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Can Developmental Disorders Reveal the Component Parts of the Human Language Faculty?

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Differential profiles of language impairments in genetic developmental disorders have been argued to reveal the component parts of the language system and perhaps even the genetic specification of those components. Focusing predominantly on a comparison between Williams syndrome and Specific Language Impairment, we argue that the detailed level of behavioral fractionations observed in these disorders goes beyond the possible contribution of genes and implicates the developmental process as a key contributor to the cognitive outcome. Processes of compensation and interaction across development make highly specific developmental deficits unlikely; in line with this view, the actual level of specificity remains controversial, even in Specific Language Impairment (a paradigmatic example of a supposedly selective deficit). We consider the challenge of characterizing the atypical developmental process from the perspectives of brain development, cognitive development, and computational modeling. Failure to take up this challenge leaves many current explanations of developmental deficits ill-specified at best and implausible at worst.

Selective impairments in adult neuropsychological patients arguably reveal the component parts of the human language faculty. Indeed, those who develop normally but sustain brain damage in adulthood often present with conditions in

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which a single aspect of language is differentially impaired. For example, patients with agrammatism produce well-formed lexical items with appropriate pragmatics, but such items are strung together without appropriate morphosyntactic markers. In contrast, some patients present with normal grammar and pragmatics but also with severe word-finding difficulties. Yet others produce normal language in terms of structure and semantics but display inappropriate pragmatics, such as poor assessment of the situations in which certain registers of language output are fitting. From this, one might conclude that different parts of the human brain are specialized in adulthood for the components of language. In the absence of an obvious account of how such a specialized modular structure might have developed, one might be tempted to conclude that the coarse structure of language is prespecified and under genetic control, with segregated modules to deal with the lexicon, the morphology, the syntax, and the pragmatics.

However, although the adult brain may be specialized and localized for different aspects of language sufficient to generate selective impairments, one cannot focus solely on adult neuropsychological patients to address the question of possible innate specification. The reason is that one cannot assume that infants start their lives with the same brain structure as adults do. Ontogenetic development may play a significant role in giving rise to adult specialization and localization of function. It could be the case that high-level modules—such as morphology, syntax, the lexicon, and pragmatics—form an emergent product of development rather than a starting state (Karmiloff-Smith, 1992). But what if developmental disorders of genetic origin point to the same fractionations of language as in cases of adult neuropsychology? This argument might favor the innate specification of our language faculty's different components. Thus, the questions become, "Do functional aspects of language come apart in developmental disorders?" and "If we do find such differential disability across atypically developing language systems, what inferences may be drawn about the development of the normal language system?"

Cross-syndrome comparisons have indeed identified apparent dissociations in the development of language components. For example, in comparing Down syndrome, Williams syndrome, autism, and fragile X, Fowler (1998) described dissociations between phonology, lexical semantics, morphosyntax, and pragmatics. These disorders illustrate that different etiologies can have dramatically different linguistic profiles. General cognition is not a reliable indicator of language function in children with learning disabilities, although we must assess this correlation with some caution since the holistic notion of IQ or mental age is less valid in disorders with uneven cognitive profiles. Language acquisition typically lags behind mental-age-level expectations in children with learning disabilities. However, disorders such as Williams syndrome and hydrocephalus with associated myelomeningocele appear to superficially represent exceptions to this pattern (Fowler, 1998). From her comparison, Fowler concluded that pragmatics and lexical semantics are more closely tied to mental age than are phonology and

morphosyntax. Tager-Flusberg and Sullivan (1997) carried out a similar comparison of the four disorders, but this time they did so seeking possible asynchronies in the early development of semantic, grammatical, and pragmatic aspects of language. These authors noted disparities in areas such as vocal development, social communicative development, gesture, lexical development, phonological development, early grammar, and pragmatics.

Although Fowler (1998) and Tager-Flusberg and Sullivan (1997) identified differences in the language development of children with Down syndrome, autism, Williams syndrome, and fragile X, they also noted similarities across the disorders. For example, in early development, there were consistent patterns of errors displayed in speech articulation; and in morphosyntax, the order of acquisition of syntactic structures appeared similar, though some disorders stopped short of mastery. These similarities prompted the same conclusions in the two reviews: that the common patterns reflected constraints in the underlying brain mechanisms of motor articulation and syntax acquisition, respectively; and that, as summarized by Fowler, the "non-deviant development is consistent with a model of language acquisition that is heavily constrained by the brain that is acquiring the language" (1998, p. 309).

Such a position downplays the possibility that commonalities in development could arise from the structure of the shared-problem domain to which all individuals are exposed, a possibility to which we return later. More important for current purposes, some theorists have taken the combination of differential patterns of atypical language acquisition and the genetic basis of these disorders to draw strong inferences about links between genes and components of the language system:

> 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 [Williams syndrome] impair their intelligence while sparing their grammar. (Pinker, 1999, p. 262)

> In 1998 [researchers] linked the [KE] disorder to a small segment of chromosome 7, which they labelled SPCH1. Now … Lai et al. [*Nature,* October 2001] have narrowed the disorder down to a specific gene, FOXP2… . The discovery of the gene implicated in speech and language is amongst the first fruits of the Human Genome Project for the cognitive sciences. Just as the 1990s are remembered as the decade of the brain and the dawn of cognitive neuroscience, the first decade of the twenty-first century may well be thought of as the decade of the gene and the dawn of cognitive genetics. (Pinker, 2001, p. 465)

Our interest in this article is to explore the kinds of links that might be appropriate between a cognitive and a genetic level of description sufficient to motivate the notion of a "cognitive genetics." Note that cognitive genetics as a discipline would appear to make stronger claims than "behavioral genetics" would about the rela-

tionship between particular genes and particular cognitive structures. Behavioral genetics is concerned with correlations in behavioral scores between individuals with different levels of relatedness—that is, different probabilistic proportions of shared genes. If cognitive genetics is to be a separate discipline, it would seem to imply links between individual genes (or between sets of genes) and individual cognitive structures. For example, Pinker indicated the sort of theoretical account that his cognitive genetics would license, again in the context of Williams syndrome. Here, a single gene is postulated to contribute to cognitive structures for spatial reasoning but not for language or face processing:

> Presumably LIM-kinase 1 [one of the 25 genes deleted from one copy of chromosome 7] plays an important role in the development of the neural networks used in spatial reasoning, possibly in the parietal lobes. The other missing genes, perhaps, are necessary for the development of other parts and processes of the brain, though not for language or face perception. (Pinker, 1999, p. 260–261)

We do not believe that many researchers hold the view that individual genes uniquely specify individual cognitive structures in the adult, although quotes such as those cited above can sometimes be read in that light (particularly when they use terminology such as *spared* and *impaired* modules to describe the end state of genetic developmental disorders). However, Pinker's comparison of Williams syndrome and Specific Language Impairment provides a useful basis for discussion of the relevant issues.

The structure of this article unfolds as follows. First, we argue that, to be plausible, links between genes and adult cognitive structure are contingent on a certain level of granularity of fractionation in the language system—for example, a fractionation between the lexicon and the grammar. We then review evidence from Williams syndrome demonstrating that fractionation actually occurs on a much finer level than is plausible for the expression of genetic effects. Second, we argue that links between genes and differentially impaired cognitive structures in adults with developmental disorders rely on accurate descriptions of the specificity of cognitive deficits in these adults. We then review evidence from Specific Language Impairment that indicates a current lack of agreement on specificity of outcome. In the second half of this article, we argue that the developmental process itself is an essential component of the explanation of the detailed fractionations we see in developmental disorders (Karmiloff-Smith, 1998). Of course, characterizing the developmental process is a significant challenge, one that we address in four ways. First, we review the mechanisms that the *brain* offers for genes to specify particular cognitive structures. Second, we discuss two features that any explanation of the atypical developmental process must incorporate at the *cognitive* level: interactivity and compensation. Third, in the face of the challenge of development, some theories simply ignore its contribution (see critical discussions in Karmiloff-Smith, 1992, 1998; Thomas &

Karmiloff-Smith, 2002a). We demonstrate that even a rudimentary developmental process applied to theories of this sort finds them wanting as coherent end-state descriptions of an atypical language system. Finally, we discuss one possible method to aid characterization of typical and atypical developmental processes: computational modeling.

DEVELOPMENTAL FRACTIONATIONS OF THE LANGUAGE SYSTEM: WILLIAMS SYNDROME AND SPECIFIC LANGUAGE IMPAIRMENT

The search for links between genes and components of the language system rests on the intuitive assumption that genes target a particular level of *granularity* in the cognitive system. For example, we have seen research pointing to developmental dissociations between phonology, lexical semantics, morphosyntax, and pragmatics across different genetic disorders. One might imagine that genes could reduce the efficiency of, say, memory systems in the developing brain, or of motor articulation systems. However, one would not expect genes to target the acquisition of a semantic category such as vehicles *but not* that of tools, or target the ability to form present-tense verb inflections *but not* past-tense inflections. The idea of granularity remains somewhat intuitive because the specificity of outcome is itself up for debate. But let us bear in mind the intuition of a plausible level of cognitive granularity for genetic fractionations as we consider the example of language development in Williams syndrome.

Williams syndrome

The genetic disorder Williams syndrome (WS) involves the deletion of some 25 genes on one of the copies of chromosome 7 (for full details of the syndrome, see Donnai & Karmiloff-Smith, 2000). It has been hailed as an example of the so-called sparing of the language faculty in general or of certain language components in particular. For example, Clahsen and Almazan (1998) have argued that, with WS, grammar develops normally but that the lexicon develops atypically and suboptimally. Individuals with WS usually present with IQs in the 50–60 range, with exceedingly poor spatial and numerical cognition. Yet despite this, their language seems surprisingly proficient. Indeed, although they display an initial delay in language development, many exhibit large vocabularies by adolescence and adulthood that coexist with relatively good scores on standardized grammatical tests. Could it then be that language or grammar develops independently of intelligence, under the control of a different set of genes, as Pinker (1999) has claimed?

It is worth recalling that IQ scores, rather than mental age, can give a misleading picture of the situation (Karmiloff-Smith, 1998). Most individuals with WS who possess high vocabularies and proficient syntax also have a mental age of around

7–9 years, an age by which all normal children have developed rather sophisticated language. In other words, language can be made to look impressive given IQ but is unremarkable given mental age. Nevertheless, language emerges late in development for those with WS—a fact that, given their extreme attention to sounds and faces in early development, seems at first sight to be inexplicable.

Is the initial delay in language development for those with WS merely reflective of the late maturation of a set of language-specific genes, or are there more complex developmental reasons? Studies from at least four different laboratories in the United Kingdom, the United States, and Italy have shown that infants and toddlers with WS present language development surprisingly late (Mervis & Bertrand, 1997; Paterson, Brown, Gsödl, Johnson, & Karmiloff-Smith, 1999; Singer Harris, Bellugi, Bates, Jones, & Rossen, 1997; Vicari, Brizzolara, Carlesimo, Pezzini, & Volterra, 1996; Volterra, Capirci, Pezzini, Sabbadini, & Vicari, 1996). Why? Several factors interact. Our studies of infants segmenting words out of the speech stream show that those with WS are some 10–20 months behind their typical controls (Nazzi, Paterson, & Karmiloff-Smith, 2002). This difficulty contributes to the delay. Furthermore, despite their abilities with dyadic interaction, infants and toddlers with WS are surprisingly atypical in triadic interaction and in their understanding of the referential function of pointing (Laing et al., 2002; Mervis & Bertrand, 1997)—which is one of the ways that children normally learn new words. In addition, while toddlers with WS behave like controls in mapping similarities between perceptual features of objects, they are significantly poorer than controls at using words to map identity of object categories (Nazzi & Karmiloff-Smith, 2002).

What about older children with WS, who by then have quite well-developed language? Was their language simply delayed and then subsequently followed a normal developmental trajectory? This appears not to be the explanation. Brain-imaging data hint that underlying structures may be atypical. Mills et al. (2000) reported that children with WS have atypical hemispheric specialization with respect to the difference between open and closed class words, suggesting a lack of the normal progressive specialization and localization of brain function in WS compared to controls. Behavioral studies have also revealed subtle differences in cognitive processing. For example, Karmiloff-Smith et al. (1998) found that when individuals with WS monitored a sentence for a target word, performance was disrupted by syntactic violations except when those violations involved lexically based information—for example, subcategory constraints such as transitive/intransitive. This finding led the authors to propose that in WS, there is a deficit in integrating different sources of linguistic information in real-time processing.

In some respects, the developmental trajectory appears normal in WS. For instance, Mervis, Morris, Bertrand, and Robinson (1999) have noted that, while the syntactic abilities of children with WS (39 children, 2.5–12.0 years old) were considerably delayed, syntactic complexity was nonetheless appropriate for the mean length of utterance. This finding contrasts with Down syndrome, autism, and frag-

ile X, where syntactic complexity turned out to be less than would be expected at mean length of utterances over 3. This result prompted Mervis et al. to claim that WS is the first syndrome in which the normal relation between utterance length and complexity has been demonstrated. Might this constitute evidence of a normal developmental trajectory within an isolated domain-specific module for grammar, despite the atypical patterns of brain activation?

Again, the answer appears to be no. On closer inspection, inconsistencies appear in the pattern of grammatical development for those with WS. First, fewer errors are made in syntax than in morphology—verb tense agreement, personal pronouns, grammatical gender (Karmiloff-Smith et al., 1997; Volterrra, Capirci, Pezzini, Sabbadini, & Vicari, 1996). Second, although syntactic performance is often broadly in line with mental-age controls (Zukowski, 2001), within syntax itself WS reveals fractionated development that is appropriate to neither chronological nor mental age (Grant, Valian, & Karmiloff-Smith, 2002; Mervis et al., 1999). For example, Mervis et al. (1999) reported that the syntactic complexity scores of children with WS were significantly higher than expected when based on spatial constructive ability; however, they were significantly lower than expected when based on receptive vocabulary ability, verbal ability, and auditory short-term memory. Across a large sample of 77 participants between 5 and 52 years of age, Mervis et al. (1999) reported that performance on the Test of Receptive Grammar (Bishop, 1983) was poor for complex constructions. Only 18% of the participants (22% of the adults) passed the test block that assessed relative clauses, and only 5% (9% of the adults) passed the block-assessing embedded sentences.

This fine level of fractionation within grammar acquisition brings us back to the wider issue of granularity. The dissociations we find occur within domains to a degree of specificity of language structure that seems beyond the reach of anything like targeted gene expression (Thomas, in press-b). The deep level of fractionation is a pattern that reappears in other areas of WS language. Thus pragmatics, less advanced in WS than grammar, also exhibits within-domain fractionation. Despite relatively good performance in social sensitivity—for example, dyadic eye contact and sensitivity to nonverbal cues—problems arise in such areas as greeting behaviors, topic maintenance, and question answering (Semel & Rosner, 2003). In lexical-semantics, a relative strength in category concepts—e.g., animals versus clothing—contrasts with problems understanding semantic relational concepts such as spatial-temporal terms. Even within category concepts, recent evidence has indicated differential naming problems across categories (Temple, Almazan, & Sherwood, 2002; Thomas et al., 2004). It seems unlikely that genetic events are uniquely to blame for each of these fractionations.

Outside the domain of language, the fractionation proceeds apace (for a review, see Semel & Rosner, 2003). Although sociability is a strength in those with WS (Bellugi, Lichtenberger, Jones, Lai, & St. George, 2000), within sociability there is a fractionation between, on one hand, friendliness and success with adults and,

on the other, the disinterest or ineptness shown when interacting with peers. There is a fractionation between the sensitivity of individuals with WS toward others' emotions and the difficulty they often exhibit in respecting the private space of peers. Within the domain of memory, there are fractionations between relative skill in phonological working memory tasks (e.g., in digit span) and poor performance in visuospatial memory tasks (e.g., Corsi span). Within phonological memory itself, there is a fractionation between strength in learning words but not in learning to read phonologically similar words (Laing et al., 2001) nor in repeating nonwords (Grant et al., 1997). There is a strength in remembering semantically salient items such as poems, stories, and songs over long periods but not in learning or retaining facts over a few minutes (Semel & Rosner, 2003). To these we may add the domain of numeracy, where children with WS reveal a weakness in global quantity judgments but mental-age appropriate learning of the count sequence (Ansari et al., 2003). Last, there is a highly salient dissociation between weaknesses in some visuoperceptual skills (e.g., deciding which of two lines is longer) and a strength in recognizing faces, the latter of which has been the focus of much research (for discussion, see Karmiloff-Smith et al., 2004; Tager-Flusberg, Plesa-Skwerer, Faja, & Joseph, 2003).

In short, what started out as a neat theoretical example of a disorder where language develops normally and uniformly in the face of general and uniform cognitive impairment turns out to be a disorder suffused by cross-domain and within-domain fractionations. Language ability in WS is clearly unusual when compared against other disorders. However, the fractionations are so detailed that a genetic explanation is unlikely to account for much of the variation. Moreover, evidence from the brain level is suggestive that, in terms of localization and specialization of language function, development has not proceeded normally in WS.

Let us now turn to Specific Language Impairment, proposed as another neat theoretical example but one in which language is impaired with normal intelligence. Specific Language Impairment is a disorder that, by its very definition, promises a tidier developmental fractionation than that found in WS.

Specific Language Impairment

To establish potential links between genes and differentially impaired cognitive structures in developmental disorders, one must establish the specificity of the cognitive deficits, which will set bounds on the locus of the putative genetic effects on cognition.

For Pinker's "genetic double dissociation" (Pinker, 1999), Specific Language Impairment (SLI) presents a case opposite to that of WS. That SLI has a genetic component is clear: It runs in families, particularly in males, and twin studies have shown a strong genetic component to the disorder (Bishop, 1992), even though molecular genetics has yet to identify all the genes that may contribute to the outcome.

In 1998, and more specifically in 2001, the scientific world became quite excited about the discovery of what came to be known as the "gene for speech and language." A British family, the now well-known KE family, had been identified as one in which some members had an allelic variation in the FOXP2 gene that gave rise to serious impairments in speech and language; however, family members without this allelic variation developed language normally (Pinker, 2001; Vargha-Khadem et al., 1998). Is this a gene that is novel to the human genome and can thus explain the onset of language and its component parts in the human species? Does such a gene have a unique and specific effect on speech and language in humans? Some researchers have claimed that this might indeed be the case (Pääbo, 1999).

Once more, however, a closer look at the phenotypic outcome in the affected KE family members highlights the need for a more complex explanation. To begin with, the deficits are not specific to language, nor even to speech output. The dysfunctions in the affected family members not only involve oral-facial movements but also particular aspects of the perception of rhythm as well as the production of rhythmic movements of the hands (Alcock, 1995; Watkins, Dronkers, & Vargha-Khadem, 2002). How these affect the language outcome over developmental time has yet to be clarified. Moreover, at the behavioral level, it is far from clear what we can ascribe to the action of this gene in terms of its contribution to human language. When a genetic mutation causes dysfunction of a particular behavior, it does not mean that intactness of that same gene causes the proper functioning of the behavior. (Numerous analogies make this point obvious. For example, if the carburetor of a car is not functioning properly, the car will not run, but it is not the carburetor that explains how the car runs in normal circumstances.) More important, it is highly unlikely that a single gene or even a specific set of genes will explain the development of human language. In the vast majority of cases, genes involve many-to-many mappings, not one-to-one mappings. In other words, the genes that affect the outcome of language structures are likely to influence other brain structures as well. Evidence from the brain level supports this view. Detailed research on affected family members in the KE family has revealed widespread structural and functional brain differences beyond those areas of the brain typically associated with language function in normal adults (e.g., Watkins, Vargha-Khadem, et al., 2002).

This finding should not come as a surprise. There is clear precedent that single-gene disorders can produce phenotypic outcomes with multiple impairments. Fragile X is one such example. In this disorder, a single mutated gene produces widespread alterations because the gene in question is deeply involved in synaptogenesis across the whole developing system (Scerif, Cornish, Wilding, Driver, & Karmiloff-Smith, 2004). Fragile X is associated with the silencing of a single gene, FMR1, whose gene product, FMRP, is normally involved in mechanisms of experience-dependent plasticity throughout the brain (Churchill et al., 2002; Greenough et al., 2001). Although the deficit to this domain-general feature is indeed associated with generalized delay, fragile X exhibits an uneven cognitive

profile in the adult phenotype. It is characterized by relative strengths in vocabulary, long-term memory, and holistic information processing, but relative weaknesses in visuospatial cognition, attention, short-term memory, and sequential information processing (Cornish, Munir, & Cross, 1999, 2001; Freund & Reiss, 1991). The uneven cognitive profile results from a complex interaction of FMRP with other proteins across development, presumably triggering a series of imbalances that have cascading effects on other elements of the developmental pathway at differing times through ontogeny (Scerif et al., 2004; for a discussion, see Scerif, 2003). Thus, a brainwide general change at the cellular level may have differential, seemingly domain-specific outcomes via interactions across developmental time (for discussion, see Karmiloff-Smith & Thomas, 2003).

Discussion of cellular-level differences in developmental disorders may seem remote from language outcome. How does one link synaptogenesis to language development? The point here is that genes are even more remote from language development, yet cognitive genetics is premised on establishing just such links between genetic mutations and language impairments.

Bates has frequently stressed (e.g., Bates, 1997) that there are numerous ways in which language can end up being impaired. These include genetic mutations in many parts of the genome, as well as social and other causes. It is therefore premature at the least to imagine that a single gene or specific set of genes will be our best bet for explaining language impairments. Indeed, the shorthand of "the gene for language" is a particularly dangerous one (see discussion in Karmiloff-Smith, 1998). Bates's subtle position on this debate is of particular relevance (Bates, personal communication, September 2002). Like us, she in no way denied that genes play a crucial role in human development in general and in language in particular. Rather, the multiple functions that each gene may have, including genetic contributions to language over evolutionary time, need to be considered. Amongst these Bates identified: (a) genetic alterations that gave us better fine motor control (like FOXP2); (b) genetic alterations that gave us better perceptual abilities; (c) genetic alterations that permitted a more direct mapping from perception to production, and cross-modal perception which is essential for imitation, a major tool of cultural transmission; (d) genetic alterations that made us faster information processors; (e) genetic alterations that led to the particular social make-up that makes us want to imitate each other and think about what other people are thinking. In Bates' view, none of these genes will end up being specific to speech/language, and yet all of them will be important for the emergence of speech, language, culture, and technology in our species. Such an interactive, emergentist position is far removed from the notion of a specific set of genes solely for language.

Despite these cautionary remarks, some researchers remain convinced that SLI is the key to unveiling the genetic determination of the different component parts of the human language faculty, and they maintain that components of grammar will turn out to be domain specific and "genetically controlled" (van der Lely, 1997, 1999). For these researchers, the KE family was a false dawn, but the sky in the east contin-

ues to brighten. There are many children who fall under a behavioral definition of SLI—that is, a developmental disorder of language found in the absence of frank neurological damage, hearing deficits, severe environmental deprivation, or learning disability (Bishop, 1997; Leonard, 1998); or, more precisely, several different developmental disorders of language with different causes (Bishop, 1997). A careful behavioral screen of these children may reveal cases where the deficits are restricted to certain aspects of language and perhaps even rare cases where the deficits are restricted to specific aspects of syntactic structure (Tomblin & Pandich, 1999; van der Lely, 1999). The heritability of general SLI provides the promissory note that the causes of the behavioral deficits in childhood are genetic, with the precise gene or genes to be revealed at a later date. However, behaviorally defined case studies necessarily confound the contribution of genotype, individual variability, and a particular history of interaction with the environment. They therefore provide ambiguous evidence at best regarding the specific contribution of genes to language structures.

The single-gene version of SLI has subsequently failed to produce language-specific effects, and the behaviorally defined version remains ambiguous in terms of its cognitive specificity. Ullman and Pierpont (in press) have identified three currently competing classes of theory regarding the cognitive-level explanation of behaviorally defined SLI. One class posits deficits in language-specific structures involved in the rule-governed movements or combinations of words into complex structures. According to different versions, children may be impaired in establishing structural relationships, such as agreement or specifier-head relations; they may lack rules for linguistic features; they may be stuck in a period of language development where marking of tense is taken to be optional; they may be solely impaired on non-local dependency relations; or they may have problems with more general language functions, such as learning implicit rules. The second class of theory views behavioral SLI as caused by a nonlinguistic processing deficit that happens to affect language in particular. Versions include claims for reduced processing rate or for capacity limitations on cognitive processing; for an information-processing deficit that particularly affects phonology; and for a low-level perceptual or temporal processing deficit. The third class of theory is espoused by Ullman and Pierpont (in press) and argues that language exploits a general duality in the cognitive system—that between declarative and procedural memory. Vocabulary is claimed to rely on the declarative system and grammar on the procedural system. SLI is then taken to be a developmental disorder of the procedural system, with the linguistic profile a result of the atypical development of the procedural system combined with the attempts of the declarative system to compensate (see also van der Lely & Ullman, 2001). Ullman and Pierpont argued that such an account can explain behavioral deficits sometimes observed outside the domain of language. In their view, such deficits are all in skills that rely on the procedural memory system (Ullman & Pierpont, in press).

Needless to say, if behavioral SLI is indeed to reveal the effects of genes on language structures during development, each of the three classes of theory provides a different target for gene expression. The ambiguity regarding the specificity of the

deficits has to be clarified before behavioral SLI can be included in a "genetic double dissociation" of grammar and general cognition (Pinker, 1999); but current research leads us to doubt that it will ever fall neatly into this framework.

THE ROLE OF DEVELOPMENT

The key difference between adult-acquired aphasia and language deficits in developmental disorders is the process of development. For developmental disorders, a central feature of explanations of the behavioral profile will be the way that language structures are acquired over time and the internal and external constraints that shape this process. It is the developmental process acting under atypical constraints that will account for the fine level of fractionation observed in disorders such as WS (Karmiloff-Smith, 1998). Specifying the nature of the developmental process, however, is a significant challenge. We begin by reviewing the mechanisms that the brain offers for genes to specify particular cognitive structures.

The Brain Level of Description

We firmly believe that for language deficits the cognitive-level explanation is the most appropriate. Nevertheless, we also believe in consistency between levels of description; that is, a cognitive-level theory should not invoke developmental mechanisms that cannot be implemented in the available repertoire of mechanisms of brain development.

The idea that uneven language profiles in genetic disorders can be explained by isolated, atypically developing functional brain systems does not fit well with what is currently known about how genes control brain development. Pennington (2001) summarized three broad classes of genetic control. These include effects on brain size, in terms of altering the number of neurons or synapses; effects on neuronal migration, sometimes differentiated across brain regions; and effects on neurotransmission, either by changing levels of neurotransmitter or the binding properties of receptor proteins. In addition, the timing of gene expression contributes a crucial aspect of the emergent organization of the functional structure (Elman et al., 1996). According to current knowledge, genetic effects do not appear to operate in specific regions over the areas of cerebral cortex that eventually underlie higher cognitive processes (Kingsbury & Finlay, 2001). Regional specialization is achieved by diffuse gradients of gene expression with activity-dependent processes. The primary sensory and motor cortices and the limbic system are to some extent exceptions to this characterization (for discussion, see Kingsbury & Finlay, 2001). The final organization of the cortex depends very much on the way in which the cortex has been activated from birth. In short, there are no current candidate genes that could impair in isolation

the development of a cognitive—let alone a syntactic—structure without other, perhaps more subtle, differences in brain development.

In line with the idea that developmental disorders do not involve region-specific structural atypicalities in the cortex (despite some apparently specific cognitive outcomes), postmortem studies of genetic disorders from the brains of individuals with developmental disorders have revealed widespread anomalies in their gross and fine anatomy. Gross anatomical differences in the relative and absolute size of large-scale structures can be found in disorders such as WS (Bellugi, Mills, Jernigan, Hickok, & Galaburda, 1999), Down syndrome (Nadel, 1999), and fragile X (Reiss, Abrams, Greenlaw, Freund, & Denckla, 1995). A similar picture emerges in terms of brain structures at a finer scale. Kaufmann and Moser (2000) list a range of neocortical cytoarchitectonic and dendritic abnormalities—such as laminar disturbance, increased neuronal packing density, reduced dendritic length, and spine dysgenesis—which have been found across a range of disorders, including Down syndrome, fragile X, WS, neurofibromatosis, Patau syndrome, tuberous sclerosis, phenylketonuria, Rett syndrome, and Rubinstein-Taybi syndrome. It is therefore hard to imagine that SLI, for example, will turn out to present a different picture. Indeed, Bishop has argued for an early generalized neuroimmaturity in SLI (e.g., Bishop, 2002), which may be undetectable in later development (see also, similar discussion in Karmiloff-Smith, 1998).

Genes clearly influence the computational repertoire of the initial six-layer structure of cerebral cortex and the broad pattern of inputs and outputs. However, differentiated, specialized processing structures seem to be contingent on the patterns of activity induced in the cortex by interaction with the environment. Given the presence of widespread brain differences in many developmental disorders and the probability that adult modules emerge as a product of development (Johnson, 2001; Karmiloff-Smith, 1992), it is clear that explaining uneven language profiles in the adult phenotype of developmental disorders will be a complex endeavor. It appears likely that a final account of developmental deficits will need to begin by identifying differences in low-level neurocomputational properties, perhaps in numbers of neurons and their thresholds, in local or global connectivity, and in activity-dependent changes within these parameters (Karmiloff-Smith, 1998; Oliver et al., 2000; Thomas & Richardson, in press). The perturbations that these initial differences cause on the subsequent developmental trajectories of emerging systems must then be mapped out more precisely, taking into account atypical interactions. These interactions will include both those taking place internally between developing components and those taking place externally with the environment.

In terms of specificity of cause and outcome, our understanding of the relationship between neurocomputational parameters and cognitive performance is at present quite limited. For example, a computational property might be anomalous throughout the brain but may only affect those cognitive domains that particularly rely on the property during development. Or it might be that the property's impact

is crucial at a particular time in development and inconsequential if it occurs at another time (Karmiloff-Smith, 1998). Or it might be that the cytoarchitectonic properties that specify regions of cortex are disrupted by diffuse gene expression gradients in such a way that computational anomalies are topologically restricted despite wider structural differences across the brain. The latter possibility might support a restricted scope for the cognitive domains affected during development. Such issues remain open.

From the brain level of description, then, we may conclude that little in the repertoire of developmental brain mechanisms seems able to target specific high-level components of the adult language system (let alone restricted aspects of linguistic structure) while allowing others to develop normally. We now turn to consider the cognitive level, where we identify two features that any explanation of the atypical developmental process should incorporate.[1]

The Cognitive Level of Description

At a pure cognitive level, how must a developmental theory explain the uneven language profile found in some developmental disorders? We believe that it must emphasize at least two (linked) characteristics. These are *interactivity* and *compensation*. A further characteristic is developmental timing which we alluded to above and is dealt with in more detail elsewhere (Elman et al., 1996).

Several authors have argued that early language development is characterized by *interactions* between multiple sources of information (e.g., Bishop, 1997; Chiat, 2001; Karmiloff-Smith, 1997, 1998; McDonald, 1997). For example, Chiat (2001) maintained that language acquisition should be construed as a mapping task between sound and meaning through which the words and sentence structures of a language are established. To achieve this mapping, multiple sets of information are exploited. When semantics is ambiguous, phonology can be used to bootstrap the extraction of meaning. When phonology is ambiguous, semantics can be used to bootstrap the extraction of word-sound information. Together, phonological and semantic information help bootstrap the acquisition of morphosyntax. In a developmental disorder where there are indications of differential deficits across the components of the language system, any explanation of behavioral impairments must incorporate the altered pattern of interactions (and their timing) between the different information sources across development.

Chiat (2001) carried out this very exercise for behavioral SLI. She favored an account that considers the language deficits as arising from impaired phonological processing and the consequent disruption of the interactions inherent in the mapping

[1]We discuss the brain level and the cognitive level separately, under the view that these levels of description are mutually constraining. However, a causal theory needs to remain at a single level of description, in the sense that neural events do not *cause* cognitive events but *are* cognitive events. We discuss the issue of levels of description and causal models in developmental disorders elsewhere (see Karmiloff-Smith & Thomas, 2003; Mareschal et al., in press).

process. Evidence for phonological defecits in individuals with SLI has been mixed, however. Such impairments may exist early in development yet fail to be measurable in the mature system (Karmiloff-Smith, 1998). In other words, the failure to find, say, a phonological deficit in an adult with SLI cannot be assumed to mean that a phonological deficit did not exist in infancy and disrupt the network of interactions from early on. The enabling condition in early development is simply no longer evident in overt behavior during later development. While the absence of phonological deficits in adults may comprise the falsifiability of the causal theory in the endstate, the theory is eminently testable using longitudinal studies in children with SLI.

The second characteristic that any theory of atypical development must incorporate is *compensation*. The importance of this characteristic can be illustrated by a triangular comparison of adult aphasics, healthy children following early focal brain damage, and children with developmental disorders (see Karmiloff-Smith & Thomas, 2003; Thomas, 2003). First, following focal brain damage to their left hemispheres, adults can show persistent selective deficits in their language abilities. Second, and to the contrary, healthy children following similar damage usually demonstrate recovery from initial aphasic symptoms to later perform within the normal range (see Bates & Roe, 2001, for a review). That the child brain has greater effective plasticity than an adult brain presumably permits compensation and reorganization of function. Third, and as a consequence, when we compare adults who had focal lesions as children with adults who have developmental disorders of language, we find significant deficits only in the latter. Pointing to the presence of deficits in a developmental disorder is somewhat tautological, but the third comparison does raise the following question: If genetic developmental disorders of language are to be characterized by initial deficits to language-relevant structures, then why has compensation-to-recovery not occurred as it does in the children with early focal lesions? The answer is that compensation in the developmental disorder probably has occurred to some extent but that the constraints of the system are insufficient to allow performance to develop to a level within the normal range (Mareschal et al., in press; Thomas, 2003). This must be true for behaviorally defined disorders because any child that had successfully compensated for an initial deficit would not be diagnosed as having a disorder.

There are two implications of including *interactivity* and *compensation* into theories of atypical cognitive development. They become most stark in the context of theories that seek to explain developmental language impairments in terms of the architecture of the normal language system, with selective components of the system being under- or over-developed. The implications are best phrased as questions. First, if a deficit arises from initial damage to a selective component, why hasn't this impairment been smeared across other components through the *interactions* that occur between components during development? Second, if a deficit arises from initial damage to a selective component, why haven't other components in the system managed to *compensate* for this deficit and so attenuate the impairment across development?

In many cases, answers to these two questions are hard to formulate because the precise nature of the developmental processes involved in normal language acquisition, let alone atypical language acquisition, remain ill-specified. In the next two sections, we employ two worked examples to illustrate this point.

Specifying the Developmental Process: Example 1

In the face of the challenge of characterizing the (atypical) developmental process, some theories respond by simply ignoring its contribution. Such theoretical approaches are often accompanied by an empirical approach that makes development "disappear" by using "age-matched" or "ability-matched" controls. When the results of these studies are discussed, they focus on whether the atypical group differs from controls or not. It is only a small step to redescribe any significant differences in behavioral data as reflecting a process that is "intact" in the control group and "impaired" in the atypical group. The onus to construct a developmental account of the structures involved has vanished (see Karmiloff-Smith et al., 2004, for discussion of the importance of building task-specific developmental trajectories in evaluating developmental deficits).

It is instructive to take a static model of a developmental deficit and attempt to add a developmental process. The following example comes from work on WS. Clahsen and Almazan (1998) reported evidence from four children with WS in English past tense formation. These children exhibited worse performance on inflecting irregular past tenses than regular past tenses. This effect has proved hard to replicate in larger samples of individuals with WS (see, Thomas et al., 2001). But for our purposes, it is the nature of the explanation that is of interest. Clahsen and Almazan (1998) proposed that children with WS had a specific deficit in their language system in which they experienced problems in accessing the "subnodes" of lexical entries but not in accessing the nodes themselves. This account falls within a theory of inflectional morphology that proposes qualitatively separate mechanisms for producing regular and irregular forms (Pinker, 1999). The theory is still controversial (for discussion, see Thomas & Karmiloff-Smith, 2003), but we will accept it as correct for the purposes of this example. Regular forms are inflected by a rule mechanism—for English verbs, add *-ed* to the verb stem—whereas irregular inflections are stored as individual entries in the lexicon. In this theory, lexical representations have hierarchical structure whereby the past-tense form of an irregular verb is stored as a subentry of the lemma for the verb's stem. For WS, Clahsen and Almazan suggested that normal access to nodes permits regular verb stems to be operated upon by the rule, but access to sub-nodes further down the hierarchy is restricted, thereby impairing irregular inflection (see also Temple, Almazan, & Sherwood, 2002).

The important point here is that the specification of the inflectional mechanism in this account is *nondevelopmental*. It is a specification of the normal adult system or, if one assumes a smaller lexicon, a static picture of the child

language system. Let us try to turn this into a developmental account by proposing a normal development process by which these structures could have been put into place. The developmental account requires at least three components. First, there is a mechanism for learning rules of inflection, able to spot the relationship between present and past tense forms (see Pinker's "epiphany" mechanism, 1999; and Marcus et al., 1992, for ideas on the information that such a rule-learning mechanism might exploit). Second, there is a mechanism for storing lexical entries. This establishes "nodes" for individual word forms, along with a specification of their meaning. For example, the verb "to drink" might be registered as a verb with semantic features corresponding to "the consumption of liquid." Third, there is a mechanism for attaching sub-entries to these nodes so that, per our example, when the child hears the word form "drank" in the context of "consuming liquid in the past," DRANK is established as a sub-entry of DRINK, as a specification of its past tense form. Similarly, in time, DRUNK may be established as a sub-node specifying the past participle.

With a normal developmental process in place (and with our tongues tied as to the psychological plausibility of these learning mechanisms!), let us return to the claims regarding WS. Doing so, we immediately spot an ambiguity. Clahsen and Almazan (1998) have characterized the WS behavioral impairment as stemming from a problem with accessing subnodes, as if those subnodes were already present. This implies no problem with learning subnodes but one with accessing information that has already been learned. One could presumably establish this distinction empirically by showing that retrieval is inconsistent rather than nonexistent, although Clahsen and Almazan have reported no data of this nature. Alternatively, one could interpret the claim in terms of an impairment to the mechanism for learning subnodes so that insufficiently robust representations are put into place following exposure to irregular past-tense forms. Either version of the account would fit within the framework of a disorder in which some components of a normal system develop normally (the first and second mechanisms)[2] while others develop atypically (the third, subnode mechanism).

Now, let us return to one of our previous questions: If a behavioral deficit arises from initial damage to a selective component, then why haven't other components in the system managed to compensate for this deficit and so attenuate the impairment across development? Applied to this example, if children with WS are struggling to learn DRANK as a sub-node of the entry DRINK (or access the information once learnt), why can't they exploit either of their other normally functioning learning mechanisms to achieve normal-looking behavior? Why can't they exploit

[2]Of course, this holds only if one puts aside the recognized fact that language development is delayed in those with WS. For the sake of argument, let us say that the first and second mechanisms are developing normally but slowly, although developmental delay cannot be simply negated as irrelevant (see discussion in Karmiloff-Smith, Scerif, & Ansari, 2003).

the first mechanism to learn DRINK ⇒ DRANK as a mini past-tense rule? Why can't they exploit the second mechanism to establish DRANK as a lexical node (rather than a faulty sub-node) and give it the extended semantic specification of "consuming liquid in the past"? To show a behavioral impairment with irregular inflection (if that is indeed what some individuals with WS show), such compensation *cannot* be available. Why not?

We have attempted to build a possible developmental process for the acquisition of inflections within the words-and-rule theory. Evidently, there must be additional constraints that we have missed, which serve to prevent compensation. We don't know what they are, but then, this is not our theory. We had to attempt the exercise because the Clahsen and Almazan proposal did not include any hypotheses about the developmental process. If the model aswe have described it fails to predict selective developmental defecits, it is contingent on the authors to explain how development is supposed to work in theory. Without additional developmental constraints, the static proposal is at best incomplete, at worst implausible.

Our conclusion from this example is that if questions of compensation (or the lack thereof) are to be addressed, it is vital to first attempt a specification of the developmental process. In the aforementioned example, the initial plausibility of the explanation of the developmental deficit rides on a nondevelopmental characterization of the relevant language structures; but it is far from obvious that such a characterization would remain plausible if upgraded to a developmental explanation. Either way, one can explore these issues only if one attempts to specify a developmental process.

Specifying the Developmental Process: Example 2

Clahsen and Almazan are not unique in underspecifying developmental mechanisms. This is a challenge that has long faced developmental psychologists in general. One possible response to the underspecification has been to implement computational models of the developmental process to evaluate the impact of using different types of learning algorithms, different types of representational constraints, and different training environments on the subsequent success of acquiring cognitive abilities (for a review, see Thomas & Karmiloff-Smith, 2002c). Building computational models necessarily involves simplification and restriction, often to single domains such as inflectional morphology, vocabulary acquisition, and parsing; but it also provides a level of precision lacking in most other forms of theory.

One of the computational architectures most applied to developmental problems has been that of connectionist networks. These have been used in league with a variety of theoretical commitments, from their use as a reasonably theory-neutral tool for exploring the information available in certain learning environments to their servicing the much stronger claim that associative mechanisms are sufficient to explain language acquisition. In this example, we appeal to the role these models can play in expanding the set of candidate inferences that one can draw about underlying cognitive structure based on certain patterns of surface behavior

(Thomas & Karmiloff-Smith, 2002b). If we know what kinds of mechanisms can produce what kinds of behavior, this lets us know what types of explanation are available when a certain kind of behavior is observed.

Recently, connectionist models have been increasingly applied to developmental disorders. For example, in our own work, we have explored the implications of damaging a learning system in its initial state (analogous to a developmental disorder) compared with damaging a system in its trained state (analogous to an adult-acquired deficit; Thomas & Karmiloff-Smith, 2002a). The results demonstrated that some types of damage hurt the system much more in the adult state (e.g., severing network connections) whereas others hurt the system much more in the infant state (e.g., adding noise to processing). The adult system can tolerate noise because it already has an accurate representation of its knowledge; but loss of network structure leads to a decrement in performance since connections contain established knowledge. By contrast, the infant system can tolerate loss of connections because it can reorganize remaining resources to acquire the knowledge; but it is impaired by noisy processing since this blurs the knowledge to be acquired. Empirical evidence supports the importance of a good representation of the input during language acquisition. When McDonald (1997) analyzed the conditions for successful and unsuccessful language acquisition across a range of typical and atypical populations—including late second-language learners and individuals with Down syndrome, WS, and SLI—the results indicated that good representations of speech sounds were key in predicting the successful acquisition of a language, including its syntax.

In other work, we have applied connectionist models to a much more detailed, data-driven consideration of one domain and one developmental disorder—namely, the acquisition of English past-tense formation in individuals with WS (Thomas & Karmiloff-Smith, 2003). The latter model provides a framework in which to evaluate a recent proposal regarding the cause of language deficits, once more in the domain of English past-tense formation but this time in SLI (Ullman & Pierpont, in press). Most important, Ullman and Pierpont's proposal included a process of compensation in explaining the final behavioral impairment.

As we described earlier, Ullman and Pierpont (in press) have put forward a theory of SLI that is contingent on the differential involvement of two memory systems in normal language acquisition. In Ullman and Pierpont's theory, there is a distinction between procedural memory (for fast, sequential, automatic processing) and declarative memory (for slower, parallel, conscious processing). The acquisition of grammar relies on the former and the acquisition of the lexicon on the latter. SLI, with its primary behavioral impairments in grammar, is then construed as a developmental disorder of the procedural system. Most important, Ullman and Pierpont's account explains the SLI profile as including the attempts of the declarative system to compensate for the developmental shortcomings of the procedural system. Van der Lely and Ullman's (2001) English past-tense data are illustrative here. Children with SLI showed low levels of inflection for regular and irregular

verbs (10–20% correct) and similarly low levels of extension of the regular rule to novel stems. Since regulars are normally inflected more accurately than irregulars, this amounts to a greater deficit for regular verbs—viewed as a kind of fractionation. Van der Lely and Ullman's explanation of this pattern of behavior again relies on a linguistic theory that distinguishes separate mechanisms for acquiring regular and irregular verbs. Regulars are learned by a rule-implementing mechanism (part of the procedural system), irregulars by an associative memory (part of the declarative system). According to van der Lely and Ullman, the children with SLI are unable to learn the regular rule with their procedural system, and the few regulars and irregulars that correctly inflect reflect the compensatory action of the declarative system. The idea that regulars are now inflected by an associative memory instead of a rule mechanism is supported by the presence of increased frequency effects in regular inflection in the SLI group compared to typically developing children. Frequency effects are taken to be the hallmark of domain-general associative memory.

It is important to be clear about the chain of inference in this case because it illustrates how researchers move from behavioral evidence to deducing structural fractionations of the language system. The relatively greater impairment of regular inflections with the increased frequency effects in residual regular inflection are taken as evidence that, in SLI, there has been a start-state deficit to a *domain-specific* computational structure responsible for learning regular past-tense forms.

Ullman and Pierpont are to be lauded for their attempts to be more specific about the developmental process in explaining the behavioral data in a developmental disorder and, in particular, for including compensation in their account. Their theory may turn out to be the correct one—however, there is a difficulty. Computational models of atypical development have indicated that intuiting how compensation "will probably work" can be a hit-and-miss affair. This turns out to be the case in past-tense acquisition when we look at an implemented model.

The computational model of past-tense acquisition that we recently explored combines lexical-semantic information about a verb with phonological information about the verb's stem to generate its past-tense form (Thomas & Karmiloff-Smith, 2003; for discussion of this architecture, see Lavric, Pizzagalli, Forstmeier, & Rippon, 2001). *As an outcome of the developmental process*, the network comes to rely differentially on the two sources of information for driving each type of inflection. In particular, it relies heavily on lexical-semantic information for driving irregular inflections so that in the trained model, a lesion to lexical semantics differentially impaired irregulars (see also Joanisse & Seidenberg, 1999). The model employs a three-layer architecture where a layer of internal processing units intercedes between input and output layers. This layer is a common representational resource involved in processing regular, irregular, and novel inflections.

Recently, we demonstrated that manipulating the discriminability of the activation function of the processing units in the internal layer and the output layer of the initial, untrained network led to a system that exhibited a "developmental disor-

der" (Thomas, in press-a). The alteration to the connectionist network roughly had the effect of making computations fuzzier. It reduced the ability of the system to make sharp categorizations so that it required much more training to produce very different outputs patterns for similar input patterns. When the disordered network was "aged matched" to a normally developing past-tense network, it exhibited low levels of regular and irregular inflection and poor regularization of novel stems. In other words, the disordered network gave an approximate fit to the SLI data presented by van der Lely and Ullman (2001). Moreover, just as in the empirical data, regular verbs in the model exhibited an elevated frequency effect. Subsequent analysis of the network revealed that regular inflection was being driven more strongly by lexical-semantic input than in the normal network. In effect, the system was treating regulars in the same way as irregulars, as if all verbs were exceptions to be generated via support from the lexicon.

On the face of it, this model would appear to parallel van der Lely and Ullman's explanation (2001) of their SLI data: Residual performance on regular inflection reflects the action of the declarative memory system storing word-specific information. In the disordered network, regulars and irregulars are treated in the same way, with equivalent reliance on lexical-semantics and equivalent frequency effects. Crucially, however, the start-state manipulation to the connectionist network was *not to a domain-specific processing structure* affecting only regulars, as assumed by Ullman and Pierpont (2004) and van der Lely; instead, the manipulation targeted a general processing resource used to inflect regular and irregular verbs. However, the particular computational property that was altered was one upon which regular verbs differentially relied. The result was a deflection of the developmental trajectory that suggested a fractionation between regular and irregular verbs, but this behavioral pattern did not in fact reflect a partition within the functional structure.

In effect, the start-state manipulation altered a computational property that was *domain relevant* to regular inflection rather than domain specific (Karmiloff-Smith, 1998). Specifically, an essential characteristic of the regular rule is to treat all items within a category in the same way. For this, the system required the ability to form sharp category boundaries. Reduced discriminability of the processing units caused delayed learning to all verbs but particularly impaired the network in forming the sharp categories necessary to learn and generalize regular inflections. These initial alterations to the common computational resource had the effect of altering the balance of the information sources on which the network relied to generate past-tense forms. Phonological regularities were downplayed, whereas word-specific information was emphasized. The atypical constraints of the learning system served to alter the interaction between phonological and semantic sources of knowledge during development of this morphosyntactic ability.

These computational simulations do not demonstrate that Ullman and Pierpont's procedural-declarative theory of SLI is wrong. What they demonstrate is that the inferences made by these authors are not the only ones legitimized by their behavioral data. Inferences drawn from developmental behavioral deficits

to affected underlying structures are *entirely contingent on a precise specification of the developmental process*. Crucially for the general argument in this example, details of the developmental process fully determine whether a behavioral dissociation should be taken as evidence for an initial deficit to domain-specific processing structure (and therefore, indicative of a fractionation of the language system) or should be taken as an initial deficit to a general processing resource that results in a seeming domain-specific outcome (and no structural fractionation).

Without specification of the developmental process, we do not know whether there are domain-specific effects (to be explained by gene expression), modality-specific effects, or domain-general effects. Links between genes and language can never be answered without considering the details of the process of development.

The Importance of the Problem Domain

Until now we have focused on *differences* between normal and developmentally disordered systems. Lastly, we return to consider the possible implications of behavioral *similarities* between the patterns of language development exhibited by typical and atypical populations. Recall: one explanation is that these similarities reflect internal constraints of the language development system. On theoretical grounds, however, similarities between typical and atypical development may have another explanation. It is possible that the range of behaviors that individuals exhibit in language development is constrained to some extent by the physical and social environment in which the individual's cognitive system is embedded. That is, behaviors normal or otherwise are in part constrained by the structure of the problem domain to which the cognitive system is exposed, whatever its underlying architecture. The extent to which cognitive architecture is visible in the behavioral changes and error patterns exhibited across development is a serious and unresolved issue. The simplest illustration of this would be a cognitive domain that had an easy part and a hard part. A range of learning systems would naturally acquire the easy part before the hard part. Consequently, a developmental fractionation here would tell us little about the actual learning system involved. Computer simulations can again provide a means to probe this question further. When we exposed a variety of associative architectures to the past-tense domain, there was great variation across the developmental profiles (Mareschal et al., in press). Nevertheless, the systems also exhibited similarities in their profiles: Regular acquisition was usually in advance of irregular acquisition, and generalization of the regular rule was usually weaker to novel stems that rhymed with irregulars than to those that did not. These patterns were a result of the common problem domain to which the systems were exposed (see Thomas & Redington, 2004, for a similar exercise in modeling atypical syntax processing). The developmental commonalities across "disordered" networks would not, in this instance, be strongly supportive of claims

that language acquisition is primarily constrained by the brain that is acquiring the language—perhaps the greater contribution arises from properties of the common problem domain. Yet, as we saw in the introduction, commonalities in the developmental trajectories across different development disorders have been used to draw just such a conclusion (see Fowler, 1998; Tager-Flusberg, & Sullivan, 1997).

CONCLUSION

Can developmental disorders reveal the component parts of the human language faculty? In a sense, we are left with a puzzle to which we can only begin to sketch a solution. There does appear to be fractionation between the different aspects of the language system in developmental disorders. In Williams syndrome, such fractionation occurs at a very fine-grained level. In SLI, there were initially claims for a neat fractionation, but increasingly it appears that deficits are differential, simply with lesser impairment in non-linguistic domains. How then are we to explain the level of fractionation that we encounter within the development of disordered language systems?

We have argued that the observed fractionations are the consequences of the processes of ontogenetic development acting on a neonatal brain that has been constructed with (perhaps subtly) altered initial neurocomputational biases. Elsewhere, we have referred to this theoretical framework as "neuroconstructivism" (Karmiloff-Smith, 1998; Karmiloff-Smith & Thomas, 2003): On the one hand, it is unlikely that genetic effects during brain development in neurogenetic disorders are uniform across the entire brain; on the other, they are unlikely to be highly region specific. Rather than target certain circuits, differential effects are probably graded, particularly with regard to the higher cortical functions identified in adults (Kingsbury & Finlay, 2001).

We still await further data here. For example, Reiss et al. (2004) recently reported that, compared to controls, a sample of adults with WS presented decreases in volume of gray matter and in densities of subcortical and cortical regions forming the human visuospatial system, a finding that the authors argued was associated with visuospatial impairments in the disorder. They also reported increases in volume of gray matter and in densities of several areas, including the amygdala, the orbital and medial prefrontal cortices, the anterior cingulate, the insular cortex, and the superior temporal gyrus—areas involved in emotion and face processing. Reiss et al. argued that these increases could be associated with "enhanced emotionality and face processing." However, emotion and face processing rarely present at the appropriate chronological age level in those with WS and have been argued to be atypical in the disorder. Moreover, the level of specificity of the brain differences is still way short of the level of fractionation (frequently within domain) that we saw in the behavioral evidence. In developmental disorders, it is

therefore likely that *the granularity of genetic differences in cortex is at a coarser level than that of cognitive modules or parts thereof.*

The final highly differentiated cognitive profile in disorders such as WS is due to the result of complex processes of development, attenuating or exaggerating (perhaps initially subtle) neurocomputational differences. At the brain level, the normal emergence of an interactive network of neural systems may be perturbed by several factors: by the differing effect of the atypical computational biases on the ability of various areas to process the signal with which they are provided by the initial large scale input–output connectivity of the brain; by anomalies in the emergence of specialized circuits through pruning or competition; by subtle differences in timing due to maturational delay; by compensatory changes during interactions between different brain regions; and by the atypical subjective environment to which the individual with the disorder is exposed (for discussion, see Mareschal et al., in press). At the cognitive level for the domain of language, the interaction between separate sources of information—phonological, semantic, morphological, syntactic, and pragmatic—may be altered following initial problems in one or more domains, thereby leading to an uneven profile. However, patterns of strengths and weaknesses must also be viewed through the lens of what is hard and what is easy for all learners in language acquisition. For developmental disorders, then, the outcome at the cognitive level is *a granularity of subsequent behavioral fractionations likely to be considerably finer than cognitive-level modules.*

In our own neuroconstructivist framework, we have a view of what we expect developmental processes to look like. However, our main argument here is that explanations of developmental deficits depend on having a developmental account *of some kind*, and in many cases even this minimal requirement is absent. Particular methodological approaches emphasize development—the use of longitudinal studies, the construction of task-specific developmental trajectories, the tracing of childhood and adult deficits back to their precursors in infancy, the use of developmental computational models to simulate behavioral data. But a developmental perspective can also be applied to data gained from traditional methodologies.

Apparent fractionations of the language system in developmental disorders can tell us about the constraints that shape the development of the language and even how genes may influence those constraints. Merely stating that disorders have a genetic component tells us nothing about how genes are expressed. So, this story is really just beginning, and in our view the developmental process itself will eventually lie at its heart.

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