Developmental Psychology
2009, Vol. 45, No. 1, 56–63

0012-1649/09/$12.00 DOI: 10.1037/a0014506

Nativism Versus Neuroconstructivism: Rethinking the Study of Developmental Disorders

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This article argues that one dominant position in psychology, linguistics, neuroscience, and philosophy about how genetic disorders point to the innate specification of dissociated modules in the human brain should be replaced by a dynamic, neuroconstructivist approach in which genes, brain, cognition, and environment interact multidirectionally. The article challenges current thinking about a series of questions: (a) Do significantly better scores in one domain necessarily indicate an intact module? (b) What do scores in the normal range suggest? (c) What is wrong with mental-age matching? (d) Why is the notion of an intact module unlikely? (e) Do developmental disorders suggest associations rather than dissociations? (f) Is the environment the same for atypically developing individuals? The article concludes by examining the implications of taking a neuroconstructivist approach and by arguing that human intelligence is not a state (i.e., not a collection of static, built-in modules that can be intact or impaired) but a process (i.e., the emergent property over developmental time of dynamic, multidirectional interactions between genes, brain, cognition, behavior, and environment) with domain-specific outcomes impossible without the process of development.

Keywords: nativism, neuroconstructivism, intact/preserved modules, emergent intelligence, gene/brain/cognition/behavior/environment interactions

In this article, it is argued that one of the hitherto dominant positions in the fields of psychology, linguistics, neuroscience, and philosophy concerning the way in which genetic disorders inform the scientist about the innate specification of dissociated modules in the human brain should be replaced by a dynamic, neuroconstructivist approach in which genes, brain, cognition, and environment interact multidirectionally. The following quotations illustrate with respect to autism these two very different approaches (tagged as [a] nativist and [b] neuroconstructivist):

> [a] Autism involves a damaged theory-of-mind module (Leslie, 1992, p. 21).
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> [a] Results implicate the orbito-frontal cortex as the basis of this ability (p. 640) . . . specifically damaged in autism (pp. 642–643; Baron-Cohen et al., 1994).
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> [b] Autism affects the interconnectivity among and within various cognitive systems . . . . In autism, functional brain development goes awry such that there is increased intra-regional specialization and less inter-regional interaction (Carpenter et al., 2001, p. 373).
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> [b] . . . the crucial role of unbalanced excitatory-inhibitory networks . . . complex pathogenetic pathways . . . leading to Autism Spectrum Disorder through altered neuronal morphology, synaptogenesis and cell migration (Persico & Bourgeron, 2006, p. 349).

Similar fundamental differences in approach exist with respect another disorder, Williams syndrome (WS), as the following quotations bear witness:

> [a] Overall, the genetic double dissociation is striking . . . . The genes of one group of children [specific language impairment] impair their grammar while sparing their intelligence; the genes of another group of children [WS] impair their intelligence while sparing their grammar (Pinker, 1999, p. 262).
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> [a] For instance, children with WS have a barely measurable general intelligence and require constant parental care, yet they have an exquisite mastery of syntax and vocabulary. They are, however, unable to understand even the most immediate implications of their admirably constructed sentences (Piattelli-Palmarini, 2001, p. 887).
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> [b] We argue that rather than being the paradigm case for the independence of language from cognition, WS provides strong evidence of the interdependence of many aspects of language and cognition (Mervis & Becerra, 2007, p. 3).
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> [b] In sum, brain volume, brain anatomy, brain chemistry, hemispheric asymmetry, and the temporal patterns of brain activity are all atypical in people with WS. How could the resulting system be described as a normal brain with parts intact and parts impaired, as the popular view holds? Rather, the brains of infants with WS develop differently from the outset, which has subtle, widespread repercussions (Karmiloff-Smith, 1998, p. 393).

Where do arguments for innate modularity stem from? There are at least four sources. First, they draw on cases from adult neuropsychology where some patients whose brains had previously developed in a typical way subsequently suffer a brain trauma and end up with a pattern of relatively dissociated impairments (e.g., cases of agrammatism, prospagnosia, or agnosia). The second source can be found in one version of evolutionary psychology, which maintains that the human brain has evolved into the equivalent of a Swiss army knife in which each innately specified

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module is exquisitely adapted for each specific, independent function (Barkow, Cosmides, & Tooby, 1992; Duchaine, Cosmides, & Tooby, 2001). (The analogy ignores the fact that most users of the Swiss army knife actually employ for all purposes only a few of the numerous special-purpose tools.) Third, surprisingly early competences discovered in young infants have been hailed also as a demonstration of innately specified, core knowledge/core principles (e.g., Gelman & Butterworth, 2005; Pinker, 1999; Spelke & Kinzler, 2007). Fourth, genetic disorders, that present with uneven cognitive profiles displaying a juxtaposition of scores "in the normal range" in one or more domains alongside serious deficiencies in others are claimed to illustrate the dissociation of general intelligence from specific domains like grammar, number, face processing, and the like, as many of the above [a] quotations illustrate. So, why do these data and arguments turn out to be less compelling than they at first blush seem?

Do Significantly Better Scores in One Domain Necessarily Indicate an Intact/Preserved Module?

There is a worrying tendency in the developmental disorders literature (as well as in adult neuropsychology) to slip from relative comparisons to absolute ones. So, for instance, a clinical group may score significantly better in Domain A than in Domain B. This is then interpreted as Domain A being intact/preserved and Domain B being impaired (e.g., Tager-Flusberg, Boshart, & Baron-Cohen, 1998). But camouflaged beneath such interpretations is the fact that the scores in both domains may actually be well below those of chronological-age-matched controls. In other words, both domains are impaired; it is simply that, because of its processing demands, one domain is more impaired than the other, not that one is impaired and the other intact, or even, as many authors write *relatively intact*—a theoretically loaded expression with which users want to have their modularity cake and eat it. It is thus critical when reading arguments in favor of innately specified modularity to ensure that the reporting of findings has not slipped from relative comparisons to absolute ones. But, what if scores in proficient domains are actually in the normal range?

What Do Scores in the Normal Range Tell Researchers?

The study of genetic disorders has indeed identified syndromes in which certain domains of relative proficiency coexist with others of more serious impairment. This frequently leads to a research strategy in which the domains of proficiency are merely assessed by standardized tests, with little further exploration once scores fall in the normal range. Subsequently all the research effort is concentrated on the domain(s) of serious deficit. But, ascertaining that scores fall in the normal range can ignore several crucial factors that raise a number of challenging questions: (a) Are the cognitive processes underlying the proficient overt behavior the same as those used by typical controls? (b) Are the brain networks underlying the proficient behavior the same as those used by typical controls? (c) Given the potential of siblings within the same family, are the scores of the atypical sibling closer to the level expected for the family, even though they are within the normal range as well as being significantly higher than the average for the disorder? (d) Did the individual display impairment earlier in development, which has been compensated for subsequently via an atypical trajectory? (e) Is the standardized test sensitive enough to identify subtle impairments that may remain after compensation? And, in general, has the actual trajectory of development been taken into account?

The first two questions can be addressed by work by my colleagues and me on the genetic disorder, WS (see Donnai & Karmiloff-Smith, 2000, and Meyer-Lindenberg et al., 2005, for details of the WS genotype and phenotype). Research on face processing is a relevant example. Individuals with WS score in the normal range on the Benton Test of Face Recognition (Bellugi, Wang, & Jernigan, 1994) as well as on the Rivermead Test of Face Memory (Udwin & Yule, 1991). These findings have been replicated by several labs worldwide (Annaz, Karmiloff-Smith, & Thomas, in press; Bellugi et al., 1994; Grice et al., 2003; Karmiloff-Smith et al., 2004; Rossen, Jones, Wang, & Klima, 1996; Udwin & Yule, 1991). So, the behavioral facts are not challenged. However, the cognitive and brain processes underlying this proficient behavior are hotly debated. For some researchers, face processing in WS is claimed to be no different from controls without learning difficulties (e.g., Rossen et al., 1996; Tager-Flusberg, Pless-Skewer, Faja, & Joseph, 2003). For others, face processing in WS differs from controls at both the cognitive and brain levels (Deruelle, Mancini, Livet, Casse-Perrot, & de Schonen, 1999; Grice et al., 2003; Karmiloff-Smith et al., 2004; Mills et al., 2000; Neville, Mills, & Bellugi, 1994). In fact, researcher by my colleagues and me on electrophysiological studies of face processing in the WS brain have shown that even by adulthood, individuals with WS fail to display the gradual localization and specialization of function (Grice et al., 2003; Karmiloff-Smith et al., 2004) that is the progressive brain signature of normal development (Cohen-Kadosh & Johnson, 2007; de Haan, Humphreys, & Johnson, 2002; Johnson, 2001). So, scores in the normal range do not necessarily entail normal developmental trajectories.

With respect to question (c), in-depth studies are urgently needed of individual differences in the environment and genetic potential within the families of individuals with a disorder of known genetic origin. Take the following hypothetical example. If all of the siblings in a family are particularly talented and eloquent language is continually used in the family environment, with siblings' verbal IQs being around 140–160, and if the atypical sibling has a verbal IQ of 89 and thus falls in the normal range, is that individual more impaired than one whose verbal IQ is also 89 but whose family verbal IQ average is 98? Some of my participants with WS, for instance, reach the upper end of the normal range on vocabulary tests, whereas others are more delayed. However, responses to a preliminary questionnaire indicate that in these more high-functioning cases other family members are often well above the normal range, suggesting a probable impairment in those individuals with WS despite their normal scores. Moreover, studies of other disorders (e.g., specific language impairment [SLI]) already speak to the importance of the environmental and genetic factors. Meta-analyses of research on SLI reveals that although individuals may score in the normal range for nonverbal intelligence, they are sometimes as much as two standard deviations below their potential compared to their siblings, with their levels fluctuating considerably over developmental time (Botting, 2004; Chiat, 2001; Conti-Ramsden & Botting, 1999). This suggests that although language is indeed more seriously impaired in SLI,

nonverbal intelligence may also be impaired, albeit to a lesser degree. This raises the possibility of early, more general, low-level processing deficits that affect several domains but to differing degrees and at different developmental times (Benasich & Spitz, 1999; Karmiloff-Smith, 1998). Even if this turns out to be an erroneous assumption for SLI, such a possibility must always be explored when uneven profiles present themselves, rather than using an uneven profile automatically to assume the existence in the brain of independently functioning, domain-specific modules.

Question (d) underlines the crucial role of developmental time in developmental disorders (Karmiloff-Smith, 1992, 1998, 2007). Paradoxically, numerous studies of development (typical or atypical) are not developmental at all, because studying children by no means guarantees a developmental approach (Karmiloff-Smith, 1992, 1998). And one can study adults developmentally (Cornish, 2008). The truly developmental, neuroconstructivist perspective embraces a developmental way of thinking, irrespective of the age of the population studied; even studies of infants can be nondevelopmental. In my view, to understand developmental outcomes, it is vital to identify full developmental trajectories, to assess how progressive change occurs from infancy onwards, and how parts of the developing system may interact with other parts differently at different times across ontogenesis. A process that is vital, say, at Time 2 may no longer play a role at Time 5. Yet its delay at Time 2 may have been crucial to a healthy developmental trajectory and outcome. Indeed, developmental timing is among the most important of factors that need to be taken into account when endeavoring to understand human development, particularly in the atypical case. Whatever the case, in a developmental disorder when scores in the normal range are found in some domains, it is vital to probe the cognitive and brain processes that underlie such efficient behavior and not merely concentrate on domains of deficit. Sometimes the discovery of a different trajectory in a proficient domain (e.g., a featural strategy for face processing) can provide clues to the mechanisms that have gone awry in an impaired domain (e.g., impairments in numerical magnitude comparisons; van Herwegen, Ansari, Xu, & Karmiloff-Smith, 2008).

Finally, Question (e) stresses how vital it is to carry out in-depth task analyses of standardized tests before concluding that participants' scores that fall in the normal range are the same as those of controls without learning difficulties. Can the test in question really detect subtle impairments? Often the granularity of the test is rather coarse and at best provides the researcher with a ballpark idea of how well the individual might be performing compared to the general levels of typical controls. If compensation has taken place over developmental time, it is unlikely that any remaining subtle deficits will be detected. Moreover, standardized tests rarely differentiate between overt behavior and underlying cognitive or brain processes. Hypothesis-driven neurocognitive experiments must be devised to address such questions.

What Is Wrong With Mental-Age Matching?

Of course, if the researcher is of a nativist persuasion, then paradoxically she or he should not need to match on mental age at all, because this implies that general intelligence does play a role in specific domains and that this role must be neutralized by the matching procedure.

Cross-syndrome comparisons at the behavioral, cognitive, and brain levels can involve very large numbers of mental-age-matched controls, where decisions as to the measures on which to carry out the matching can favor one syndrome and disadvantage another because of their differing, uneven phenotypic profiles. For instance, if Syndrome A shows greater strength in language and Syndrome B greater strength in spatial cognition, then matching on either verbal or nonverbal scores could radically change the results and conclusions drawn therefrom. For this reason, it is vital to develop new approaches in which full, task-specific developmental trajectories are first built of the typical developmental profile (Annaz et al., in press; Ansari, Donlan & Karmiloff-Smith, 2007; Ansari et al., 2003; Ansari & Karmiloff-Smith, 2002; Cornish, Scerif & Karmiloff-Smith, 2007; Karmiloff-Smith, 1998; Karmiloff-Smith et al., 2004; Scerif, Cornish, Wilding, Driver, & Karmiloff-Smith, 2004; see particularly, Thomas et al., in press). A major advantage of the trajectory approach is that it is theory neutral compared with all forms of mental-age or chronological-age matching. Once a typical, task-specific trajectory is established, each of the atypical participants can be plotted on it and, via regression analyses, it is then possible to assess whether they display deviance or delay, or both, for that task. One can then carry out comparisons across tasks, across domains, and/or across syndromes (for full details, see Thomas et al., in press). The trajectory method also makes it possible to differentiate between different forms of delay even when the amount of delay is the same across domains within syndromes and/or across syndromes (Annaz et al., in press; Thomas et al., in press). To build a full developmental trajectory, it is of course important to devise processing tasks that can be used with very young children (infants and toddlers) through to older children, adolescents, and adults or, at the very least, tasks that yield clear developmental changes across a fairly wide age range, avoiding floor and ceiling effects. The developmental trajectory approach makes it possible to carry out cross-syndrome comparisons more directly than has been the case in the past and to do so at multiple levels of analysis (Johnson, Halit, Grice, & Karmiloff-Smith, 2002; Karmiloff-Smith, 1998, 2006; Karmiloff-Smith, Scerif, & Thomas, 2002; Karmiloff-Smith, Scerif, & Ansari, 2003; Thomas et al., in press).

Why Is the Notion of an Intact/Preserved Module Unlikely?

The very notion of intactness/preservation has a static flavor and implies genetic determination, as if states in the brain were hard wired, unchanging, and unaffected by developmental or environmental factors. A different, neuroconstructivist view is to consider the brain as a self-structuring, dynamically changing organ over developmental time as a function of multiple interactions at multiple levels, including gene expression (e.g., Casey, 2002; Johnson, 2001). Research on birds and mammals eloquently illustrates this point. Extensive evidence from studies of the neural and epigenetic consequences of song listening and song production in passerine birds (Bolhuis, Zijlstra, den Boer-Visser, & van der Zee, 2000) shows how gene expression changes over developmental time and may be significantly more important during learning than during final production. Rather than something fixed and predetermined, gene expression in the birds turned out to be a function of how many elements the bird copied from its tutor. A second example

comes from early mammalian development and also underlines the potential role of the environment in shaping patterns of gene expression (Kaffman & Meaney, 2007). These authors have studied brain development in rodent pups and have elegantly traced how differences in maternal behavior influence patterns of gene expression, which have lifelong effects on behavior. They showed that rather than thinking of gene expression as preprogrammed, differences in the amount of postnatal pup grooming and stroking change the chemistry of the DNA of certain genes involved in the body's responses to stress. These kinds of dynamic environment/gene relations may well be pervasive in mammalian brain development, including that of humans. In general, epigenesis is not deterministic under tight genetic control. Rather, as Gotlieb (2007) stressed, epigenesis is probabilistic and only under very broad genetic control.

Furthermore, what researchers now know about the dynamics of typical brain development points to major structural and functional changes across development that run counter to any arguments in favor of preserved versus impaired modules. First, the cortex starts out highly interconnected in the very young infant (Huttenlocher & Dabholkar, 1997; Huttenlocher & de Courten, 1987), and it is only very gradually over time that localization and specialization of brain function occur (Johnson, 2001). Second, the ratio of white matter to gray matter changes from infancy to later development (Giedd et al., 1999). Third, the thickness of fibre bundles in the corpus callosum between the two hemispheres is different in infancy compared to later development (Giedd et al., 1996). Fourth, studies of the electrophysiology of the brain in processing, say, faces or language reveal widespread activity across several regions of cortex in both hemispheres. It is only over developmental time that the electrophysiological activity becomes progressively fine tuned to predominantly one hemisphere (right hemisphere for faces; left hemisphere for grammar; de Haan et al., 2002; Johnson, 2001; Mills et al., 2000; Neville et al., 1994). These developmental examples clearly indicate that the brain is not a static entity. Indeed, cortical networks are not genetically predetermined or built in to be preserved or impaired in genetic disorders. Rather, they are the emergent outcome of progressively changing processes, which dynamically interact with one another and with environmental input over developmental time, ultimately to give rise to the structured adult brain. But this is not to imply that the neonate brain is a blank slate with no structure, as empiricists would claim. Nor does it imply that any part of the brain can process any and all inputs. On the contrary, neuroconstructivism maintains that the neonate cortex has some regional differentiation in terms of types of neuron, density of neurons, firing thresholds, and so forth. These differences are not domain specific aimed at the sole processing of proprietary inputs nor do they amount to domain-general constraints. Rather, they are domain relevant (i.e., different parts of the brain have small structural differences, which turn out to be more appropriate/relevant to certain kinds of processing over others). But initially, brain activity is widespread for processing all types of input and competition between regions gradually settles which domain-relevant circuits become domain specific over time. Emergent specialization of function (e.g., for faces) might well be viewed as the fine tuning of initially domain-relevant but coarsely coded systems (e.g., for visual patterns), but this is for visual patterns in general, not for faces in particular. The face specialization emerges from the interaction between the environment (huge numbers of face stimuli over time) and the initial visual processing constraints, not from a prespecified, dedicated face-processing module, as some would argue (e.g., Duchaine & Nakayama, 2006).

An eloquent illustration of how emergent specialization can occur comes from a computational model of the dorsal and ventral streams. A small difference in activation levels (equivalent to a difference in neuronal firing thresholds) between two streams sufficed, after competing to process identical inputs, to result in one stream ending up processing where objects were, and the other stream processing the features of objects. In other words, the *where* and *what* pathways were not built in with one only processing spatial information and the other only processing object featural information (O'Reilly & McClelland, 1992). Although both streams initially processed all inputs, an initial, tiny difference in the speed of activation levels was sufficient, then, to progressively give rise to gradual specialization of the where/what pathways. Without this domain-relevant difference in firing thresholds, both streams would have continued to process all inputs in a domain-general way. Moreover, one can then see how a small deficit in, say, the dorsal stream could not only impair spatial processing but could also interfere with the processing of low-spatial frequencies, which could in turn have a cascading subsequent effect on global and configural processing.

Cascading developmental effects of small perturbations are particularly relevant to researchers' understanding of human disorders. While there is still much to be learned about the details of the relations between gene expression and phenotypic outcomes, researchers' current knowledge does allow us to begin to make educated guesses, particularly where the genotype and phenotype have been studied in some depth. In WS, for instance, the genetic mutations are present from conception (due to a misalignment on one copy of Chromosome 7 during meiosis). A half-dosage of the protein that would normally have been expressed in the brain will be missing throughout cortical regions, so the effects of the deletion are likely to be widespread, not specific to a single region of cortex nor to a single purported module. But their deficient expression will affect different regions to greater or lesser degrees, depending on how vital their expression is to the most relevant processing in that region. In fact, developmental disorders will in my view only be explicable at very different levels from higher cognitive modules; rather, they will turn out to be explained in terms of perturbation in far more basic processes very early in development, such as a lack of sufficient pruning, or of overexuberant pruning, of differences in synaptogenesis, in the density or type of neurons, in differing firing thresholds, in poor signal-to-noise ratios in neuronal processing, 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), that is, 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 input (Elman et al., 1996). In other words, neuroconstructivism maintains that if the adult brain contains modules, then these emerge developmentally during the ontogenetic process of gradual modularization (Karmiloff-Smith, 1992, 1998). In this sense, domain-specific outcomes may not even be possible without the gradual process of development over time.

Do Developmental Disorders Point to Associations More Than to Dissociations?

The search for dissociations, and particularly for double dissociations, has been a primary goal of much of the work in adult neuropsychology and of many studies of genetic disorders in children. By contrast, it has been argued that to understand the nonstatic atypical processes of human development, the quest for double dissociations is both theoretically misconceived and empirically dubious (Karmiloff-Smith et al., 2003). In fact, developmental disorders often turn out to pinpoint associations across domains rather than dissociations (Bishop, 2002). In the case of WS, for instance, a featural processing bias has been documented across several different domains, such as face processing, spatial processing, auditory processing, and numerical processing (Bellugi et al., 1994; Elsabbagh, 2005; Karmiloff-Smith et al., 2004; Paterson, Brown, Gsodl, Johnson, & Karmiloff-Smith, 1999; van Herwegen et al., 2008). This featural bias points to early, common processing problems across several domains before, in the typically developing individual, they would have become naturally segregated and specialized over developmental time.

Where does this featural bias in WS originate? A study of gamma-band oscillatory activity in frontal cortex in the brain's reaction to viewing upright and inverted faces yielded unusual patterns in participants with WS and in those with autism (Grice et al., 2001), both of whom have been claimed to process featurally and to have difficulties in integrating/binding features into a configural whole. However, although gamma-band bursts were atypical in both clinical groups, the gamma-band patterns in the brains of individuals with WS and autism differed radically, suggesting that researchers need to rethink the notion of featural processing at the cognitive level. In the case of WS, our studies of infants suggest that featural processing may originate in sticky fixation and in the inability to plan rapid saccadic eye movements (Brown et al., 2003), which could result in difficulties in rapid configural processing. Interestingly, infants with Down syndrome do not have problems planning saccadic eye movements (Brown et al., 2003) and they do not suffer from a featural processing bias later in development. The cause of the featural bias in autism is likely to be different from that of WS, given the significant differences in gamma-band activity. In general, researchers need to recall that similar behavioral outcomes may stem from very different cognitive/brain causes (see, also, comparisons in the social domain between autism and WS in Tsirempolou, Lawrence, Lee, Ewing, & Karmiloff-Smith, 2006).

Is the Environment the Same for Typically and Atypically Developing Individuals?

A frequent question asked by nativists and behavioral geneticists is whether specific aspects of human cognition are due to nature or nurture. But from the neuroconstructivist point of view, this is clearly a false dichotomy. Genes, brain, and environment play a dynamic, multidirectional role in shaping, not merely triggering, developmental outcomes. However, a question that is rarely addressed in the developmental neuroscience literature is whether the environment is the same for individuals developing typically and those developing atypically. In other words, does having a developmental disorder not only involve genetic mutations but also subtly change the environment in which the atypical infant/child develops? A couple of examples serve to make a strong case for hypothesizing that the environment does indeed change with respect to atypically developing infants, probably continuing to do so throughout development. The first example is from motor development. Informal observations of families who visit our lab reveal that parents of infants and toddlers with genetic syndromes find it difficult (compared to parents of typically developing children) to allow their atypically developing offspring to mouth objects freely and to crawl/walk uninhibited in order to fully explore their environment. This reticence is probably because of greater fear of potential danger and accidents, but it results in a less richly explored environment. The second example comes from the learning of vocabulary. In the typical case, parents allow young children to overgeneralize in the early stages of language acquisition when they start to name things (e.g., when the toddler says "cat" when it is actually a dog, the parent often lets it go because both are small animals and they know their child will learn the correct term in the long run). By contrast, in the case of parents of toddlers with, for instance, Down syndrome, parents tend to rapidly veto any overgeneralization (C. Mervis, personal communication, July 10, 1999), probably because they fear that the child with lower intelligence will never learn the correct term if allowed to overgeneralize. However, initial overgeneralization in the healthy child may actually encourage category formation, known to be subsequently impaired in the atypical case. Such unconscious assumptions about atypical development may lead parents to provide less variation in linguistic input, shorter sentences, and in general a less richly varied environment. These quite subtle changes in the child's environment are likely to compound over time, such that the environment of the atypically developing child may increasingly differ from that of the typically developing child. There is, therefore, a vital need for scientific studies of how having a developmental disorder subtly changes the social, cognitive, linguistic, and physical environment in which the atypically developing child grows, which has important implications for intervention.

Implications of a Neuroconstructivist Approach

Recent changes in position of hitherto staunch nativist theorists might be seen as bringing them closer to the neuroconstructivist framework. For example, Baron-Cohen (1998) argued for a "minimalist innate modularity theory" (p. 184), suggesting that the theory-of-mind module might emerge from lower-level systems for detecting eye gaze, intentionality, and shared attention. Likewise, van der Lely (2005) has indicated that her initial syntactic hypothesis has now been replaced by the computational grammatical complexity hypothesis, arguing now that both syntax and phonology rely on a common computational property operating over linguistic representations, which is claimed to be atypical in grammatical SLI. However, like Baron-Cohen's thinking, this remains a very domain-specific hypothesis (complex computations confined to operating solely on linguistic representations) and does not consider possible interactions with nonlinguistic parts of the developing system over developmental time. In other words, these hypotheses do not pertain to domain-relevant processes but do pertain to domain-specific computations over proprietary inputs. Indeed, the theorizing is based on the claim that the grammatical

neurocircuitry underlying language is a developmentally unique higher cognitive system in the functional architecture of the brain, which can be selectively impaired (Fonteneau & van der Lely, 2007).

Nonetheless, the notion of impaired versus intact brain systems in uneven cognitive profiles might be considered useful for clinical practice, even if theoretically it underplays the role of development. If a patient has scores in the normal range in a specific domain, surely there is no need to consider remediation in that domain? The nativist would agree, but the neuroconstructivist would not rule out training in a proficient domain. For instance, take a patient who presents with a serious deficit in, say, number and scores in the normal range in face processing, seemingly very different domains. It would be tempting in such a case to tailor remediation solely to the domain of number. But that misses the very point of the neuroconstructivist framework. Once one explores multiple, low-level interacting processes that underpin face processing early on in development, this leads to a more dynamic view of remediation. In adulthood, the proficient behavior of individuals with WS on face processing tasks is due to a focus on features. Now, if an infant cannot plan saccades and therefore has sticky fixation (as is the case for those with WS, e.g.), then they are more likely to fixate on features than rapidly process the configuration of a face. So, despite the proficient behavioral outcome, face processing in WS is based on different cognitive and brain processes compared to controls (Grice et al., 2001, 2003; Mills et al., 2000). Thus, remediation is necessary in a domain where the outcome gives rise to scores in the normal range. Moreover, early on in development domains are not isolated from one another in their developmental trajectories. Number, too, is influenced by deficits in visual scanning and planning of saccades. Infants with WS fail to scan numerical displays as fully as required in order to discriminate changes in number (van Herwegen et al., 2008). So remediation for number and for face processing might start out by stimulating saccadic movements, and not with number or face processing training at all. Furthermore, take the case of a patient with spatial and numerical deficits, and a parietal cortex that is small compared to healthy controls. The tempting conclusion might be that the atypical size of the parietal region causes the deficits in numerical outcome. But there are two caveats to such a conclusion. First, data about brain regions is usually from adult brains. This tells researchers nothing about possible proportional changes over developmental time. Second, the opposite direction of causality is also possible. From inadequate processing of inputs relevant to parietal cortex, this region may develop less connectivity over time and end up smaller. Only a truly developmental, neuroconstructivist approach could settle such differing interpretations.

Concluding Thoughts

Neuroconstructivism does not rule out domain specificity; it argues that it cannot be taken for granted and must always be questioned. Unlike the nativist perspective, neuroconstructivism offers a truly developmental approach that focuses on change and emergent outcomes. And, every aspect of development turns out to be dynamic and interactive. Genes do not act in isolation in a predetermined way. Even the FOXP2 gene, about which there was much excitement regarding its role in human language, must be thought of in terms of the downstream gene targets to which FOXP2 binds. The profiles of those downstream genes suggest roles in a wide range of general, not domain-specific, functions including morphogenesis, neurite growth, axon guidance, synaptic plasticity, and neurotransmission (Teramitsu & White, 2007). This is a very different level from theorizing at the level of cognitive modules and points to the multilevel complexities of understanding human development in any domain.

For many decades, the notion of plasticity tended to be reserved for the human system's response to damage. By contrast, it has become abundantly clear that development—whether typical or atypical, whether human or nonhuman—is fundamentally characterized by plasticity for learning, with the infant brain dynamically structuring itself over the course of ontogeny. While some macrostructures like the overall six-layer structure of cortex may well be under general genetic constraints, much of the microcircuitry of cortex turns out to be the result of complex multilevel interactions over time. Human intelligence is not a state (i.e., not a collection of static, built-in modules handed down by evolution and that can be intact or impaired). Rather, human intelligence is a process (i.e., the emergent property of dynamic multidirectional interactions between genes, brain, cognition, behavior, and environment).

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Received October 16, 2007
Revision received September 2, 2008
Accepted October 1, 2008 ■