but have serious word-finding difficulties (Miozzo, 2003). Yet others may retain completely normal language but have extreme difficulties in recognizing faces—that is, prosopagnosia (Farah, Levinson, & Klein, 1995). Data from other adult patients display interesting dissociations between, say, the semantic aspects of number and the semantics of other cognitive domains (Cappelletti, Butterworth, & Kopelman, 2001). Such cases have been invoked to bolster claims about the existence of independently functioning cognitive modules in the brain and to motivate the search for specific genes that map onto these modules.

One particularly impressive case of the seeming dissociation between language and the rest of cognition is that of a so-called savant linguist (Smith, 1999; Smith & Tsimpli, 1995). Christopher, who is in his 30s, has learned some 20 languages yet cannot tie his shoelaces. His nonlinguistic performance IQ is low, and this dissociation has led to the claim that his language skills must have developed independently of the rest of his intelligence (Smith & Tsimpli, 1995). Other theorists have argued for within-domain modular specializations between, say, nouns and verbs (Rapp & Caramazza, 2002). The most crucial data for this type of argument lie in the existence of double dissociations in which Patient A can process, say, faces but not objects and Patient B can process objects but not faces, or Patient C has impaired semantics but intact syntax and Patient D impaired syntax but intact semantics. These and other such claims using the double dissociation logic have abounded in the literature from studies in adult neuropsychology.

Espousing similar theoretical goals, many researchers who have studied children with genetic disorders have used the same logic as that for adult neuropsychology, seeking modular impairments and dissociations alongside normal scores in other domains claimed to be "intact/preserved/spared" (Baron-Cohen, 1998; Leslie, 1992; Smith & Tsimpli, 1995; Tager-Flusberg, Boshart, & Baron-Cohen, 1998; Temple, 1997; see the critical discussion in Karmiloff-Smith, Scerif, & Ansari, 2003). Researchers have focused on a number of genetically based disorders to assert the existence of a juxtaposition of modular deficits and preservations. For example, language and face processing have been claimed to be preserved in the neurodevelopmental genetic disorder known as Williams syndrome (WS; Bellugi, Marks, Bihrle, & Sabo, 1988; Bellugi, Wang, & Jernigan, 1994; Clahsen & Almazan, 1998; Pinker, 1994, 1999; Rossen, Klima, Bellugi, Bihrle, & Jones, 1996; Tager-Flusberg et al., 1998; Tager-Flusberg, Plesa-Skwerer, Faja, & Joseph, 2003). The impressive behavioral proficiency in WS with language and face processing has been found to coexist with a mean IQ of 56 (Mervis, Robinson, Rowe, Becerra, & Klein-Tasman, 2004) and with seriously impaired spatial and numerical cognition (Bellugi et al., 1994; Donnai & Karmiloff-Smith, 2000). Impaired dorsal versus intact ventral pathways in the brain have also been argued to explain some of the visuospatial problems encountered in WS (Atkinson et al., 2001).

Modularity claims have also been made with respect to specific language impairment (SLI; Gopnik & Crago, 1991; Rice, 2002; van der Lely, 1997) and developmental prosopagnosia (Kress & Daum, 2003)—that is, when one aspect of the cognitive system is seriously impaired (grammar in the former case, face processing in the latter), with the remaining cognitive functions operating normally. Arguments for double dissociations in developmental disorders have also been marshalled, as the following claim from Pinker (1999) bears witness: "overall, the genetic double dissociation is striking. . . . The genes of one group of children [SLI] impair their grammar while sparing their intelligence; the genes of another group of children [WS] impair their intelligence while sparing their grammar" (p. 262).

When the logic of adult neurospsychology is used, these various developmental data and the interpretations thereof seem to suggest that the brain is strictly modular, with genetic disorders helping the scientist to discover that the content of modules is likely to be innately specified in our genetic make-up. We will now turn to arguments that challenge these claims.

A Neuroconstructivist Perspective

Let us reexamine the data presented above that have been used to bolster claims about the innate specification of language and other cognitive modules. We will first reconsider the case of Christopher, the so-called linguistic savant (Smith & Tsimpli, 1995). Is Christopher really the language machine that he is claimed to be? It is true that this young man has, over the years, taught himself a large number of languages to a surprising degree of proficiency. This has astonished researchers, given Christopher's low IQ (Smith & Tsimpli, 1995). However, using an IQ measure, rather than mental age, can be misleading (see the discussion in Karmiloff-Smith, 1998). This 30-year-old's nonverbal IQ indeed sounds very low when compari-

sons are made with his linguistic prowess, but in terms of mental age, Christopher reaches a nonverbal level of a 9-year-old. Moreover, unlike most normal individuals, he spends a very large percentage of his waking hours studying languages. Another critical factor is that, in the main, Christopher learns his languages through written media. In other words, Christopher is not an oral "linguistic savant"; he is capable of *reading*, and he has learned to do so in several different scripts. As impressive and interesting as the feats of what we might call this "language spotter" are, the fact that Christopher has a nonverbal mental age of 9 and can read fluently challenges the notion of an isolated oral language capacity that has developed independently of general intelligence. In my view, many 9-year-olds who spent so much of their time devoted almost exclusively to learning languages would reach comparable or even greater achievements. In other words, the innate specification of a language module developing independently of intelligence is not demonstrated by this particular (albeit fascinating) case.

As was mentioned earlier, several developmental disorders have been highlighted as presenting examples of a single deficit alongside a pattern of normality throughout the rest of the cognitive system. Such disorders have been used to sustain arguments for the innate modularity of the mind/brain, prespecified in our genetic makeup. However, the arguments that pure cases of single deficits exist in children, as they may appear to in adult neuropsychological patients (although this remains debatable), turn out to be very difficult to sustain.

For example, SLI is, by definition, considered to involve a single impairment in language, with the rest of the cognitive system operating normally. However, in recent years, disorders such as SLI have been shown to stem from lower level deficits and to be accompanied by numerous other subtle impairments in the hitherto presumed "intact" nonlinguistic domains, such as motor skills, numbers, and fine auditory processing (Benasich & Spitz, 1999; Bishop, 1997, 2002; Botting, 2005; Chiat, 2001; Norbury, Bishop, & Biscoe, 2002). Moreover, even when performance IQ falls within the normal range, the IQ of the SLI individual is often significantly lower than that of his siblings (Botting, 2005), pointing to a more general impairment despite so-called "normal" scores. Furthermore, longitudinal studies have shown that the pattern of deficits and normal scores in developmental disorders change quite considerably over developmental time, with drops in nonverbal scores of some 10–20 points (Conti-Ramsden & Botting, 1999; Howlin, Mawhood, & Rutter, 2000; Krassowski & Plante, 1997; Mawhood, Howlin, & Rutter, 2000). All of these data highlight the fact that, contrary to the nativist views discussed earlier, developmental disorders rarely display a neat juxtaposition of intact and impaired modules onto which one can map specific genes.

In my view, instead of invoking the preformation of cognitive-level modules, they are more likely, in the adult, to be the product of the dynamics of ontogenetic development. The fact that some adult neuropsychological patient data seem to suggest a modular brain is actually orthogonal to the issue of whether modules are innately specified, because, as has been stated, modules could be the result of ontogenesis over developmental time, not its starting point. Hence, to address these questions, a developmental perspective is essential.

In our studies of WS, we examined in detail the domain of face processing (Karmiloff-Smith et al., 2004), since this is a domain for which many authors have claimed intactness of the functioning of a face-processing module in WS, analogous to that found in typical development (e.g., Bellugi et al., 1994; Rossen, Jones, Wang, & Klima, 1995; Tager-Flusberg et al., 2003). We challenged this conclusion on several fronts. First, although people with WS display proficient *behavior* that falls in the normal range on some standardized face-processing tasks, such as the Benton (Rossen et al., 1995) or the Rivermead (Udwin & Yule, 1991), the means by which they achieve this success turns out to be different from that used by controls. Whereas the controls in our studies used configural processing (i.e., the ability to differentiate faces on the basis of sensitivity to second-order processing of the spatial distances among internal features), the WS group used featural processing (i.e., the ability to differentiate faces on the basis of individual details, such as the eyes, nose, mouth, chin, or cheeks) or holistic processing (i.e., the ability to glue together facial features and hairline into a gestalt, without conserving the spatial distances between features; Maurer, LeGrand, & Mondloch, 2002; Tanaka & Farah, 1993), just as they do for nonface spatial stimuli (Karmiloff-Smith et al., 2004; see also Deruelle, Mancini, Livet, Cassé-Perrot, & de Schonen, 1999; Karmiloff-Smith, 1998). Moreover, our brain-imaging studies of WS face processing and spatial stimuli, using high-density event-related potentials, point to similar conclusions (Grice et al., 2003; Grice et al., 2001). When we compared brain processing of human faces, monkey faces, and cars, we found that typical controls displayed a strong N170 (the early electrophysiological marker of face processing) for human and monkey faces, with no such increase in amplitude for the brain processing of cars. By contrast, the adolescents and adults with WS showed a very reduced N170, which was similar for all three types of stimuli. In other words, for the WS group, faces were processed in much the same way as cars. In other words, the WS brain did not display specialization of function. Thus, the behavioral scores on some standardized tasks for which people with WS fall "in the normal range" seem to be sustained by cognitive and brain processes different from those of controls. It is not the case, then, that individuals with WS have an intact face-processing module and an impaired spatial module; both domains are impaired in similar ways. Rather, individuals with WS seem to fail to modularize their face processing over developmental time. So, a lack of modularization in developmental disorders may reveal *common initial processes* across domains, before each domain would, in the normal case, have become increasingly segregated and modularized over time.

Multiple Contributions to the Onset of Language

Although some have argued for direct mappings between specific genes and language (e.g., Gopnik & Crago, 1991; Pinker, 1999), here, we argue for multiple interacting sources of early language acquisition. So let's examine earlier claims regarding an intact language module in WS (Bellugi et al., 1994; Pinker, 1994, 1999; Smith, 1999). First, an abundance of empirical studies from numerous laboratories across the world now challenge these claims in respect of all aspects of WS language—for example, the lexicon (Jarrold, Hartley, Phillips, & Baddeley, 2000; Temple, Almazan, & Sherwood, 2002), morpho-syntax (Grant, Valian, & Karmiloff-Smith, 2002; Karmiloff-Smith et al., 1997; Thomas et al., 2001; Volterra, Capirci, & Caselli, 2001), phonology (Grant et al., 1997), and pragmatics (Laws & Bishop, 2004). Brain imaging of language processing again points to atypical processing in WS (Neville, Mills, & Bellugi, 1994). Moreover, despite the superficially fluent language peppered with erudite-sounding words of adolescents and adults with WS, language onset in this clinical population is very late, often not until the 5th or 6th year (Singer Harris, Bellugi, Bates, Jones, & Rossen, 1997).

Why is WS language so delayed? Is this due to a late-maturing module? Or is there a *developmental* explanation? In my view, the roots of the delay reside in deficits in multiple earlier processes to which multiple genes must contribute. For example, infants and toddlers with WS are extremely delayed in hand movements and babbling (Masataka, 2001), as well as in segmenting the speech stream (Nazzi, Paterson, & Karmiloff-Smith, 2003), a capacity seen as early as 8 months in typically developing infants. Second, unlike typical controls, toddlers and young children with WS rely more on perceptual cues than on linguistic labels when identifying new objects (Nazzi, Gopnik, & Karmiloff-Smith, 2005). Third, early categorization abilities in WS are impaired (Nazzi & Karmiloff-Smith, 2002), and exhaustive sorting follows word onset, rather than preceding it, as is the normal case (Mervis & Bertrand, 1997). Fourth, pointing is also atypical in WS toddlers. Whereas in typical development, referential pointing precedes the onset of language, in WS this order is reversed (Mervis & Bertrand, 1997). Moreover, our recent studies revealed that early on and unlike their peers, WS toddlers do not use or follow eye gaze for referential communication and do not understand the referential function of pointing (Laing et al., 2002). Finally, in the normal case, young children's comprehension outstrips their level of production. This clear-cut asymmetry does not hold for WS (Paterson, Brown, Gsödl, Johnson, & Karmiloff-Smith, 1999). In sum, many different aspects of communication show an early, unusual pattern in WS, jointly contributing in very complex ways to the explanation of the late onset of language.

However, an even earlier deficit outside the domain of language may offer a compelling explanation of some of these early deficits: atypical eye movement planning. In a study of saccadic planning in infants and toddlers with WS and Down syndrome (DS), in comparison with mental age and chronological age matched controls, we found that although children with DS resembled the controls, apart from being somewhat slower, the infants and toddlers with WS displayed a range of impairments (Brown et al., 2003). Some stayed fixated on one stimulus without moving their eyes at all, whereas others made a single eye movement but failed to make the two saccades made by the controls and the infants with DS. For the infants with WS who did make a double saccadic movement, two errors appeared: Either they failed to update their retinal image after the first eye movement and ended up in the wrong location after their second saccade (the retinocentric error), or they summated the two saccades before moving, thus making the vector summation error typical of normal 2-month-olds. In other words, making eye movements to explore the environment, as well as to follow another's eye gaze and pointing gestures, is atypical in infants and toddlers with WS. Thus, early visuospatial deficits in the WS developmental trajectory *outside the domain of language* can have cascading effects over developmental time on several *emerging* higher level linguistic and cognitive domains. The fact that domains are highly interrelated early in brain development (Huttenlocher, 2002; Johnson, 2001; Neville, 2006) turns out to play a critical role in the formation of more general, albeit sometimes subtle, deficits in later development.

Is Language Specified in Our Genes?

Because of our differences from other species, human language has been the domain most consistently claimed to be specified in our genes. Leading linguists and psycholinguists, such as Wexler and Pinker, have consistently asserted that language is an innately specified endowment of our biology, as the following quotations bear witness (italics added): "It is uncontroversial that the development [of universal grammar] is essentially guided by a biological, *genetically-determined program*" (Wexler, 1996); "The mind is likely to contain *blueprints for grammatical rules* . . . and a *special set of genes* that help wire it in place" (Pinker, 1994).

The excitement over such claims was recently reinforced with the revelation of a specific genetic mutation in a family pedigree with speech and language deficits. The now well-known KE family was genotyped across several generations. It was found that affected family members had a point mutation on one copy of the *FOXP2* gene on chromosome 7 that encodes a protein of 715 amino acids belonging to the forkhead class of transcription factors. The point mutation was not found in family members with normal language abilities (Fisher, Vargha-Khadem, Watkins, Monaco, & Pembrey, 1998; Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001). Although the geneticists remained cautious about the conclusions to be drawn from these cases, some linguists were quick to claim that a gene directly implicated in speech and language had been identified (Gopnik & Crago, 1991), a seemingly exciting discovery that Pinker claimed marked the "dawn of cogni-

tive genetics" (Pinker, 2001). One implication from such a claim is that we may soon be able to map more or less directly from genes and their protein products to the cognitive level, something that in my view is highly unlikely. Moreover, it turns out that the problems of the KE family are not specific to speech and language (Alcock, Passingham, Watkins, & Vargha-Khadem, 2000a, 2000b; Vargha-Khadem et al., 1998). Family members with the mutation display impairments in multiple domains, such as fine motor control, gait, oro-facial movement, perception, and production of rhythm, all of which may subsequently impact on speech and language from infancy onward. The KE family's problems may be more overtly obvious in speech and language in the phenotypic outcome, but this does not mean that they were originally rooted only in speech and language modules. The origins may have been at a much lower level in the development of the motor system—that is, in the learning of skilled coordination of rapid movement sequences and their timing.

Not only is the genetic origin of the language deficit in the KE family far more indirect than is implied in the original claims (Gopnik & Crago, 1991; Pinker, 2001), but also this gene is likely to be a very tiny and even nonnecessary contributor to impaired linguistic outcome. Indeed, different laboratories have genotyped (for the *FOXP2* allelic mutation) hundreds of children selected for their low language scores (Meaburn, Dale, & Craig, 2002; Newbury et al., 2002). Not a single individual was found to have the *FOXP2* mutation, despite all having serious language deficits. This again points to the premature nature of claims about the discovery of a single gene thought to be implicated in a deficit in the cognitive-level outcome of development. If *FOXP2* is implicated in language, its contribution is likely to be minute and extremely indirect, in interaction with multiple other genes.

Variable Gene Mapping

One approach to mapping genotype/phenotype relations is to find patients who, for instance, have a deletion within the WS critical region (WSCR), but one that is smaller and contains fewer genes. Could this be a way to map single genes to a cognitive-level outcome? For example, WS has been used to support claims of direct gene–behavior mappings. In identifying patients with a small deletion within the WSCR, researchers have attempted to delineate the functions of specific genes. Families have been identified, some of whose members had a small deletion of two genes within the WSCR (Elastin and Limkinase1 [*LIMK1*]), with other family members having no such deletion. Elastin is a gene implicated in the building of connective tissue throughout the body, particularly the arterial walls, and is likely to be linked to the supravalvular aortic stenoses suffered by people with WS and by these small deletion patients (Curran et al., 1993; Frangiskakis et al., 1996). *LIMK1* is a protein kinase gene, expressed in the developing brain (Proschel, Blouin, Gutowski, Ludwig, & Noble, 1995; Tassabehji et al., 1996). Interestingly, it turned out that family members with the *LIMK1* deletion displayed spatial deficits similar to those in people with WS, whereas family members without the deletion had no spatial problems (Frangiskakis et al., 1996). From these data, the Frangiskakis group concluded that the half dosage of *LIMK1* plays a vital role in contributing to the visuospatial constructive cognition deficits that occur in WS.

However, other patients with similar or even larger deletions in the WSCR, including *LIMK1*, failed to corroborate these conclusions (Karmiloff-Smith, Grant, et al., 2003; Tassabehji et al., 1999). The study with Tassabehji and collaborators included four patients with centromeric deletions, including Elastin and *LIMK1*, and yet none showed an imbalance between their language and spatial scores. Two of these patients had normal intelligence, one had intelligence well above normal, and one was in the lower end of the normal range. Yet none displayed a specific spatial deficit. An in-depth follow-up study of two of the patients, using nearly 20 different neuropsychological tests of spatial and navigational cognition (Gray, Karmiloff-Smith, Furnell, & Tassabehji, 2006; Karmiloff-Smith et al., 2006), showed no deficits whatsoever in these patients, despite their half dosage of *LIMK1*. Yet again, it turns out that the claims of a direct link between spatial cognition and a specific gene, *LIMK1* (Frangiskakis et al., 1996), were premature and based, in my view, on a false assumption—that is, that a single gene will turn out to be linkable to a specific cognitive-level outcome. If *LIMK1* plays a role, it is probably in low-level processes, interacting with other genes at the telemetric end of the typical WS deletion, ultimately to *result* over developmental time in the spatial cognition deficit.

Animal Models of Genotype/Phenotype Mapping

The fact that a mouse knockout model came to conclusions similar to those of the Frangiskakis group regarding *LIMK1*-deletion patients seems to make their claims plausible after all. Chromosome 5G on the mouse genome conserves all of the WS-relevant genes on chromosome 7 and their order (albeit reversed). So the mouse is a potentially excellent model of the WS human case. Meng et al. (2002) created a single knockout of *LIMK1* and demonstrated serious spatial learning problems in the mouse's behavior in the Morris water maze. Thus, both the mouse model and the small-deletion patient data seemed to point to the same conclusion: Deletion of the *LIMK1* gene is directly linked to the impaired visuospatial module in WS. But we need to treat the animal data with some caution when generalizing to the human case. Although the spatial deficit found in the mouse's behavior in the Morris water maze seems to replicate the spatial deficit found in individuals with WS, who also all have a deletion of *LIMK1*, there are several problems with this model. First, it is a single gene knockout, whereas WS involves the deletion of some 28 contiguous genes that may interact with one another. Second, several of these genes are transcription factors. Third, *LIMK1* does not target a specific brain region responsible for spatial cognition, in the human case, but is expressed widely across the brain early in embryonic development. Its protein products are thought to con-

tribute to something far more general developmentally: dendritic spine growth and synaptic regulation across the brain. Even if the function of *LIMK1* were the same in human development as in the mouse, it is highly unlikely to target a spatial module in the parietal cortex; rather, it is likely to have subtle widespread effects. Moreover, although the mouse–human comparison stressed the spatial deficits found in both species, other impairments were found in the knockout mouse that do not occur in WS, and vice versa. Finally, animal models of single knockouts of many other genes than *LIMK1* turn out also to give rise to spatial deficits in the Morris maze. This leads me to conclude that the maze measures something like *general retardation* in the mouse, and not a specific spatial impairment. Moreover, we are comparing navigational skills in the mouse with table-top spatial skills in the human case for which it is unnecessary to represent one's position in space. So, the generalization from animal models to the human case must always be treated with extreme prudence.

All the above provisos hold with respect to other animal models, such as the chimpanzee (Enard et al., 2002; International Chimpanzee Chromosome 22 Consortium, 2004) or the bird (Haesler et al., 2004). With the discovery of the *FOXP2* mutation in some humans and its potential relationship to speech and language, researchers moved to genotyping our closest cousin, the ape. The *FOXP2* proteins of the chimpanzee, gorilla, and rhesus macaque are identical to one another. *FOXP2* is an extremely conserved gene across mammalian species and has shown no changes in the chimp lineage since it separated from the human lineage some 4–6 million years ago. By contrast, it acquired two amino acid changes in the human lineage, one of which is likely to be functional, dated to some 200,000 years ago (Enard et al., 2002). Not surprisingly, this was an exciting discovery, since the timing of the protein changes in the human lineage roughly coincides with estimates of when language started to emerge in our species (Botha, 2004; Hurford, Studdert-Kennedy, & Knight, 1998; Newmeyer, 2004). Put together with the data from the KE family, it is a small step to then claim that the change in a single base pair in *FOXP2*, altering protein synthesis, must be a direct contributor to the evolution of human language. Likewise, outside the field of language, a number of authors have made sweeping claims about the evolutionary underpinnings of cognitive-level modules (e.g., Barkow et al., 1992; Duchaine et al., 2001; Pinker, 1997).

First, it should be recalled that *FOXP2* is a transcription factor; its expression affects many other genes. Second, over time, the expression of *FOXP2* is increasingly refined to the cerebellum and motor skills. Third, although the evidence from primate comparisons of *FOXP2* is, at first blush, very exciting, more recent research on *FOXP2* expression in birds, which is very similar to *FOXP2* expression in humans, tends to make the original claims about the relation of *FOXP2* to language seem premature. Researchers compared songbirds that learn their vocalization (e.g., zebra finches, canaries, etc.) with songbirds that produce their vocalization without learning (Haesler et al., 2004). The findings were revealing. In the avian learners, *FOXP2* had greater expression in the equivalent of the basal ganglia during phases of song *learning* than during song production. The scientists concluded that *FOXP2* expression is associated with the learning of skilled coordination of rapid movement sequences and their timing—that is, that *FOXP2* expression was an important contributor to *vocal plasticity* (Haesler et al., 2004). Such findings tend to challenge the notion that evolution has created increasingly complex genes that specify the content of cognitive-level modules. Rather, evolution may have opted for genetic changes that contribute to *increased plasticity for learning*. Of course, the claim that *FOXP2* is found in birds and contributes to vocal plasticity does not automatically contradict the finding of rapid recent evolution of *FOXP2* in primates or the claim that the allelic mutation may be a contributor to human language. What it does challenge is any notion that the *FOXP2* mutation gave rise to a "grammar gene" (Gopnik & Crago, 1991). Moreover, as I have repeatedly argued, one cannot simply take for granted homology of function or identical timing of genetic expression of the same gene across different species (Karmiloff-Smith, Scerif, & Thomas, 2002). It has to be demonstrated empirically. Nonetheless, the fact that *FOXP2* in birds is not a gene that encodes a specific bird song but, rather, one that facilitates the ability to *learn* highlights the need for extreme caution in assuming that *FOXP2* is a gene contributing directly to language in humans.

The Future

A vital issue for future consideration is the fact that we now know, from studies of normal development, that the microcircuitry of the brain develops massively during the postnatal months, followed by a period of pruning when nonused connections are weakened and used connection weights are strengthened. Yet we know very little about atypical development in this respect. What is this process like in infants with developmental disorders? Does pruning fail to occur, due to the lack of progressive modularization? Do brain areas in atypical development remain more highly interconnected over time, failing to progressively modularize, than is the case for typical development? Is this the same across different disorders, or does each syndrome display its own particular brain signature? In sum, there is an urgent need for longitudinal cross-syndrome, cross-domain studies of infant behavior and of progressive brain development in a variety of different developmental disorders.

How can we achieve a coherent, nonmodular developmental explanation of the contrasting profiles found in different developmental disorders? How do genetic mutations alter the way in which brains develop over time? Finally, it is also vital for scientists to understand how having a developmental disorder changes the social and physical environment in which a child is raised (Cicchetti, 2002; Karmiloff-Smith & Thomas, 2005). Parental expectations are altered simultaneously with the knowledge

of a child's condition, and however subtle these changes may be, they impact on the learning situation and gene expression through the interaction between environment and child over developmental time.

Perhaps researchers should be turning some questions on their heads. Instead of searching only for dissociations and attempting to map specific genes to them, we should now focus on associations between disorders. Let's reflect on the following: WS is caused by a deletion of some 28 genes on one copy of chromosome 7; DS is caused by an additional whole chromosome 21; Fragile X is caused by a mutation of a single gene on the X chromosome; velocardiofacial syndrome (or diGeorge syndrome) is caused by a large deletion on chromosome 22. Yet all four syndromes display both delay and deviance, mental retardation, gross and/or fine motor deficits, impaired sleep patterns, memory deficits, number impairments, and often hyperactivity. Three of them show better language skills than spatial skills. Clearly, we cannot invoke a single genetic origin to these deficits, so how do we explain the associations between genetic disorders with such different causes? These are questions that are rarely tackled, but they will, in my view, come to be as important for our understanding of the complexities of gene expression as the search for dissociations.

In conclusion, we have seen that simple, direct mappings between genes and cognitive-level outcomes are not sustainable. In fact, genes are likely to contribute to much more general constraints, such as developmental timing, neuronal migration, neuronal type/size/density/orientation, myelination, lamination, ratio of gray matter to white matter, firing thresholds, neurotransmitter differences, and so forth (Bates & Roe, 2001; Elman et al., 1996), any or all of which may be atypical in developmental disorders. Any of these factors may turn out to be domain relevant—that is, more appropriate for one domain of processing than for others. Over time in normal development, such domain relevance can, with repeated processing, become domain specific and modularized (Karmiloff-Smith, 1992). But if deficient early on, domain specificity of processing may not emerge developmentally. Moreover, once one thinks from a truly *developmental* neuroconstructivist perspective, it is easy to imagine how even a tiny asynchrony or impairment early on in development can have a huge, cascading impact on the phenotypic outcome.

This article has strongly argued that it is theoretically and empirically misleading to view genetic developmental disorders as illustrations of a juxtaposition of *intact* versus *impaired* modules onto which specific genes may be mapped. Rather, the study of these syndromes points to altered constraints on neural plasticity in a *developing* organism, often affecting plasticity itself, involving an extremely tortuous route between the complexities of gene expression and the phenotypic outcome.

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