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Domain-specific cognitive systems: insight from Grammatical-SLI

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Specific language-impairment (SLI) is a disorder of language acquisition in children who otherwise appear to be normally developing. Controversy surrounds whether SLI results from impairment to a 'domain-specific' system devoted to language itself or from some more 'domain-general' system. I compare these two views of SLI, and focus on three components of grammar that are good candidates for domain-specificity: syntax, morphology and phonology. I argue that the disorder is heterogeneous, and that deficits of different subgroups potentially stem from different underlying causes. Interestingly, although poor sensory or non-verbal abilities often co-occur with SLI, there is no evidence that these impairments cause the grammatical deficits found in SLI. Moreover, evidence suggests that impairment in at least one subgroup is specific to grammar.

Introduction

A current debate surrounds whether cognition is organized into domain-specific systems [1–3]. Domain-specific systems, or their underlying mechanisms, are thought to have dedicated neural circuitry that serves one cognitive function. Language is perhaps the best known candidate for a domain-specific system [1,4,5], and one way to test for domain-specific systems is to see whether the developmental course of language dissociates from other cognitive systems.

Specific language-impairment (SLI), a developmental disorder of language, occurs in children who are otherwise developing cognitive abilities normally (see [6,7] for reviews). In contrast to most children, who by 3 years old can talk using simple sentences, children with SLI are prone to make errors (e.g. '*Who did Marge see* ***someone****?*' '*Yesterday I* ***fall*** *over*') [8,9]. Therefore, SLI provides a prima facie candidate for a domain-specific deficit.

SLI has a genetic component [10–12], but the picture is complex. To date, one gene (FOXP2) has been linked to a rare (and non-specific) form of language impairment exhibited by some members of the 'KE' family [10], and loci on chromosomes 16q and 19q have been linked with phonological and expressive grammatical deficits, respectively [11]. These genetic discoveries have propelled SLI to centre stage because of its potential for helping us to understand the unfolding genetic contributions, alongside environmental interactions, to specialized cognitive systems [13]. Furthermore, the different genotypic–phenotypic relations emphasize the need to consider more than one cause of SLI. However, it is important to make a clear distinction between the molecular expression of genes and functional, domain-specific systems. It is only by identifying pertinent SLI phenotypes that we can illuminate functionally specialized cognitive systems.

Investigations of SLI reveal that some children show additional deficits in auditory, other cognitive or even motor abilities [6,7,14,15], suggesting that the disorder, and by implication language systems, are not so specific as once thought. The heterogeneity of the disorder has resulted, historically, from its being studied from a clinical perspective [6] and being defined by exclusion. Recent integration of new theoretical approaches to studying SLI from linguistics, psycholinguistics, neuroscience, molecular genetics and evolutionary biology is bringing finer-grained criteria and better psycholinguistically motivated tests for identifying SLI. These developments are enabling us to identify subgroups within the SLI population, refining our understanding of the disorder.

In this article, I present selective phenotypic data from children with a relatively rare form of SLI, Grammatical(G)-SLI, who show evidence of a discrete grammatical deficit. To evaluate these phenotypic data, I adopt a developmental framework and focus on what I take to be the 'core' deficits (see Box 1). However, to understand the relevance of core deficits, we need to consider that language is the sum of a complex set of systems, only some of which are likely to be domain-specific [4,16]. Strong candidates for domain-specific systems are three components of grammar: syntax, morphology and 'grammatical-phonology' [1,4,5] (Box 1, Figures 1,2). This is because these computational aspects of language show computations, such as recursion or hierarchical non-local dependencies, not found in other cognitive domains [4,5]. In addition, they dissociate from other cognitive functions in acquired disorders [17–19] and (as I will argue below) in developmental disorders [1,20–22]. Focussing on detailed investigations of these aspects of language, I argue that G-SLI provides evidence for a discrete developmental grammatical deficit and thus for domain-specific systems that appear to be selectively impaired [20].

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Box 1. Components of language and SLI

Language is a highly complex system that consists of different subsystems or 'components'. In this article I focus on three components of computational grammar: **syntax** – the structural rules combining words into sentences; **morphology** – the rules combining words or parts of words into new words (e.g. *jump*+*ed*); and **grammatical-phonology** – the rules for combining sounds into words (see Figures 1,2 in main text). In addition to these grammatical components, another essential aspect of language, among many, is the **lexicon** (vocabulary) – the store of words.

Different SLI subgroups evince different degrees of impairment across language components. As a working hypothesis, I take the core impairment to be the most impaired component of language. A core deficit will be significantly below age-matched peers' performance, and often below other language abilities: for example, grammatically impaired children perform significantly worse on tasks that tap aspects of morpho-syntax than younger children matched on vocabulary, or on general measures of grammar (e.g. mean length of utterance, or sentence understanding) [6,8,36,58,61]. Conversely, those with lexical deficits might show the reverse pattern [47,64]. A core deficit does not rule out secondary language impairments.

In the light of recent interdisciplinary work, I evaluate the adequacy of hypotheses from two perspectives to explain the grammatical deficits of children with SLI: the domain-general and domain-specific perspectives. These perspectives reflect different accounts of the origins of specialized systems.

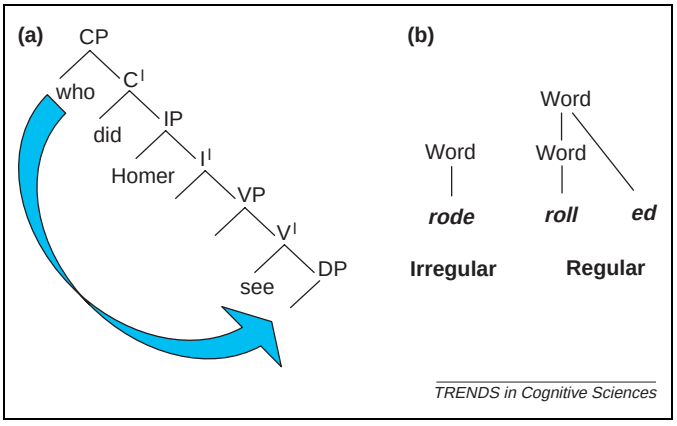

Figure 1. The hierarchical structure in **(a)** syntax and **(b)** morphology. (a) shows the hierarchical arrangement of syntactic structures and the non-local dependency in questions between the wh-word 'who' and the position after the verb that is normally filled by the object noun phrase. CP=complement phrase, IP=inflectional phrase, VP=verb phrase, DP=determiner phrase. (b) illustrates the hierarchical morphological structure of irregular and regular verbs. According to Pinker's Words and Rules model, irregular verbs are stored in memory, whereas morphologically regular (complex) forms are computed using a symbolic rule that takes the verb stem and adds the past tense affix (*roll*+*ed*). Therefore the phonological form (or complexity) of the regular stem is not expected to affect the ability to add the affix *-ed*.

Domain-general perspectives of SLI

Hypotheses of SLI falling under this perspective assume that domain-general or 'domain-relevant' cognitive systems only '*become* domain-specific as a result of processing different kinds of input' ([2] p. 390) – that is, through experience. According to Karmiloff-Smith and colleagues [2,23], no specialized mechanism is genetically specified, and atypical development cannot produce selective deficits. This is because, (i) genetic factors do not determine *a priori* the specificity of mechanisms, (ii) no mechanism is unique to any one system, and (iii) mutual compensation can occur between mechanisms. Hypotheses consistent with these assumptions claim that SLI is caused by underlying 'input-phonology' (speech) and/or processing deficit (Figure 2). Input-phonology is considered to be at the interface between language and either defective auditory processing [6,15,24], phonological short-term memory [25], processing capacity or speed [26–28]. This implies that genetic factors disrupt memory, auditory or general processing. Specifically, slow processing is hypothesized to cause difficulty in processing sounds with rapid acoustic transitions (*t*/*d*) and/or perceiving phonemes with 'low-phonetic salience' (i.e. difficult to hear sounds) (*t*/*d*, *s*/*z*). This affects past tense (*jump**ed***, *play**ed***), agreement/tense (*jump**s***) and plural (*pen**s***) inflections [6].

Domain-specific perspectives of SLI

The alternative, domain-specific, perspective postulates that specialized cognitive mechanisms develop under

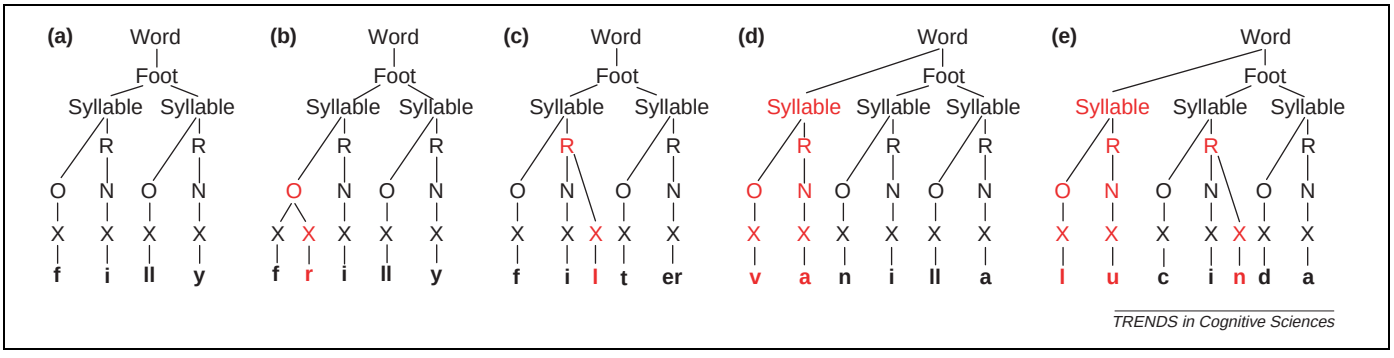

Figure 2. Phonological hierarchical structure: marked prosodic structures. 'Phonology' has a varied definition within the SLI literature, and is applied to (1) 'speech' and articulation processes [42] (which when impaired produce motor-speech problems); (2) the linear organization of sounds (phonemes) into words, in which length, perceptual saliency, and frequency and/or meta-linguistic knowledge determine performance [24,25], and (3) the hierarchical structural organization of sounds into words that is common to all languages [70]. Whereas domain-general theories refer to (1) and (2) domain-specific theories such as the CGC refer to (3). Here we use the term 'input-phonology' for (2), and 'grammatical-phonology' for (3). This latter concept is illustrated in **(a)**. 'Prosodic structure' shows a similar hierarchical arrangement to syntax [68], and can be understood with respect to parameters that regulate syllable and 'metrical' structure: these structures can be either 'marked' (shown in red) **(b–e)** or 'unmarked'. O=Onset; R=Rhyme, N=Nucleus. Marked structures can occur in combination and the greater the number of marked structures the more complex the word or non-word. Many G-SLI children show increasing errors in non-word word repetition as the number of marked parameters increases [31,37]. Moreover, even short but phonologically complex non-words can cause difficulties [37,59].

genetic control and underlie different domains of cognition [1,3,5,19]. According to this view, the unfolding genetic contribution plays a crucial role in determining the neural circuitry underlying specialized cognitive systems. This provides the basis for learning systems such as grammar [16]. Without some genetic organization, no amount of environmental interaction can 'create' the specialized system that allows children to respect the subtle grammatical distinctions necessary for fluent language. This perspective contends that, alongside domain-general systems that can impair language [16,23], genetically controlled domain-specific systems exist and can be selectively impaired.

Hypotheses consistent with these assumptions contend that some forms of SLI are caused by a deficit to certain aspects of grammar [20]. The hypotheses differ in the breadth of the characterization of grammatical deficits, which might either reflect real differences in SLI populations or the fact that the deficit is not fully revealed until late childhood [29–31]. For example, according to the Extended Optional Infinitive (EOI) hypothesis and its variants (the Unique Checking Constraint), genetically guided maturation of the language system is disrupted in young SLI children and affects many language components, but the core deficit is in the part of syntax responsible for accurate tense and agreement marking [9,32] (e.g. *Homer kiss**ed** Marge*). Conversely, according to the deficit in Computational Grammatical Complexity (CGC) hypothesis (a development of the Representational Deficit for Dependent Relations (RDDR) account [30]), children with G-SLI are impaired in the computations underlying hierarchical, structurally-complex forms in one or more component of grammar [31].

Importantly, several assumptions that the domain-specific theories do *not* make are that: (i) there is a one-gene, one-system relation; (ii) each deficit within the grammatical system equates with 'a module' (e.g. 'a module for canonical linkage rules' ([2] p. 389); (iii) domain-specific systems are rigid and unaltered by external input; (iv) the grammar system is mature from day one; or (v) language is a completely encapsulated module that does not rely on other cognitive processes in any respect. Most researchers in the domain-specific camp would agree that both domain-general and domain-specific mechanisms are likely to contribute to specialized systems such as syntax [16,31,33].

The pertinent questions to distinguish these two perspectives are: Do we have evidence that: (1) deficits outside the grammar system cause, or merely co-occur, with SLI?; (2) some mechanisms are specific to grammar or its components?; and (3) such mechanisms can be differentially impaired and their functions not substituted or compensated for by other mechanisms?

Predictions for three components of grammar

A domain-general perspective predicts that SLI should always manifest with particular co-occurring deficits, for example, auditory perceptual or motor impairments [15,34], and that direct relations should arise between these more general deficits (e.g. auditory processing deficits) and grammar or its components [6,28,35]. For example, Leonard and colleagues propose that inflections that add a syllable ('syllabic inflections', e.g. *wan-t**ed**, pu-sh**es**, pu-sh**ing***) are perceptually salient (easy to hear) and therefore easier to process and produce than those that are not syllabic (*push**ed**, want**s***) [28].

Conversely, domain-specific hypotheses argue that the core deficits of some forms of SLI are restricted to the grammatical system. The EOI hypothesis predicts, in particular, that syntactic tense errors (*yesterday he jump_…*) can occur without any other core deficits in the grammar system [9,32]. The CGC hypothesis, by contrast, emphasizes the distinctions between syntactic, morphological and phonological hierarchical structural complexity, and their independent and differential effects on sentence processing and production [31]. This predicts a pervasive deficit in grammatical components determined by structural complexity (see Figures 1,2). Specifically, syntactic complexity can be understood with respect to structural 'non-local dependencies', such as those found between words in questions, for example, '***Who** did Joe see__?*' where ***who*** and the 'gap' following the verb (which in declarative sentences is filled by the object) form a non-local dependency [8] (Figure 1a). Thus, in syntax, not only marking tense, but all structures requiring non-local dependencies are predicted to be problematic. These include passive sentences (e.g. '*Joe was hit by Jill*'), and pronominal reference ('*Bill said Joe hit **him/himself***'). Morphological complexity can be understood with respect to Pinker's 'Words and Rules' model [19], whereby normally developing children store irregular verb forms whole in monomorphemic (simple) forms, but compute morphologically regular (complex) forms using a symbolic rule (*roll+ed*) (Figure 1b). Frequency and phonological properties affect stored irregular words, but have little effect on regular forms. However, the CGC predicts that for children with a morphological deficit, regularly inflected verbs might be preferentially stored, and thus subject to word effects (e.g. frequency) that are typically found for only irregulars. For phonologically impaired children, inflected words such as *roll**ed*** are predicted to be harder than *row**ed***, as phonologically the word-end in *rolled* is more structurally complex.

It is not trivial to distinguish domain-general and domain-specific theories empirically as in many cases they make similar predictions. For example, both predict that *jump**ed*** will be hard (perceptually non-salient, syntactically-affixed tense *-ed* inflection) and *jump**ing*** will be easy (salient, lexically-affixed aspectual *-ing* inflection) [28,36]. Box 2 highlights potential problems in distinguishing these theories. Fortunately though, their predictions on some linguistic phenomena differ markedly, for example in question and past tense formation.

The evidence from empirical data

The empirical data show that, on the one hand, some children with SLI have co-occurring deficits in sensory or non-verbal abilities [15,35]. By contrast, a small but significant number of individuals – the G-SLI subgroup – show a discrete deficit in grammar [20,30,37]. G-SLI is characterized by normal non-verbal abilities and articulation, but a persistent core deficit in the grammatical

Box 2. Interpretative caveats

Processing and representation

Both domain-general and domain-specific hypotheses make claims about sentence processing deficits [8,28]. Research in language acquisition highlights the potential problem of using impaired processing to distinguish the two theories. This is because phonological acquisition alters phonological processing [6,65], morphological acquisition alters morphological processing, and syntactic acquisition alters syntactic processing [66]. Because experimental tasks can never directly tap representations, but only the outcome of some sort of processing, distinguishing the effects of processing versus grammatical deficits is far from obvious. Processing and knowledge are not independent, so impaired processing of sentences or sounds *per se* cannot be taken as evidence for either position: an SLI child's inability to process and remember particular sound sequences might be due to impaired representations of the hierarchical structure of the sound combinations that make up that sequence, as much as to some lower-level processing or memory deficit. More cautious conclusions are required when SLI children show impaired language processing, and finer grained predictions need to be made *a priori* to distinguish the theories.

Relations between language components in acquisition

Although the evidence militates in favour of a domain-specific approach, different components of language can influence one another over the course of development. Indeed, all theories of language acquisition assume that partial knowledge of some components is necessary to acquire parts of others: this is the essence of proposals such as prosodic/phonological bootstrapping for word segmentation and syntactic learning [67], semantic bootstrapping for syntax acquisition [1], and syntactic bootstrapping for semantic learning [68]. Therefore, even when an initially component-specific deficit is postulated, it is predicted that there will be consequences on the acquisition of other components of language, so that totally pure deficits are not expected.

system [20,37] (see Box 3). This therefore constitutes evidence for discrete deficits. But can the domain-general perspective adequately explain the deficits found in syntax, morphology and phonology, notwithstanding the possibility that low-level domain-general impairments could cause, interact with or exacerbate some forms of SLI? This perspective attributes all forms of SLI to deficits in domain-general factors, such as auditory processing speed and memory, that would create 'bad input' to a domain-general processor. Although this theory was popular in the early and mid-1990s, there is now wide-ranging evidence against it.

Heritability

Twin studies reveal that impairments in components of grammar, such as phonology, are highly heritable, but auditory impairments are not [38]. This is unexpected if genetic factors cause SLI by impairing auditory processing, as predicted by a domain-general view [15,24].

Prevalence of auditory deficits

Current studies reveal that many SLI children do not have impaired auditory abilities [14,37,39,40]. In fact, Neville and colleagues' imaging study of language-impaired children revealed that those with grammatical deficits were not the ones with auditory deficits [41]. Interestingly, some children with *normal* language development have impaired auditory abilities [37,39,40], indicating that an auditory deficit is neither sufficient nor necessary to cause SLI [39]. As yet, no form of SLI has been found to co-occur consistently with any type of auditory or non-verbal deficit.

The significance of co-occurring deficits

Many children with language impairments show co-occurring sensory and/or non-verbal deficits [34,35] or even motor immaturity [34], and vice versa, but the data indicate a statistical co-morbidity of impairments rather than a common source [38,42]. If sensory or domain-general deficits cause SLI, we should expect the number or severity of domain-general impairments to correlate with the severity of grammatical deficits. However, both cross-sectional and longitudinal studies show that such co-occurring domain-general deficits have little impact on the nature or severity of grammatical deficits [9,35,37,39,43,44]. For instance, marking tense and understanding passive sentences are developmentally independent from vocabulary and non-verbal abilities [9,35,45].

Dissociations between input-phonology or speech and grammar

Phonologically-impaired children do not necessarily show syntactic deficits, and vice versa [9,22,37,42,46,47]. Of course, it could be that syntactically impaired children had input-phonological or speech impairments earlier in development, but that such impairments are no longer detectable. However, longitudinal studies indicate that children with phonological short-term memory deficits and those with phonological deficits associated with dyslexia or mild-moderate hearing loss do not later exhibit the morpho-syntactic deficits that domain-general hypotheses predict they should [22,43,46–48]. Furthermore, Tomblin and colleagues' large (>7000) epidemiologically obtained sample showed little overlap between speech and language impairments [42].

The effect of phonetic saliency

Inflections that produce an extra syllable (*pu-sh**es**, wan-t**ed***) are more perceptually salient than their non-syllabic counter parts (*cat**s**, miss**ed***) [28], and are more problematic for SLI children [6,49]. Cross-linguistic studies further rule out a low-phonetic-saliency account. For example, in French the same non-salient form (e.g. l*e*) can be either a determiner (*the*) or a pronominal clitic (*him*), but only the clitic requires forming syntactic non-local dependencies for interpretation. French SLI children are impaired in producing and understanding clitic pronouns but not determiners [50].

Thus, when careful comparisons are made between grammatical deficits and other cognitive or motor deficits, the data reveal little evidence for domain-general deficits causing SLI. It could be that domain-general deficits do cause some forms of SLI. But, if so, we need to know the impact of such a deficit on language and which form(s) of SLI result.

Insights from Grammatical-SLI

Whereas the co-occurrence of SLI with other deficits tells us little about the developmental functional autonomy of domain-specific systems, the existence of individuals with

Box 3. Characteristics of the G-SLI subgroup

G-SLI children are defined as having a persistent deficit in syntax and morphology at age 9 years and older. From children who meet the criteria for SLI, based on standardized language and non-verbal tests [6], selection of G-SLI children is based on tests designed to probe core aspects of morpho-syntax; for example, passives, agreement and tense [20,31]. On these tests they have to produce more than 20% errors at an age when normally developing children make few or none. Many G-SLI children are also impaired in grammatical-phonology [37], but their speech for known words is clear and they do not show any articulation impairments [69]. Vocabulary is impaired, but not as significantly as their core grammar impairment [45], and initial investigations indicate that the vocabulary deficit is, at least partially, caused by their grammatical deficits [57]. All children exhibit normal development in other respects including their pragmatic use of language [20,31]. Their performance on non-verbal IQ tests is average, and extensive testing on both non-verbal cognitive and auditory abilities has not revealed any consistent deficits [20,37,40]. Within the SLI population over 9 years old with persisting deficits and normal non-verbal abilities, the prevalence of G-SLI is around 10–20% [44,69]. Investigation of familiar aggregation of language impairments in first-degree relatives is consistent with an autosomal dominant inheritance [69].

G-SLI [20,37] provides insights into this issue. First, the G-SLI subgroup reveals that severe grammatical deficits do not necessarily occur with impairments in non-verbal or auditory abilities [20,37]. Second, alongside SLI more generally [32], G-SLI children show that grammatical impairments, like language acquisition, are not random [8]. Within syntax, G-SLI is a broad but discrete deficit, which includes impairments in marking tense, understanding passives, assigning pronominal reference and producing questions [8,20,30,31] – just those structures requiring non-local dependencies that are core to the syntactic system [4,5]. These findings are supported by data from both typologically close and distant languages (German and French, Hebrew, Greek) [50–54] and by other groups who have identified G-SLI individuals [44,53]. Our studies of the same cohort of children over many years provide evidence that the same deficits are evident at 4–6, 9–12, and 15–18 years of age [20,31,55–57]. Therefore, their deficits persist and are far from compensated for by other mechanisms. However, deficits are often only revealed in test situations when contextual cues and avoidance strategies cannot facilitate performance [8,58]. These data are incompatible with the predictions of a domain-general deficit hypothesis [23].

Structural deficits predicted by the CGC hypothesis are also found in grammatical-phonology [59,60] and morphology [58,61] in G-SLI children. The next section illustrates the impact and cumulative effect of different component deficits.

Evidence for the autonomy of grammatical components

If the CGC hypothesis is correct, then G-SLI will manifest itself in some linguistic forms more than others, as a function of hierarchical structural complexity in each grammatical component. (Note that such complexity is considered to be independent from general cognitive complexity [20,31].) Thus, one reason that tense marking is such a good clinical marker of SLI in English speaking children is because it involves complex hierarchical structures in syntax, morphology and grammatical-phonology. This makes it a good starting point for exploring the potential autonomy but cumulative effects of impairments in these different components.

First, if we take the syntactic properties of tense marking, domain-general theories of SLI predict that because the tense change in irregular verbs is perceptually salient (e.g. *fall–fell*) in contrast to that in regular verbs (e.g. *jump–jump**ed***), irregular forms should not be problematic for SLI children [6]. By contrast, domain-specific theories predict deficits in irregular as well as regular tense marking. The data show that G-SLI children do not tense mark both regular and irregular verbs [31,58], illustrating the syntactic nature of this deficit, and militating against a purely perceptual deficit (cf. [24]).

Second, with respect to the morphological properties of tense, whereas domain-general hypotheses argue for a single system underlying regularly and irregularly marked forms [23,24]; domain-specific accounts argue for separate underlying systems [17,19]. Specifically, both perspectives predict deficits with regular past-tense formation, but they make different predictions for the pattern of performance of children with SLI in comparison with normally developing children. The domain-general hypothesis predicts that SLI children are like normally developing children in using one system to form past-tense forms, so both groups should show the same pattern of performance on regulars compared with irregulars. By contrast, the CGC hypothesis predicts that because normally developing children are using different systems to form the regular and irregular past tense, but G-SLI children might be preferentially storing all forms lexically, the two groups will show a qualitatively different pattern of performance. Data do indeed reveal qualitative differences: (i) G-SLI children perform at a similar level on regular–irregular past-tense marking, whereas normally developing children show a regularity advantage; and (ii) in contrast to normally developing children, G-SLI children show frequency effects for both regular and irregular verbs [58]. Interestingly, the G-SLI children, but not the controls, behave in the way that single-mechanism accounts might predict if only one system underlies tense marking in English. Individuals with a non-specific language impairment (from the KE family) show the same regular and irregular pattern as G-SLI children [34], whereas other SLI children do not [49]. Thus, G-SLI performance is not just worse than that of typically developing children, but is qualitatively different; it is not simply a case of the same system not functioning as well as normal.

We predicted that if G-SLI children preferentially store regular forms whole (like irregular forms) they should use such forms inside compounds (**rat**s**-eater*), whereas ordinarily regulars (but not stored irregulars) are dispreferred in compounds [19]. Furthermore, domain-general hypotheses would predict they should omit these non-salient inflections inside compounds, as they do elsewhere. We discovered that G-SLI children produce regular plurals inside compounds, whereas normally developing children and adults rarely do so [61] (cf. [49]). Thus, G-SLI

children show a consistent and predicted pattern indicating that, alongside their syntactic deficit, they have a morphological deficit.

Turning, third, to phonological structural complexity, a non-word repetition task revealed that most G-SLI children are significantly impaired as a function of increasing phonological structural complexity, whereas younger vocabulary matched controls are not [37,60]. Domain-general hypotheses would predict that performance should be determined by saliency rather than structural considerations [6]. Thus with respect to past tense, we predict that for regular forms, increasing the phonological complexity of the verb-end (vvd-*row**ed***, vcd-*ro**lled***) (see Figure 2) will significantly decrease correct performance in children with SLI who have a phonological deficit, but not in those with normal phonological development. Bortolini and Leonard's study provides initial support for this prediction [62]. They found a significant correlation between levels of final cluster reduction in monomorphemic words and omission of inflections that create clusters word-finally.

These data need to be considered in the context that not all children with SLI exhibit morphological and/or phonological deficits [47,49,53,63], and not all children with phonological deficits reveal the morpho-syntactic deficits typically found in SLI [22,42,43,46].

Conclusions

The heterogeneity of SLI phenotypes indicates that the impairment can have multiple causes [9,20] and to talk of a unified disorder is increasingly untenable (see also Box 4). The existence of a subtype of SLI specific to grammar provides a challenge to domain-general theories: how could such discrete deficits that persist into adulthood [8,20,31] exist if the systems underlying grammar are 'domain-relevant' and can be compensated for?

Empirical data from numerous SLI investigations, and particularly from G-SLI, suggest that developmental deficits in grammar are best accounted for by the hypothesis that the brain contains domain-specific systems. I have argued that deficits in each of three components of grammar (syntax, morphology, phonology) can co-exist, and might all dissociate [31]. The challenge is now to provide a full picture of the individual phenotypes, and subsequently unravel the underlying genetic and molecular variations that ultimately contribute to these phenotypes.

Box 4. Questions for future research

- What genotypes underlie the different SLI phenotypes?
- Can a child with SLI achieving 80% correct performance on a grammatical task at age 14 (and never improving further), and a typically developing child achieving 95% at age 4–5 be using the same grammatical system(s)? And if not, what does this tell us about the specialization of systems?
- How do deficits in different grammatical components affect lexical learning?
- Do grammatical deficits in different disorders manifest themselves in the same way, for example, in complex structures in the components?
- What are the neural correlates of syntactic, morphological or phonological processing in SLI individuals, and how does impaired grammatical development affect the structure of the brain?

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References

1 Pinker, S. (1994) *The Language Instinct*, W. Morrow & Co.
2 Karmiloff-Smith, A. (1998) Development itself is the key to understanding developmental disorders. *Trends Cogn. Sci.* 2, 389–398
3 Fodor, F.J. (1983) *The Modularity of Mind*, MIT Press
4 Hauser, M.D. *et al*. (2002) The faculty of language: What is it, who has it, and how did it evolve? *Science* 298, 1569–1579
5 Chomsky, N. (1986) *Knowledge of Language: Its Nature, Origin and Use*, Praeger
6 Leonard, L. (1998) *Children with Specific Language Impairment*, MIT Press
7 Bishop, D.V.M. (1997) *Uncommon Understanding: Comprehension in Specific Language Impairment*, Psychology Press
8 van der Lely, H.K.J. and Battell, J. (2003) Wh-movement in children with grammatical SLI: A test of the RDDR hypothesis. *Language* 79, 153–181
9 Rice, M. *et al*. (2004) Grammatical tense deficits in children with SLI and nonspecific language impairment: Relationships with nonverbal IQ over time. *J. Speech Lang. Hear. Res.* 47, 816–834
10 Lai, C. *et al*. (2001) A forkhead-domain gene is mutated in a severe speech and language disorder. *Nature* 413, 519–523
11 SLI-Consortium. (2002) A genomewide scan identifies two novel loci involved in specific language impairment. *Am. J. Hum. Genet.* 70, 384–398
12 Stromswold, K. (1998) Genetics of spoken language disorders. *Hum. Biol.* 70, 297–324
13 Marcus, G.F. and Fisher, S.E. (2003) FOXP2 in focus: What can genes tell us about speech and language? *Trends Cogn. Sci.* 7, 257–262
14 Bishop, D.V.M. and Mcarther, G.A. Individual differences in auditory processing in specific language impairment: A follow-up study using event-related potentials and behavioural thresholds. *Cortex* (in press)
15 Tallal, P. (2002) Experimental studies of language learning impairments: From research to remediation. In *Speech and Language Impairments in Children* (Bishop, D. and Leonard, L., eds), *Speech and Language Impairments in Children*, pp. 131–156
16 Marcus, G. (2004) *The Birth of the Mind: How a Tiny Number of Genes Creates the Complexities of Human Thought*, Basic Books
17 Marslen-Wilson, W. and Tyler, L. (1997) Dissociating types of mental computation. *Nature* 387, 592–594
18 Cuetos, F. *et al*. (2000) Dissociation of semantic and phonological errors in naming. *Brain Lang.* 75, 451–460
19 Pinker, S. (1999) *Words and Rules: The Ingredients of Language*, Weidenfeld & Nicolson
20 van der Lely, H.K.J. *et al*. (1998) Evidence for a grammar-specific deficit in children. *Curr. Biol.* 8, 1253–1258
21 Clahsen, H. and Almazan, M. (1998) Syntax and morphology in Williams Syndrome. *Cognition* 68, 167–198
22 Ramus, F. (2003) Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? *Curr. Opin. Neurobiol.* 13, 212–218
23 Thomas, M. and Karmiloff-Smith, A. (2002) Are developmental disorders like cases of adult brain damage? Implications from connectionist modelling. *Behav. Brain Sci.* 25, 727–750
24 Joanisse, M.F. and Seidenberg, M.S. (2003) Phonology and syntax in specific language impairment: evidence from a connectionist model. *Brain Lang.* 86, 40–56
25 Gathercole, S. *et al*. (1999) Phonological short-term memory and vocabulary development: Further evidence on the nature of the relationship. *Appl. Cogn. Psychol.* 13, 65–77
26 Chiat, S. (2001) Mapping theories of developmental language impairment: Premises predictions and evidence. *Lang. Cogn. Process.* 16, 113–142

27 Miller, C. *et al*. (2001) Speed of processing in children with specific language impairment. *J. Speech Lang. Hear. Res.* 44, 416–433
28 Montgomery, J.W. and Leonard, L.B. (1998) Real-time inflectional processing by children with specific language impairment: Effects of phonetic substance. *J. Speech Lang. Hear. Res.* 41, 1432–1443
29 Rice, M. (2004) Language growth of children with SLI and unaffected children: Timing mechanisms and linguistic distinctions. In *Proceedings of the 28th Annual Boston University Conference on Language Development* (Vol. 1) (Brugos, A. *et al*., eds), *Proceedings of the 28th Annual Boston University Conference on Language Development*, pp. 28–49, Cascadilla Press
30 van der Lely, H.K.J. (1998) SLI in children: Movement, economy and deficits in the computational syntactic system. *Language Acquisition* 72, 161–192
31 van der Lely, H.K.J. (2004) Evidence for and implications of a domain-specific grammatical deficit. In *The Genetics of Language* (Jenkins, L., ed.), pp. 117–144, Elsevier
32 Wexler, K. (2003) Lenneberg's Dream: Learning, normal language development, and specific language impairment. In *Language Competence Across Populations: Toward a Definition of Specific Language Impairment* (Levy, Y. and Schaeffer, J., eds), pp. 11–62, Erlbaum
33 Dominey, P. and Ramus, F. (2000) Neural network processing of natural language: I. Sensitivity to serial, temporal and abstract structure in the infant. *Lang. Cogn. Process.* 15, 87–127
34 Watkins, K.E. *et al*. (2002) Behavioural analysis of an inherited speech and language disorder: comparison with acquired aphasia. *Brain* 125, 452–464
35 Norbury, C. *et al*. (2002) Does impaired grammatical comprehension provide evidence for an innate grammar module? *Appl. Psycholing.* 23, 247–268
36 Rice, M.L. *et al*. (1999) Grammaticality judgments of an extended optional infinitive grammar: Evidence from English-speaking children with specific language impairment. *J. Speech Lang. Hear. Res.* 42, 943–961
37 van der Lely, H.K.J. *et al*. (2004) Grammatical language impairment and the specificity of cognitive domains: Relations between auditory and language abilities. *Cognition* 94, 167–183
38 Bishop, D.V. *et al*. (1999) Different origin of auditory and phonological processing problems in children with language impairment: evidence from a twin study. *J. Speech Lang. Hear. Res.* 42, 155–168
39 Bishop, D.V. *et al*. (1999) Auditory temporal processing impairment: neither necessary nor sufficient for causing language impairment in children. *J. Speech Lang. Hear. Res.* 42, 1295–1310
40 Rosen, S. (2003) Auditory processing in dyslexia and specific language impairment: Is there a deficit? What is its nature? Does it explain anything? *J. Phonetics* 31, 509–527
41 Neville, H.J. *et al*. (1993) The neurobiology of sensory and language processing in language-impaired children. *J. Cogn. Neurosci.* 5, 235–253
42 Shriberg, L.D. *et al*. (1999) Prevalence of speech delay in 6-year-old children and comorbidity with language impairment. *J. Speech Lang. Hear. Res.* 42, 1461–1481
43 Norbury, C. *et al*. (2001) Production of English finite verb morphology: A comparison of SLI and mild-moderate hearing impairment. *J. Speech Lang. Hear. Res.* 44, 165–178
44 Bishop, D. *et al*. (2000) Grammatical SLI: A distinct subtype of developmental language disorder? *Applied Psycholinguistics* 21, 159–181
45 van der Lely, H.K.J. (1996) Specifically language impaired and normally developing children: Verbal passive vs. adjectival passive sentence interpretation. *Lingua* 98, 243–272
46 Gathercole, S. *et al.* Developmental consequences of phonological loop deficits during early childhood: A longitudinal study. *J. Child Psychol. Psychiatry* (in press)
47 Nation, K. *et al*. (2004) Hidden language impairments in children: parallels between poor reading comprehension and specific language impairment? *J. Speech Lang. Hear. Res.* 47, 199–211
48 Snowling, M.J. (2000) *Dyslexia*, Blackwell
49 Oetting, J. and Rice, M. (1993) Plural acquisition in children with specific language impairement. *J. Speech Hear. Res.* 36, 1241–1253
50 Jakubowicz, C. *et al*. (1998) Determiners and clitic pronouns in French-speaking children with SLI. *Language Acquisition* 7, 113–160
51 Hamann, C. *et al.* Aspects of grammatical development in young French children with SLI. *Dev. Sci.* (in press)
52 Hamann, C. *et al*. (1998) German impaired grammar: The clause structure revisited. *Language Acquisition* 7, 193–245
53 Friedmann, N. and Novogrodsky, R. The acquisition of relative clause comprehension in Hebrew: A study of SLI and normal development. *J. Child Lang.* (in press)
54 Stavrakaki, S. (2002) A-bar movement constructions in Greek children with SLI: evidence for deficits in the syntactic component of language. In *Clinical Linguistics: Theory and Applications in Speech Pathology and Therapy. Current Issues in Linguistic Theory* (Fava, E., ed.), pp. 131–153, John Benjamins
55 van der Lely, H. and Dewart, H. (1986) Sentence comprehension strategies in specifically language impaired children. *Br. J. Disord. Commun.* 21, 291–306
56 van der Lely, H.K.J. and Harris, M. (1990) Comprehension of reversible sentences in specifically language impaired children. *J. Speech Hear. Disord.* 55, 101–117
57 van der Lely, H.K. (1994) Canonical linking rules: Forward versus reverse linking in normally developing and specifically language-impaired children. *Cognition* 51, 29–72
58 van der Lely, H.K.J. and Ullman, M. (2001) Past tense morphology in specifically language impaired children and normally developing children. *Lang. Cogn. Process.* 16, 113–336
59 Marshall, C.R. *et al*. (2002) Investigating the impact of prosodic complexity on the speech of children with Specific Language Impairment. In *UCL Working Papers in Linguistics* (Vol. 14) (Vermeulen, R. and Neeleman, A., eds), *UCL Working Papers in Linguistics*, pp. 43–68, University College Press
60 Marshall, C. *et al*. (2003) The nature of phonological representations in children with Grammatical-Specific Language Impairment (G-SLI). In *The University of Cambridge First Postgraduate Conference in Language Research* (Hall, D. *et al*., eds), pp. 511–517, Cambridge Institute of Language Research
61 van der Lely, H.K.J. and Christian, V. (2000) Lexical word formation in children with grammatical SLI: a grammar-specific versus an input-processing deficit? *Cognition* 75, 33–63
62 Bortolini, U. and Leonard, L.B. (2000) Phonology and children with specific language impairment: status of structural constraints in two languages. *J. Commun. Disord.* 33, 131–149
63 Oetting, J.B. and Horohov, J.E. (1997) Past-tense marking by children with and without specific language impairment. *J. Speech Lang. Hear. Res.* 40, 62–74
64 Dockrell, J.E. *et al*. (1998) Children with word-finding difficulties. *Int. J. Lang. Commun. Disord.* 33, 445–454
65 Pallier, C. *et al*. (1997) Language-specific listening. *Trends Cogn. Sci.* 1, 129–132
66 Hahne, A. and Friederici, A. (2001) Processing a second language: Late learners' comprehension mechanisms as revealed by even-related brain potentials. *Bilingualism: Language and Cognition* 4, 123–141
67 Morgan, J.L. and Demuth, K. eds (1995) *Signal to Syntax: Bootstrapping from Speech to Grammar in Early Acquisition*, Erlbaum
68 Gleitman, L. (1990) The structural source of verb meaning. *Lang. Acquisition* 1, 3–55
69 van der Lely, H.K.J. and Stollwerck, L. (1996) A grammatical-specific language impairment in children: An autosomal dominant inheritance? *Brain Lang.* 52, 484–504
70 Harris, J. (1994) *English Sound Structure*, Blackwell