Language acquisition in developmental disorders

Book Chapter

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Abstract

In this chapter, I review recent research into language acquisition in developmental disorders, and the light that these findings shed on the nature of language acquisition in typically developing children. Disorders considered include Specific Language Impairment, autism, Down syndrome, and Williams syndrome. I argue that disorders of language should be construed in terms of differences in the constraints that shape the learning process. I outline the integrative nature of this learning process, and how properties such as redundancy and compensation may be key characteristics of learning systems with atypical constraints. These ideas, as well as the new methodologies now being used to study variations in pathways of language acquisition, are illustrated with case studies from Williams syndrome and Specific Language Impairment.

1: Introduction

What light can developmental disorders shed on language development? Can they reveal the extent to which language development is channelled by biological constraints? Can they demonstrate whether language learning relies on general cognitive mechanisms or whether it is domain-specific? In this chapter, we consider what has been learned by the comparison of language development across multiple disorders, as well as the unresolved issues that still exist in this field.

First, let us clarify what is meant by developmental disorders. Developmental disorders can be split into four groups. The first are disorders caused by well-understood genetic abnormalities, such as Down syndrome (three copies of chromosome 21) and Williams syndrome (deletion of around 28 genes from one copy of chromosome 7; Tassabehji, 2003). In these neurogenetic disorders, cognitive

impairments are typically not restricted to a single cognitive domain. The second group are disorders defined on the basis of behavioural deficits, such as dyslexia, Specific Language Impairment and autism. In these disorders, behavioural genetics indicates sometimes substantial heritability, but the causal genes are not yet known and may well not be mutations (that is, they may be spectrum disorders corresponding to an unlucky accumulation of normal genetic variations that each add a small risk for the target disorder). In these disorders, it is sometimes argued that the deficits are restricted to single cognitive domains (e.g., reading in dyslexia, language in Specific Language Impairment) but there remain doubts as to whether these disorders are indeed homogeneous rather than behavioural clusters with milder associated deficits and heterogeneous causes. The third group correspond to disorders where there is learning disability but its cause is unknown. The final group correspond to disorders caused by environmental factors, such as acquired brain damage, viral infections or an impoverished environment, be it cognitive (such as neglect) or biological (such as in Foetal Alcohol syndrome). The first and last of these four groups index the primary locus of causality – the first group nature, the last group nurture – while the middle two reflect our current lack of knowledge about the cause of some disorders. A given behavioural impairment may be generated in more than one way. For example, poor reading may be the consequence of either dyslexia or limited opportunities to learn to read. Our discussion will predominantly focus on the first two of these four groups – neurogenetic and behavioural disorders (see, e.g., Goldin-Meadow, 2005, for discussion of language development under conditions of impoverished input). Disorders of development that are caused by early acquired brain damage will be considered briefly in section 4.

Both dissociation and association methodologies have been applied to developmental disorders of language (see Bishop, 1997, Karmiloff-Smith, 1998, Temple, 1997, for discussion). Where *ability A* develops normally but *ability B* develops atypically, a possible inference is that they are subserved by independent systems that do not interact during development. Where *ability A* and *ability B* both develop atypically, one possible inference is that a common system subserves their development; another is that they are subserved by two systems that causally interact across development (Morton, 2004). Very different explanatory frameworks have been deployed in interpreting language deficits in developmental disorders. On the one hand, some researchers have extended the logic of adult cognitive neuropsychology to developmental disorders, hypothesising that patterns of behavioural deficits should be related to normal modular theories of the language system (for the appropriate age); deficits are then viewed as the failure of individual components to develop (e.g., Clahsen & Temple, 2003). On the other hand, other researchers stress the interactive, adaptive nature of the developmental process; they argue that the normal adult modular structure is the product of the developmental process rather than a precursor to it and, since cognitive components interact across development, impairments are likely to spread; moreover, genetic effects in disorders are typically widespread in the brain rather than equivalent to focal lesions; together, these researchers infer that the language system in developmental disorders may be qualitatively atypical and therefore one need expect no direct correspondence to the normal language system (e.g., the *neuroconstructivist* position; see Karmiloff-Smith, 1998; Mareschal et al., 2007; Thomas & Karmiloff-Smith, 2002, 2005; see Thomas, Pursuer, & Richardson, in press, for a more detailed comparison of these position). Currently, then, some researchers believe that at best, developmental disorders of

language may offer a direct window onto the structure of the normal language system while others argue that at worst, disorders may tell us nothing about normal language development beyond an inkling of the constraints shape it. One goal of this chapter is to indicate where in between these two extremes the truth may lie.

The following examples illustrate the types of claims that have been made about language development in development disorders. It has been argued that Specific Language Impairment may be a genetic failure of language (and in some cases, only syntax) to develop against a background of otherwise normally developing cognition (e.g., as assessed by non-verbal intelligence tests) (Pinker, 1999; van der Lely, 2004). Williams syndrome, a rare neurogenetic disorder, shows an uneven cognitive profile, with relatively strong language ability (for overall mental age) and especially in receptive vocabulary, a particular weakness in visuospatial construction and a background of learning disability. Based on early reports, Pinker (1994, 1999) argued that language might develop normally in this disorder despite deficits in general cognition. In high-functioning individuals with autism, it has been argued that the structural parts of language can be acquired appropriately but these individuals do not master its use in social situations, which is crucial for effective communication (Happé, 1994). These three claims revolve around disorders that exhibit dissociations. Equally, we need explanations of associations, for example where all aspects of language development are delayed in a disorder but individuals nevertheless seems to follow normal milestones, though perhaps terminating at a lower level of sophistication (e.g., as in Down syndrome). What property of a cognitive system could produce general language delay? Speculations about how language development can go wrong rely on a detailed understanding of how it works in the normal case.

2. Language as a learning problem

The effects of developmental damage to the language system may be quite different to the effects of acquired damage in adulthood, because in the former case one cannot assume that there is already a language system in place (Thomas & Karmiloff-Smith, 2002). Instead, developmental deficits must be interpreted as disruptions to an adaptive learning process. Theories of language development differ depending on how tightly constrained they view the learning process to be (very tightly in nativist theories, where environmental input serves to 'trigger' adult states; weakly in empiricist theories where structure in input-output mappings serves to construct the adult state from more general resources). Minimally, developmental disorders must be viewed in terms of changes to the constraints under which language development takes place, whether learning is tightly or loosely constrained. But learning theories bring into play a range of other concepts. These include the interactions between different information sources or processing mechanisms, the importance of the quality of input and output representations, changes in plasticity with age, compensation between processing components when some are initially impaired, and the possibility of redundancy (i.e., multiple developmental pathways to success).

At the most abstract level, Tager-Flusberg and Sullivan (1998) characterised normal language development as involving the integration of three streams of information, about the physical world, about people, and about the structure of language itself. Ultimately, these will form the basis of lexical semantics, pragmatics, and phonology/syntax respectively. These information streams are depicted in Figure 1. The most important point is that language development involves the *integration* of these information sources – to use some linguistic structure to convey some meaning

to achieve some social goal. But integration may be a complex process: some types of information may be redundantly available in more than one information stream; or information in one stream may help resolve ambiguities in the other and so aid its acquisition (the basis of the developmental notion of *bootstrapping*). In this way, Chiat (2001) emphasised how disorders of language development must construe observed impairments in terms of the way each disorder changes the problem of learning the mapping from sound to meaning and from meaning to sound.

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Insert Figure 1 about here

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Cross-syndrome comparisons are potentially most informative about the different ways in which the developmental process can be deflected. Figure 2 demonstrates data from our lab that illustrate the sorts of patterns that can be observed when disorders are compared (see Annaz, 2006; Thomas et al., 2009, for general methods). These data depict cross-sectional developmental trajectories for 18 children with Williams syndrome (WS), 15 children with Down syndrome (DS), 16 high-functioning children with autism (HFA), and 17 low-functioning children with autism (LFA) between the ages of 5 and 12, against a typically developing (TD) sample of 25 children. The left panel shows performance on a standardised test of receptive vocabulary (a task where the child has to point to the picture that goes with a word), while the right panel shows performance on a non-verbal test of visuospatial construction (a task where the child has to complete a simple puzzle, building a target pattern from geometric shapes). In both cases, test (mental) age is plotted against chronological age.

Two of the disorders show similar profiles across verbal and non-verbal measures, illustrating *developmental associations*. For the HFA group, development is slightly below the TD trajectory but within the normal range, while the DS group shows very delayed and only slowly improving performance on both measures. By contrast, the WS group shows development parallel to and just below the normal range for language (similar to the HFA group), but very delayed development on visuospatial construction (similar to the DS group). Meanwhile, the LFA group shows poor performance on language development (indeed, there is no significant improvement with chronological age in this cross-sectional sample) but then development within the normal range for visuospatial construction (similar to the HFA group). These latter two cases illustrate *developmental dissociations*.

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Insert Figure 2 about here

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Such cross-syndrome comparisons have been carried out to explore associations and dissociations within the domain of language itself, both in early development (Tager-Flusberg & Sullivan, 1998) and later childhood (Fowler, 1998) (see also Rice, Warren & Betz, 2005). These comparisons focused on phonology, syntax, semantics and pragmatics, and identified several contrasting profiles. For high-functioning children with autism, problems primarily occur in pragmatics, in line with the social disengagement typical of the disorder. For low-functioning children with autism, there are additionally problems with lexical semantics and concept formation. Problems in lexical semantics and concepts also characterise the development of children with learning disability (or 'mental retardation', to use US terminology). In Williams syndrome, language development is mostly characterised

by delay but with a relatively successful eventual outcome. However, there are also differences in pragmatics, but now the pattern is of hypersociability with an elevated interest in using language for social engagement. In Down syndrome, problems appear to primarily impact on the structural aspects of language, especially phonology and those parts of language that rely on phonological distinctions (morphology, syntax). Specific Language Impairment and dyslexia are also viewed as behavioural disorders that impact primarily on structural language information, with sub-types emphasising difficulties in phonology, semantics, or syntax. The contrast between these disorders is included in Figure 1.

What kinds of conclusions have been drawn from these comparisons? Fowler (1998) noted that pragmatics and semantics appear to be most closely tied to overall mental age across different disorders, while phonology and syntax can dissociate. Either pragmatics and semantics involve more general systems, or their successful development requires interactions between a greater number of cognitive components. McDonald (1997) contrasted various populations in which language acquisition is broadly successful (including WS and HFA) with those in which language acquisition is unsuccessful (including DS and SLI, but also late L1 and L2 learners). Her conclusion was that good representations of speech sounds (phonology) are crucial in predicting eventual successful acquisition. When the individual cannot encode the basic phonological contrasts over which the rules of language operate, prognosis is poor. However, as Morton (2004) argues, many cognitive components typically contribute to the successful development of an overall system, and if any one of these is impaired (and no redundancy is present) the system may fail to develop normally. Good phonology may be a necessary but not sufficient requirement for successful language acquisition.

In their reviews, both Fowler (1998) and Tager-Flusberg and Sullivan (1998) were struck by the absence of radically different pathways by which language can be developed. In most disorders, acquisition exhibits similarities to the normal trajectory, proceeding through a common sequence and via common milestones (as far as acquisition progresses in a given disorder). Their common conclusion was that these similarities must be the result of invariant internal biological constraints that shape language development in all the disorders. Thus Fowler argued that "…language acquisition [is] heavily constrained by brain structure" (1998, p.309), while Tager-Flusberg and Sullivan concluded that "there are not multiple alternative ways of acquiring language, though as each of these components [phonology, semantics, and syntax] develops over time, they may become integrated in different ways, which lead to syndrome-specific profiles" (1998, p.231). An alternative possibility is that, on computational grounds, some of the similarities to typical development are to be expected since learning systems with different properties are nevertheless trying to solve the same problem; that is, all the children are trying to solve the problem of communicating meaning via sound (Thomas, 2005a).

In the next two sections, we consider two more detailed examples of language acquisition in developmental disorders. These stress how important it is to view atypical language development in terms of the trajectory of an adaptive learning system operating under altered constraints (computational or informational). The first example shows how research has progressed over a decade or more of investigating language development in Williams syndrome, and introduces the idea of *redundancy* in language development. The second example of Specific Language Impairment reveals the emergence of new methods to address key issues in the atypical development of language, and introduces the idea of *compensation*.

3. The case of language development in Williams syndrome

Williams syndrome has been much studied over the last fifteen years due to the uneven cognitive profile observed in this neurogenetic disorder (Donnai & Karmiloff-Smith, 2000). Figure 2 depicts one of the most salient dissociations observed in standardised testing: a disparity between receptive vocabulary and visuospatial constructive skill. Individuals with WS also show a hypersociable or 'over-friendly' personality profile (Jones et al., 2000), with a relative strength in facial recognition (Annaz et al., 2009). By contrast, they have relative weaknesses in numeracy and problem solving skills, and overall IQs typically fall between 50 and 70. Based on the early findings of Ursula Bellugi from a small number of individuals with the disorder, Pinker (1994, 1999) argued that WS might constitute a genetic dissociation in which grammar develops normally but general intelligence is impaired – in support of a wider argument that normal language development involves innate, domain-specific mechanisms. Although, as with any disorder, there is variability, individuals with WS often have a surprising facility with language compared to some of their other abilities, and compared to other disorders with comparable overall mental age such as Down syndrome (e.g., as shown in Figure 2). A dissociation of this nature encourages the idea that developmental disorders might serve to 'fractionate' the cognitive system into its component parts. The simple fractionation proposed by Pinker (1994) is shown in Figure 3a.

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Insert Figure 3a and 3b about here

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These initial claims inspired a burst of research on WS that has lasted fifteen years and incorporated investigation of the genetic basis of the disorder, its effects on brain development, and a detailed consideration of the cognitive abilities of these individuals using more sensitive experimental tasks. Research on brain development has tended to indicate that the genetic effects of the mutation are fairly widespread rather than focal, consistent with most neurogenetic disorders that affect cognition (Toga, Thompson & Sowell, 2006). By contrast, research on the cognitive abilities of these individuals has revealed an increasingly complex and fine-grained picture. In the domain of language, the most salient characteristic in WS is that development is delayed (Brock, 2007). Early in childhood, the language ability of these children is on a par with children with DS (Paterson et al., 1999). Only in later childhood and adolescence does WS language development stretch away, while that of DS asymptotes. In most published empirical studies, the performance of individuals with WS is compared to a typically developing control group matched for *mental age* (MA); performance is very rarely at the level of a control group matched for chronological age. MA comparisons implicitly accept that there is no dissociation between language ability and overall mental age in WS (although the notion of a single, overall mental age is itself weakened for disorders in which component abilities are at different levels).

Various studies have reported dissociations within the domain of language, for instance problems in learning spatial prepositions, difficulties in the pragmatics of conversation, and problems with more complex aspects of morphology. Thomas and Karmiloff-Smith (2003) reviewed the literature at the turn of the century and identified two types of emerging hypothesis. The *Semantics-Phonology Imbalance hypothesis* suggested that individuals with WS are relatively strong in their language

development but that it occurs in a subtly different way. In WS, there might be greater emphasis on the sounds of words and less emphasis on their precise meaning. For example, in early language development, children with WS show vocabulary growth ahead of the normal markers of semantic development such as referential point and object sorting (see Thomas, 2005a, for a review). By contrast, the *Conservative hypothesis* suggests that there is nothing atypical about language development in WS – it is entirely in line with mental age (i.e., it is delayed). What anomalies there are stem from other characteristics of the disorder such as the visuospatial deficit that causes problems in learning spatial prepositions (*in*, *on*, *under*) and the hypersociable profile that leads these individuals to use language strategically in a way to capture and maintain attention in social interactions (see, e.g., Thomas et al., 2006, for an example in the context of unusual vocabulary use in WS). Under the Conservative hypothesis, language in WS is made to look more impressive by comparing it to other cognitive domains in which there are particular weaknesses (e.g., visuospatial construction) and to other disorders in which there are known phonological processing problems, such as DS and SLI (e.g., Ring & Clahsen, 2005).

As research has progressed in WS, methodological problems such as restricted sample sizes and inappropriate control groups have increasingly been addressed. Brock (2007) recently reviewed the status of the two competing hypotheses. He found that the *Conservative hypothesis* has gained progressively more support over the *Imbalance hypothesis.* Delay remains the most salient feature of language development in WS and performance appears to be in line with the level of general cognition (excluding the visuospatial deficit). While there are some anomalies compared to MA-matched control groups, most of these appear to stem from other non-verbal aspects of the disorder. One exception may be receptive vocabulary (e.g.,

as shown by the data in Figure 2, left panel). This skill is puzzlingly strong even compared to the rest of language and the disparity remains to be explained. Brock (2007) argues that the slow and anomalous early phase of language development in WS combined with the eventual relative success in acquisition implicates *redundancy*. That is, early language development in the disorder does not exploit the normal combination of information sources and cognitive processes; it finds a pathway to success that takes longer but is nonetheless eventually successful. This position contrasts with that of Tager-Flusberg and Sullivan (1998) who, as we saw earlier, argued against alternative pathways for successful language acquisition.

To offer a concrete example of this redundancy, Laing et al. (2002) identified deficits in shared attention in toddlers with WS. Although these toddlers scored well on dyadic interactions (sharing attention with the caregiver), they exhibited deficits in triadic interactions, where attention had to be shifted between the caregiver and an object that was being played with. The deficit was a consequence of their elevated interest in (and fixation on) the face of the caregiver. It is thought that triadic interactions are an important contributor to learning object names in situations where the caregiver labels an object that is being played with *("Look at the ball! This is a ball!"*) (e.g., Tomasello & Farrar, 1986). Therefore, the toddler with WS may to some extent be deprived of this information source in their language development. However, explicit labelling is not the only route to learning object names, and while development is slower, these children do succeed in vocabulary acquisition. The inference is therefore that other redundant pathways to success are followed, which are less efficient and take longer.

Overall, research into the cognitive profile of individuals with WS has tended to produce increasingly fine-scale fractionations between different abilities even

within cognitive domains. Although the initial fractionation in WS was argued to be between language and cognition as shown in Figure 3a, the current picture of is closer to that shown in Figure 3b. This fine-scaled fractionation contrasts with the coarse and widespread effect of the genetic mutation on brain development. One can make this point more starkly: in WS, the granularity of genetic differences in cortex is far coarser than the level of cognitive modules, yet the impact on cognitive development is a granularity of subsequent fractionations considerably finer than the level of cognitive modules (Thomas, 2006). The difference in granularity between genetic and cognitive effects arises because cognitive structure is the result of a developmental process that exaggerates or attenuates the effects of atypical constraints on learning, depending on the cognitive domain (Karmiloff-Smith, 1998). In the next section, we will see how new methods are important to specifying the nature of this developmental process.

4. The case of language development in Specific Language Impairment

SLI is a behaviourally defined disorder diagnosed by the presence of a deficit in language development in the presence of apparently normal non-verbal development and the absence of any obvious neurological impairment or environmental cause. It is a heritable disorder but the precise genes involved are unknown (although some candidate genes and chromosomal regions have been proposed; see Smith, 2007). SLI is sometimes conflated with the British KE family. Affected members of this family were reported to have particular problems with language and the cause was traced to a mutated gene on chromosome 7 called FOXP2 (see Marcus & Fisher, 2003). As with WS and in keeping with other neurogenetic disorders, subsequent research has indicated that cognitive differences and brain differences between affected and

unaffected family members are more widespread than the domain of and substrate for language (Watkins, Dronkers & Vargha-Khadem, 2002; Watkins et al., 2002). However, behaviourally defined SLI is not caused by the FOXP2 mutation (Newbury et al., 2002).

SLI is a disorder that primarily impacts on syntax and phonology, although its particular features depend on the language being acquired (Leonard, 1998). It appears to be a heterogeneous disorder, with subtypes that differentially impact morphology/syntax, semantics, and pragmatics (Bishop & Norbury, 2002). Three principal theories have been advanced for the cause of behaviourally defined SLI. First, SLI has been explained in terms of deficits to rule-based, language-specific structures (e.g., van der Lely, 2004). Versions of this theory include an impairment in specific structural relationships (agreement, specifier head-relations), absent linguistic features, fixation in a period of development where tense marking is 'optional', and problems in more general language functions (implicit rule learning, representing relationships between structures). Second, SLI has been explained in terms of a non-linguistic processing deficit that happens to particularly impact on language (e.g., Joanisse, 2007). Proposals on the nature of this impairment include reduced processing rate, capacity limitations on cognitive processing, a deficit that particularly affects phonology, and a low-level perceptual or temporal processing deficit. Third, a neurobiological proposal by Ullman and Pierpont (2005) called the *Procedural-Declarative theory* argues that grammar acquisition is like skill learning, and therefore relies on procedural or implicit memory. By contrast, vocabulary acquisition concerns the learning of explicit knowledge and therefore relies on declarative memory. SLI corresponds to a developmental impairment of the procedural system. All of these

theories identify the deficits in SLI as involving disruptions to the language information stream in Figure 1.

Ullman and Pierpont's (2005) proposal is notable in that it identifies *compensation* as a key feature in producing the language profile of children with SLI. In the face of an impairment to the procedural learning system, Ullman and Pierpont argue that the declarative memory system attempts to compensate by acquiring certain aspects of language, such as frequently used phrases or inflected words. So, for example, where a typically developing child might inflect an English past tense such as 'talked' in terms of the regularities that operate in inflectional morphology (in English, to form the past tense, add –ed to the verb stem), the child with SLI might succeed in inflecting this high frequency verb by learning it as an unanalysed whole (note, however, that the performance of these children on inflection tasks is generally fairly poor). The evidence for this is that where normal children inflect regular verbs equally accurately irrespective of their frequency, children with SLI show frequency effects, inflecting high frequency regulars more accurately than low frequency regulars (van der Lely & Ullman, 2001). Frequency effects are taken to be the hallmark of the operation of declarative memory.

What is important about Ullman and Pierpont's approach is that it emphasises the atypical learning process. Impaired behaviour is the outcome of development working under different constraints, rather than the result of focal damage to a component of a static system. That is not to say that damage to a static system might not sometimes be an appropriate explanation, for instance, to explain a similar behavioural deficit when observed in a normal adult who has suffered brain damage. For example, individuals suffering Broca's aphasia after left anterior damage exhibit particular problems in processing grammar. However, focal damage in normal,

otherwise healthy children before the age of 5-7 does not produce SLI; it causes language delay followed by recovery to within the normal range (see Bates & Roe, 2001, for a review). Interestingly, the effects of early child brain damage are similar irrespective of side of damage. By contrast, in adults impairments in processing the structural aspects of language only occur after left-sided damage. In short, then, SLI must be viewed as an atypical developmental process, not in terms of damage to pre-existing structures.

However, Ullman and Pierpont's approach highlights the fact that we don't really know what the atypical developmental process looks like (Thomas, 2005b). How does compensation actually work? Why is it not fully successful, in which case the atypical process would evidence no surface behavioural impairments? The implication is that compensatory processes are limited in some respect; but unless the processes are specified in detail, sufficient to make predictions about what level of compensation a given theory would suggest, proposals about compensation cannot be falsified and the attendant theories are untestable. Two recent methodologies have begun to make progress in specifying the nature of compensatory processes.

One of the methodologies is the use of computational models of development to provide formal, implemented simulations of the proposed atypical process (Thomas & Karmiloff-Smith, 2003). This approach begins by building a computational model of normal development for a particular aspect of language acquisition, such as learning to produce past tenses or to parse sentences. The normal developmental trajectory is the consequence both of the linguistic environment to which the system is exposed and its internal computational constraints, such as the nature of its representations and learning algorithm. Manipulations to the linguistic environment and internal computational constraints provide candidate hypotheses to explain

atypical development, if those manipulations are able to deflect the normal trajectory so that it now characterises the pattern observed in a particular disorder.

In this way, Thomas (2005b) demonstrated how altering a computational property in a connectionist model of English past tense acquisition was sufficient to deflect development from the normal trajectory to the SLI profile. This property was the discriminability of the internal processing units (roughly corresponding to the signal-to-noise ratio of a neural processing system). This manipulation was notable for three reasons. First, the property was altered in a processing channel that was shared by both regular inflections (*talk-talked*) and irregular inflections (*drink-drank*), yet it affected regular inflections more seriously than irregulars. This is because good discriminability is necessary to learn the sharp category boundaries in internal representations that will depict rules or regularities. Changes to shared resources can therefore produce uneven deficits to the separate processes that use those resources. Second, changing the processing property at the start of development altered the way the system exploited the information available to it. In the normal system, phonological input was preferentially used to drive regular past tense formation while lexical-semantic (word-specific) information was preferentially utilised to drive irregular past tense formation. In the inefficient, slowly developing atypical system, there was a greater reliance on word-specific lexical-semantic information to drive *all* past tense formation. This led to the emergence of frequency effects in regular past tense formation observed empirically by van der Lely and Ullman (2001); and it is in line with the proposal that all verbs are treated as exceptions in SLI. Third, the model captured SLI accuracy levels in children of around ten years of age. However, the atypical model was then run on to predict adult performance. The results suggested resolution of difficulties on highly practised items, but residual difficulties when the

system came to extend its knowledge to novel cases (i.e., applying the rule). In other words, externally, the system eventually seemed to compensate for highly practised items but internally it failed to normalise.

Using a similar approach, Thomas and Redington (2004) constructed a recurrent connectionist model of sentence processing to simulate the results of an experiment in which participants had to identify the agent and patient of a sentence (Dick et al., 2001). In this task, participants heard sentences that were either canonical (active: *The dog chases the cat*; subject cleft: *It is the dog that chases the cat*) or non-canonical (passive: *The cat is chased by the dog*; object cleft: *It is the cat that the dog chases*) and were required to make a binary choice as quickly as possible on which of two pictures (*dog*, *cat*) corresponded to the agent (*dog*). Dick et al. (2001) found that adults with acquired aphasia exhibited marked difficulties at identifying the agents of non-canonical sentences, that is, both passives and object clefts. When the trained 'adult' connectionist model was lesioned, it too exhibited this pattern of deficits. However, when the same model had its processing resources reduced prior to training to simulate a developmental disorder, it generated a novel prediction that the deficits should be more marked for object cleft sentences than passives.

Let us consider why this should be the case. In the aphasic model, both passive and object cleft failed together because they were low frequency constructions, and therefore less robustly represented in the network. In the atypical model, the resource limitation reduced the ability of the connectionist network to learn information across sequences of words. Object cleft sentences are identified by a noun-noun sequence (*cat* that the *dog*) and so suffered from developmental limitations in sequence processing. However, passive sentences are also (redundantly) identified by lexical cues (past participle *chased* and preposition *by*); across development, the network

learned to use these cues to identify this construction. Importantly, when Dick et al. (2004) extended their paradigm to typically developing children and children with SLI, the results supported the prediction of the model: performance on passives and object clefts was closely related in adult aphasics, while in children with SLI, passive constructions were identified more accurately than object clefts.

These models demonstrate the benefit of implementation for making theories more explicit. Together, the models demonstrate: (1) how adaptive learning systems do the best they can with atypical properties they possess; (2) that compensated systems may use information sources in different ways; and (3) that atypical processing properties may allow compensation for some parts of language but not others.

A second methodology essential to uncover the nature of compensation in developmental disorders is that of functional brain imaging. The computational simulations suggest that, with age and practise, behavioural problems can resolve even though the underlying processes have not normalised. If so, behavioural measures, especially those with poor sensitivity such as standardised tests, may be insufficient to assess developmental outcome. By contrast, functional brain imaging offers a window on the way in which the brain has adapted to perform language tasks when its computational constraints are atypical.

Using this approach, we recently imaged the brain of a 42-year-old man called CK who was diagnosed with SLI aged 6 (Richardson et al., 2006). Donlan et al. (2006) compared the language profile of CK available from standardised tests and educational records when he joined a special school for children with language impairments in 1971, with his performance as an adult in order to explore the eventual outcome of language development. CK's school records indicated a verbal IQ of 69 at

6 years of age, and particular difficulties with auditory memory and morphological inflections. The records note that CK had reduced babbling as a baby, he used only 3 words used at two years of age (girl, pig, stop) and there was then no further productive output until 5 years and 3 months (he started receiving speech and language therapy at 4 years and 11 months). CK's adult profile indicated that some aspects of his language were now within or above the normal range: receptive vocabulary was in the 99th percentile, auditory discrimination was at ceiling, picture comprehension was in the 63rd percentile, and naming showed a z-score of 0.16, i.e., slightly above average. However, CK revealed persisting deficits in tasks requiring phonological working memory: non-word repetition had a z-score of –1.94, well below the normal range, and recall of sentences as in the 1st percentile.

Functional imaging was used to explore brain activations in CK during passive listening to sentences, or reading of sentences presented one word at a time at the same rate, against a baseline of backwards speech or nonsense visual symbols. CK's performance was compared to a group of 14 adult controls. The results revealed that for CK, there was reduced activation in temporal regions normally associated with phonological processing, but *increased* activation in dorsal pre-motor and superior temporal regions, as well as in the caudate nucleus. The latter are all motor areas but note that the task CK was asked to perform included no motor component. One must interpret results of this form with care, since there are at least three ways one could explain the differences between CK and controls: (1) as adaptive compensation; (2) as a failure of the system to inhibit task-irrelevant circuits; (3) as a case of task-irrelevant activations causing interference (though those activations might be adaptive for some other task). Nevertheless, one possible interpretation of the findings is that CK was using additional sub-articulation during comprehension as a compensatory process to

support semantic retrieval during language comprehension. Interestingly, Vargha-Khadem et al. (1998) also reported increased activation in the caudate nucleus in language tasks in the affected members of the KE family. However, those individuals also showed increased activation in Broca's area, a pattern not observed in CK.

In sum, current research of developmental disorders of language is exploiting multiple, interdisciplinary methods, including genetic, computational, and brain imaging in an attempt to better characterise the nature of the atypical developmental process (see Mareschal et al., 2007, for a review of a similar multidisciplinary approach to developmental dyslexia).

5. Conclusion

Developmental disorders of language can exhibit contrasting profiles of strength and weakness. These can be traced to different information streams involved in the task of language learning. The relation of atypical language systems (such as those observed in Williams syndrome and Specific Language Impairment) to the normally developing system remains controversial, but perhaps the best approach is to view them as shedding light on the constraints that shape the learning process. However, the onus then moves onto specifying the detailed nature of this learning process, involving such ideas as redundancy (illustrated in the example of WS) and compensation (illustrated in the example of SLI). New methodologies such as computational modelling and functional brain imaging will be important complements to behavioural studies in this endeavour.

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Figure Captions

Figure 1. Information streams combined in language acquisition, along with developmental disorders in which the primary deficits relate to one of the streams

Figure 2. Cross-sectional developmental trajectories for children with different developmental disorders on two standardised tests (Annaz, 2006). Left panel: British Picture Vocabulary Scale (Dunn et al., 1997); right panel: Pattern Construction from the British Abilities Scales (Elliott et al., 1996). ASD = Autistic spectrum disorder, HF = high functioning, LF = low functioning, DS = Down syndrome, WS = Williams syndrome, TD = typically developing controls

Figure 3. Developmental fractionation of cognition in Williams syndrome: (a) early characterisation: genetic mutation produces simple fractionation between general cognition and language; (b) subsequent research indicates complex pattern of fractionation in both linguistic and non-linguistic domains (Thomas, 2006). Labelled boxes indicate dissociations reported by one or more studies in the literature. Triangles indicate domains in which there is a scale of difficulty, with individuals with WS reported to show exaggerated deficits on harder parts of the domain.

Figure 1.

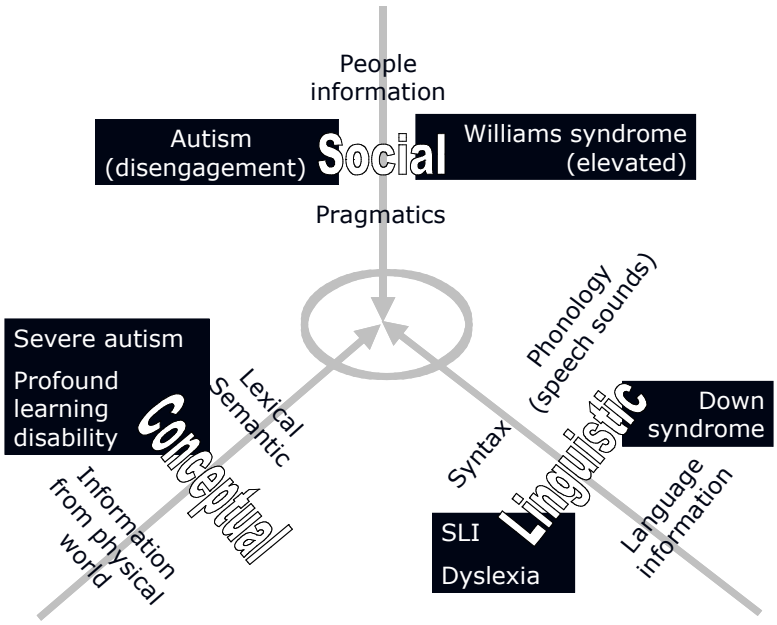
People
information
Autism
(disengagement)
Social
Williams syndrome
(elevated)
Pragmatics
Severe autism
Profound
learning
disability
Conceptual
Lexical
Semantic
Information
from physical
world
Phonology
(speech sounds)
Syntax
Linguistic
Down
syndrome
SLI
Dyslexia
Language
information

Figure 2

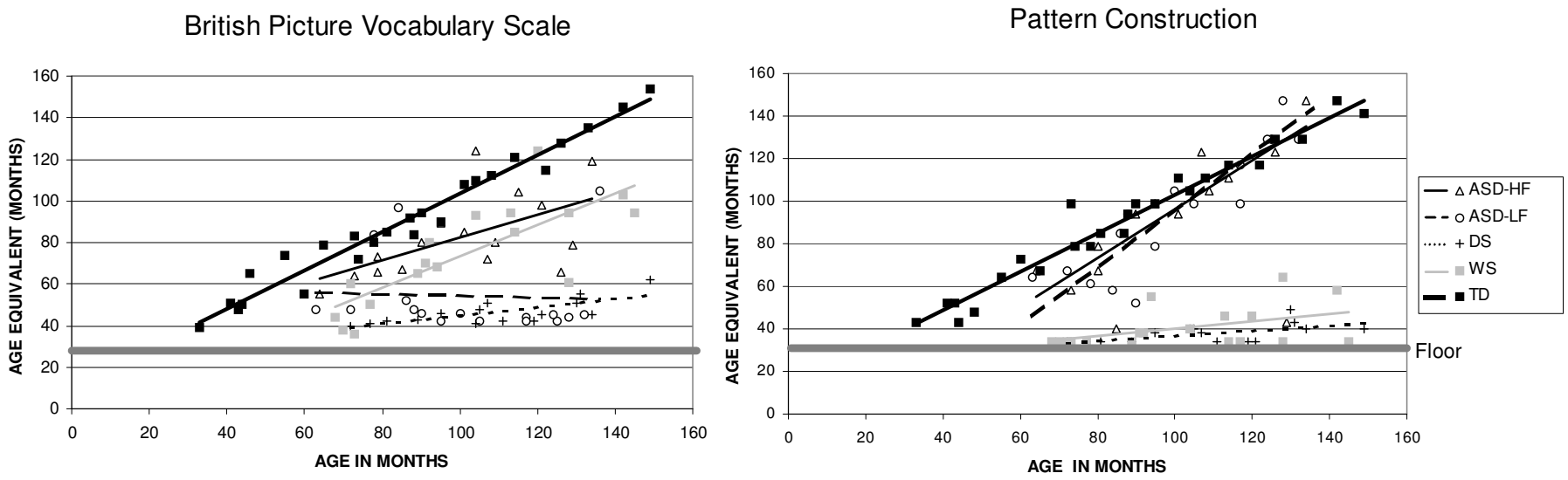
British Picture Vocabulary Scale
AGE EQUIVALENT (MONTHS)
AGE IN MONTHS
0
20
40
60
80
100
120
140
160
Pattern Construction
AGE EQUIVALENT (MONTHS)
AGE IN MONTHS
ASD-HF
ASD-LF
DS
WS
TD
Floor

Figure 3a

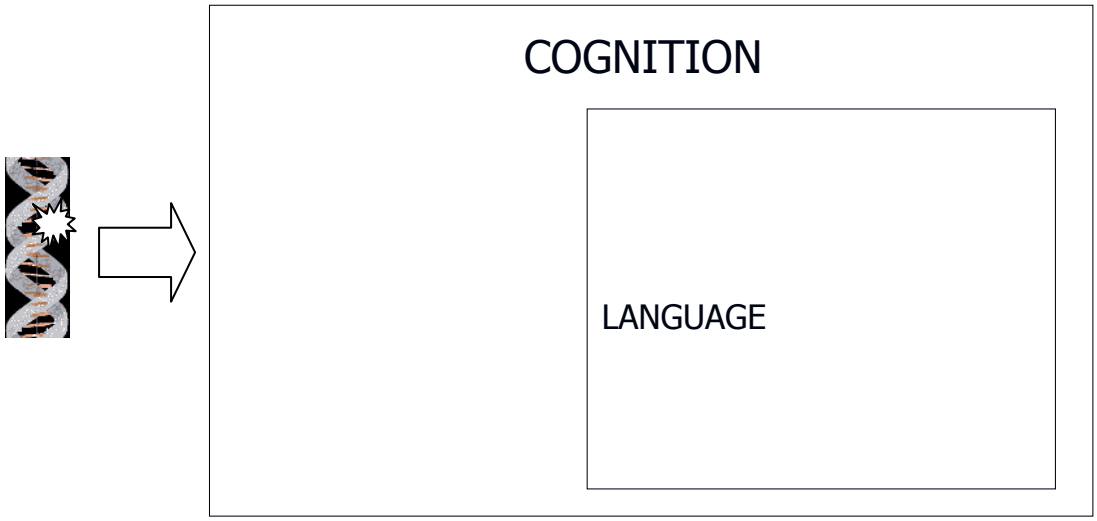

Figure 3b