20 Grammatical-Specific Language Impairment: A Window Onto Domain Specificity

Heather van der Lely and Chloë Marshall

INTRODUCTION

There has been much debate as to which aspects of language are specific to language rather than shared with other aspects of cognition, and which aspects of language are specific to humans rather than shared with other groups of animals (Hauser, 2001; Hauser, Chomsky, & Fitch, 2002; Pinker & Jackendoff, 2005). In addition, there continues to be much discussion about how this specialized language system is represented and develops in the brain. New insight into this debate comes from studying people with a developmental language disorder, Specific Language Impairment (SLI), and particularly a subtype of this disorder known as Grammatical (G)-SLI. Such insight is bidirectional: our growing understanding of language and brain-systems enhances and directs our line of enquiry into SLI, furthering our knowledge of the underlying nature of both typical and atypical language development. In turn, this informs our enquiries about language and brain systems.

In this context, our chapter reviews the findings from G-SLI, and aims to contribute to our understanding both of specialized cognitive systems, specifically language, and of the nature of typical and atypical language acquisition. We argue that our data provide evidence that certain aspects of grammar are domain-specific and can be selectively impaired.

THEORETICAL FOUNDATIONS

SLI: What Do We Agree On?

SLI is a disorder of language acquisition in children, in the absence of any obvious language-independent cause, such as hearing loss, low nonverbal IQ, motor difficulties, or neurological damage (Bishop, 1997; Leonard, 1998). Children, teenagers, or even adults with SLI may produce sentences such as "Who Marge saw someone?" (van der Lely & Battell, 2003) or "Yesterday I fall over" (Leonard, Dromi, Adam, & Zadunaisky-Ehrlich, 2000; Rice, 2003; van der Lely & Ullman, 2001), or may fail to correctly interpret sentences such as "The man was eaten by the fish" (Bishop, 1982; van der Lely, 1996). SLI is one of the most common developmental disorders, affecting around 7% of children in its "pure form" (Tomblin et al., 1997), and the prevalence is even greater when children with co-occurring impairments (e.g., Autism, ADHD) are included.

The disorder heterogeneously affects components of language such as syntax, morphology, phonology and, often to a lesser extent, the lexicon. It has a strong genetic component as shown by familial aggregation studies (for a review see Stromswold, 1998), twin studies (Bishop & Bishop, 1998), and genetic analyses (Fisher, Lai, & Monaco, 2003). The current view is that SLI is likely to have a complex geno-phenotypic profile, with different genetic forms of the disorder causing different phenotypes and possibly even the same genotype resulting in varied phenotypes (Fisher et al., 2003).

SLI: What Don't We Agree On?

It is perhaps not surprising that, given the heterogeneous nature of SLI, there is disagreement over two areas: a taxonomy of SLI, and its cause at the cognitive level.

The discussion surrounding the taxonomy of SLI concerns the variation and number of component deficits in children who fall under the broad umbrella of "SLI." Broadly speaking, the following disorders and language component deficits are found in the literature. Note that we are focusing on the 7% of children who fall within the "typical definition" of SLI; that is, those who do not have co-occurring problems. It is the particular combination or specification of component deficits that characterizes the various definitions of SLI, some of which are considered SLI subgroups. On the one hand, Bishop and Snowling (2004) reviewed children with SLI who have a double deficit of phonological deficits plus "language impairments." We understand this to mean some or perhaps any other component impairments within the language system. Therefore, we might expect a range of different profiles in the children they study. In contrast to this group of SLI, a highly restrictive subgroup are those called "Syntactic-SLI," characterized by only syntactic and morphosyntactic impairments (Friedmann & Novogrodsky, 2004). The "Grammatical(G)-SLI" subgroup, however, is defined by similar core impairment in syntax and morphology, but in addition the majority suffer from phonological impairment too (Gallon, Harris, & van der Lely, 2007). Even within these component deficits in syntax, morphology and phonology, the deficit is restricted to structures that are hierarchically complex, as we will discuss in due course. Interestingly, the distinction between phonology on the one hand and syntax and morphology on the other appears to be relevant to geno-phenotypic associations. Whereas a locus on Chromosome 16, now identified as CNTNAP2 is associated with children with phonological deficits and/or phonological memory deficits, a locus on Chromosome 19 is associated with expressive grammatical impairments, with no significant overlap between the groups (Bishop, Adams, & Norbury, 2006; Vernes et al., 2008).

In contrast, a subgroup who appear to have relative strengths in the grammatical aspects of language are those with "Pragmatic(P)-SLI," who, as their name implies, have impaired pragmatic abilities (Bishop & Norbury, 2002). There is one subgroup—which we shall refer to as Familial-SLI (of which the most famous is the KE family), which has an identified simple genotype—a mutation of the gene FOXP2 with a simple autosomal, dominant inheritance—but which results in a rather complex phenotype (Fisher et al., 2003). Familial-SLI is characterized by not only impairments in language components, most notably morpho-syntax, morphology, phonology as well as the lexicon, but also outside the language system. Specifically the impaired family members have an oral dyspraxia concerning problems in motor programming of fine articulatory movements for speech sounds (Fisher & DeFries, 2002; Watkins, Dronkers, & Vargha-Khadem, 2002). It is also of note that some members of the KE family have a low IQ; however low IQ doesn't segregate with the language impairment (Fisher & DeFries, 2002). In other children, low IQ is frequently (but not always) associated with a delayed pattern of language development, which we shall call the "delayed language" subgroup (Rice, 2004). Children in this group often exhibit delayed vocabulary acquisition and immature pragmatic development, alongside other language impairments (Rice, 2004).

In addition, many children with a diagnosis of SLI also have Dyslexia, and vice versa. However, these two disorders are not synonymous, and can occur in isolation. For the majority of children with Dyslexia, impairments center around the phonological component, either with the Phonological representations themselves, or with accessing and/or manipulating those representations (Ramus & Szenkovits, 2008; Snowling, 2000). Whether such phonological deficits are identical or different to those found in SLI children is the focus of much current research (de Bree, 2007; Marshall, Harcourt-Brown, Ramus, & van der Lely, 2009; Marshall & van der Lely, 2009 and papers in Messaoud-Galusi & Marshall 2010).

The second area of discussion concerns the cognitive cause of SLI. Given the considerable heterogeneity in the phenotype, it could well be that different cognitive causes and pathways underlie different forms of SLI. But equally, there could be different routes to a particular surface impairment, for example, omission of tense marking. In this context, we now consider the two predominant approaches to the cognitive origins of SLI that can be broadly characterized as the domain-general and domain-specific perspectives.

Domain-General (D-G) deficit proponents identify the primary deficit in general cognitive mechanisms such as temporal discrimination, lower-level sensory processing speed, processing capacity, and short-term memory (Bishop, 1997; Leonard, 1998; Montgomery, 2000; Tallal, 2002). These deficits are considered to impair auditory processing of nonspeech and speech sounds, or alternatively memory and/or general learning. The resulting phonological deficit in turn causes problems in language learning (Joanisse & Seidenberg, 1998). Although the primary source of the domain-general deficit varies across different accounts, they share the common view that the underlying deficit is not in mechanisms specific to grammar, but in lower-level processing or later nongrammatical cognitive processing. Note that whereas temporal deficits alone are proposed by some to be sufficient to cause SLI (Tallal et al., 1996), short-term memory deficits are thought to cause SLI only when found in combination with other impairments (Gathercole, 2006). Within this framework, Joanisse provides perhaps the most clearly specified model starting from an auditory or speech processing deficit, and he describes the resulting developmental trajectory of SLI (Joanisse, 2004; Joanisse, 2007).

Domain-Specific (D-S) deficit proponents, in contrast, claim that in some children the deficit affects the development of neural circuitry underlying the components of grammar (Bishop et al., 2006; Friedmann & Gvion, 2002; Rice, 2003; van der Lely, Rosen, & Adlard, 2004; van der Lely, Rosen, & McClelland, 1998). Thus, although both D-G and D-S mechanisms are likely to contribute to language (Gathercole, 2006; Jakubowicz & Strik, 2008; Marcus, 2004), SLI is thought to be caused by deficits to specialized computational mechanisms underlying grammar processing itself. Although a number of hypotheses have been proposed from this perspective, the Computational Grammatical Complexity (CGC) hypothesis (van der Lely, 2005), provides a framework for our research and investigations of the G-SLI subgroup. Specifically the CGC claims that the deficit is in hierarchical structural knowledge that is core to the computational grammatical system. Our work reveals that many school-aged children and teenagers with G-SLI lack the computations to consistently form hierarchical, structurally complex forms in one or more components of grammar that normally develop between 3 and 6 years of age. This working hypothesis emphasizes the notion that impairments in syntax, morphology, and phonology are functionally autonomous, but cumulative in their effects (Marshall & van der Lely, 2007a, b; van der Lely, 2005)

Figure 20.1 illustrates this model and in the following sections, we will elaborate on the characterization for these three components of grammar and how they affect language. For the purposes of this paper Figure 20.1 only shows the arrows that are key to the discussion below. Based on this component model of language impairment the CGC predicts that there is no causal relation between lower-level auditory abilities or short-term memory abilities and grammatical development, although these abilities, as with typically developing (TD) children, contribute to general language performance. These relations are shown by lines rather than arrows.

We will now consider the CGC in more detail with respect to each of the three impaired grammatical components, syntax, morphology, and phonology, and present evidence that the impairment in the G-SLI subgroup is domain-specific.

SYNTAX

The CGC claims that the deficit in hierarchical structure is characterized by impairment in syntactic dependencies. Specifically, whereas dependences within

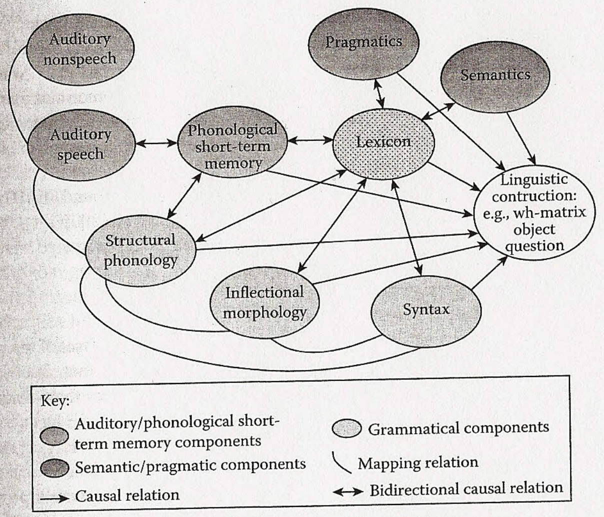

FIGURE 20.1 A component model of language acquisition and impairment. Note for clarity, only lines pertinent to the discussion have been included. There are clearly many more relations between components than depicted.

the phrase are preserved (e.g., agreement), those outside the phrase but within the clause are impaired. Broadly speaking, this can be characterized by what Chomsky terms "movement" or "feature checking"(Chomsky, 1998) or in current terminology "internal merge" (Chomsky, 2004). The impairment affects a large number of structures. For example, tense marking is impaired, resulting in errors such as "*Yesterday I walk to school*" (van der Lely & Ullman, 2001). Such errors have been eloquently described and explored by Wexler and Rice and colleagues and have led them to claim that this "extended optional infinitive" phase is the primary impairment within syntax (Rice, Wexler, & Redmond, 1999). However, the CGC claims that, at least for the G-SLI subgroup, their impairment with syntactic dependencies also affects assignment of theta roles, particularly when more general pragmatic and world knowledge is not available to facilitate interpretation, such as in reversible passive sentences (*The man was eaten by the fish*), or when assigning reference to pronouns or anaphors within sentences (*Mowgli said Baloo was tickling him/himself*; van der Lely, 1994, 1996; van der Lely & Stollwerck, 1997), as well as embedded sentences and relative clauses. The nature of the G-SLI children's syntactic deficits is clearly illustrated in a series of studies of wh-questions. Object matrix and embedded questions are particularly problematic in English because the wh-word has to move from the end of the sentence to the beginning, and "do support" requires checking of tense and question features.

Thus, G-SLI children produce questions such as "Who Joe see someone?" (van der Lely Battell, 2003) and judge such sentences to be grammatical (van der Lely Jones & Marshall, in press). They also make "copying" errors of the wh-word in embedded questions as in (1) (Archonti, 2003). This pattern is sometimes found in young children (Thornton, 1995), suggesting that such structures are syntactically simpler and easier.

(1) "Who did Joe think who Mary saw?"

Furthermore, using a cross-modal priming paradigm, we found that G-SLI children showed no reactivation at the "gap" [marked by **"t"** in (2)] in preposition object questions in scenarios such as (2), in contrast to age and language matched control groups (Marinis & van der Lely, 2007).

(2) Balloo gives a long carrot to the rabbit.

"Who$_i$ did Balloo give the long carrot to $\mathbf{t_I}$ at the farm?"

However, we found reactivation for the G-SLI children at the offset of the verb, where subcategorized arguments might be activated, suggesting that, in contrast to their peers, they were using semantic-lexical processing rather than syntactic processing (Marinis & van der Lely, 2007).

Following these findings, our next question was "Is there evidence at the brain level of impaired processing of just these syntactic dependencies, but normal functioning in other language processes?" If this was so, it would provide good evidence for the domain-specificity of this particular syntactic operation. However, the alternative domain-general hypothesis would be supported if brain correlates showed generally slow or abnormal characteristics to all (or most) language and/or auditory processing. To investigate these alternative hypotheses we recorded electrophysiological time-locked, event-related brain potentials (ERPs) in 18 G-SLI participants aged 10–21 years, plus age-matched, language-matched, and adult controls, when they were listening to questions containing a syntactic violation. The particular syntactic violation we were interested in concerns structural syntactic dependencies at the clause level such as those that occur between a question word (*who, what*) and the word, which in declarative sentences follows the verb, but typically is absent in questions. In our particular design, the first possible "wh-word-gap" following the verb was filled. Pretesting of the sentences (see 3a) indicated that, at the critical noun following the verb to which our EEG recordings were time-locked, the listener would perceive the word as a violation. This is because, if the gap is filled, the animacy property of the noun should mismatch that of the wh-word (see 3b). Therefore, an animacy match (3a) was highly unexpected (a syntactic violation), where as the amimacy mismatch (3b) provided the control condition.

(3) a) **Who** did the man push the **clown** into? (violation)

b) **What** did the man push the **clown** into? (control)

Crucially, the syntactic violation relied on a structural syntactic dependency between two nonadjacent words in the sentence. What is at issue here is not merely to know whether the children noticed the violation/unexpectancy of the noun, but to identify the different functional neural circuitries that are used to detect such a violation/unexpectancy. We found an "Early Left Anterior Negativity" (ELAN), a language-specific neural correlate associated with structural syntactic violations (Friederici, 2002), in all the control groups but not in the G-SLI children. The electrophysiological brain responses revealed a selective impairment to only this neural circuitry that is specific to grammatical processing in G-SLI. The participants with G-SLI appeared to be partially compensating for their syntactic deficit by using neural circuitry associated with semantic processing (N400), and all nongrammar-specific (N400, P600) and low-level auditory neural responses (N1, P2, P3) were normal (Fonteneau & van der Lely, 2008). Thus, we found that the G-SLI children did indeed notice the violation, but they were using a different brain system to do this compared to the control subjects. The findings indicate that grammatical neural circuitry underlying this aspect of language is a developmentally unique system in the functional architecture of the brain, and this complex higher cognitive system can be selectively impaired.

In summary, our syntactic findings from G-SLI show a consistent impairment in syntactic dependencies outside the phrase but within the clause, which are manifest across a broad range of structures. The findings from the cross-modal priming and ERP studies indicated that G-SLI children use semantic mechanisms to compensate for their syntactic deficits. The use of such a semantic system leads to the speculation that the optional pattern of performance for syntax that is commonly reported in the literature results from this imprecise form of sentence processing, rather than an optional functioning of the syntactic mechanism(s). Such imprecise processing may not sufficiently restrict interpretation or production. Further research is warranted to explore this possibility. Note that children who do not necessarily fit the G-SLI criterion exhibit syntactic impairments with similar characteristics in both English speaking children (Bishop, Bright, James, Bishop, & van der Lely, 2000; Norbury, Bishop, & Briscoe, 2002; O'Hara & Johnston, 1997) and languages that are typologically different from English, such as French, Greek, and Hebrew (Friedmann & Novogrodsky, 2004, 2007; Jakubowicz, Nash, & van der Velde, 1999; Stavrakaki, 2001; Stavrakaki & van der Lely, 2010). The largely similar characteristics of the component deficit across different SLI subgroups is also illustrated in morphology and indeed phonology, which we turn to in the next sections.

MORPHOLOGY

Within the morphology component, the CGC hypothesizes a hierarchical deficit that impacts on morphologically complex forms. Thus, over and above the syntactic deficits that affect both irregular and regular tense marking, regular forms are particularly problematic for children with G-SLI. Investigations using both elicited production (e.g., *Every day I walk to school, Yesterday I . . .*) and grammaticality judgements of correct and incorrect forms reveal a lack of the regularity advantage found in typically developing children (van der Lely & Ullman, 1996, 2001). Furthermore, individuals with G-SLI show an atypical pattern of frequency effects for both irregular *and* regular past tense forms. The Words and Rules model of past tense forms (Marcus et al., 1992; Pinker, 1999) provides a parsimonious explanation for these data: for typically developing children, irregular, morphologically simple forms are stored, which leads to frequency effects, whereas regular, morphologically complex forms are computed online and show no frequency effects. Van der Lely and Ullman (2001) therefore hypothesized that G-SLI children preferentially store regular inflected forms whole, as they do irregular forms. Thus, phonological-to-semantic mapping is key to the learning and processing of both regular and irregular forms.

We report two direct tests of van der Lely and Ullman's hypothesis. The first test explored plural compounds, building on Gorden's study of young children (Gordon, 1985). Gordon showed that 3–5 year olds use irregular plurals inside compounds, such as *mice-eater*, but avoid using regular plurals to create forms such as *rats-eater*. This is because only stored forms can enter the compounding process (Gordon, 1985). Therefore, we hypothesized that if G-SLI children were storing regular plural forms whole, then these would be available to the compounding process, just like irregular plurals. Consistent with our hypothesis, G-SLI children, in contrast to language-matched controls, produced compound forms such as *rats-eater* (van der Lely & Christian, 2000).

The second test of the hypothesis considered the phonotactics of regular past tense marking, and specifically the clusters formed at the verb end when the suffix is added. Some of these clusters also occur in monomorphemic words, for example the cluster at the end of *missed* (*mist*) and *scowled* (*cold*), and we refer to these as "monomorphemically legal clusters." In contrast, some clusters, such as those at the end of *slammed*, *robbed,* and *loved*, only occur in morphologically complex words (past tense or past participles), and we call these "monomorphemically illegal clusters." Because these illegal clusters only occur in inflected words, their frequency is much lower than that of legal clusters. Thus if, as we hypothesized, G-SLI children are storing past tense forms, they should find it harder to inflect verbs when an illegal cluster would be created. On the other hand, if typically developing children are able to compute regular past tense verb forms online, then cluster frequency should have no effect, and they should be equally able to infect the stem whatever the legality of the final cluster.

Reanalysis of the past tense elicitation data collected by van der Lely and Ullman (2001), a new elicitation experiment conducted with a new group of G-SLI children, plus re-analysis of data previously collected by Michael Thomas et al. (2001) confirmed our predictions. Whereas typically developing children from all three studies showed no differences between regular past tense forms containing legal or illegal clusters, the G-SLI children performed consistently worse on verbs containing illegal clusters (Marshall & van der Lely, 2006).

A further investigation of this phenomenon studied past participle forms in online processing of passive sentences, which contained a past participle with either a monomorphemically legal cluster (*kissed*; see 4a) or a monomorphemically illegal cluster (*bathed*; see 4b).

(4) a) I think that the squirrel with the gloves was ki<u>ssed</u> by the tortoise at his house last weekend.

b) I think that the squirrel with the gloves was ba<u>thed</u> by the tortoise at his house last weekend.

We predicted that typically developing children would be able to use the phonotactic cues provided by an illegal cluster to identify the past participle, and therefore interpret the passive sentence more accurately than when this cue was not available; that is, in the forms with legal clusters. G-SLI children, however, were predicted not to be able to use this parsing cue, and therefore show no advantage for sentences where the past participle contained an illegal cluster. Using a self-passed listening task involving a sentence-picture judgement task, this is exactly what we found: typically developing children were significantly more accurate on their judgements for sentences containing the monomorphemically illegal past participle forms. In contrast, G-SLI children did not show a difference between legal and illegal forms, suggesting that the phonotactic cue to the words morphological complexity did not facilitate their parsing of the sentence.

Our investigations into the phonotactics of clusters with respect to morphology is at the interface of morphology and phonology. We now turn to the phonology component itself and our findings from G-SLI children.

PHONOLOGY

Just as the deficit in G-SLI affects hierarchical structures in syntax and morphology, so it affects hierarchical structures in phonology. Phonological constituents such as syllables and prosodic words are grouped into successively higher levels of the prosodic hierarchy (Selkirk, 1978). Certain aspects of phonological structure cause difficulty for children with G-SLI. Children with G-SLI have clear and fluent speech, and are intelligible for known words. Their phonological deficit manifests as a difficulty with forms that are complex at the syllable and foot levels of the prosodic hierarchy (Gallon et al., 2007). In a nonword repetition task, G-SLI children simplify consonant clusters in all word positions, while unfooted syllables are deleted or cause syllabic simplifications and segmental changes elsewhere in the word (Marshall, 2004; Marshall, Ebbels, Harris, & van der Lely, 2002).

For example, in Gallon et al.'s study, *fə. klɛs. tə. lə* (where dots indicate syllable boundaries) was repeated by some children as, *fə. kɛs. tə. lə* with cluster simplification, and by others as *fə. glɛs. tə.*, with erroneous voicing of the velar stop and deletion of the final unfooted syllable (Gallon et al., 2007). Furthermore, systematically increasing the complexity of phonological structure resulted in a systematic increase in errors, regardless of the number of syllables, indicating that it is not just the length of phonological material to be retained in phonological short-term memory that is relevant to repetition accuracy, but the arrangement of that material in the prosodic hierarchy. Even monosyllabic nonwords with two clusters (e.g. *klɛst*) were more difficult for G-SLI children than those with one cluster (e.g., *klɛt*) or no clusters (e.g., *kɛt*), and for disyllabic nonwords, a marked initial weak syllable caused weak-strong forms (e.g., *bə.dremp*) to be more difficult than strong-weak forms (e.g., *drɛm.pə*). This contrasts with previous studies of nonword repetition in children with SLI, which have not shown group differences between SLI and typically developing children when nonwords are only one or two syllables long.

However, it is how these deficits in phonology and other components of language impact on language processing and production that is of ultimate concern to both researcher and clinician.

THE CUMULATIVE EFFECT OF COMPONENT DEFICITS

The CGC model hypothesizes that for children with G-SLI, the deficit is in representing linguistic structural complexity in three components of the computational grammatical system—syntax, morphology, and phonology. Deficits in these components impact on a variety of linguistic constructions as a function of their syntactic, morphological, and phonological complexity. The regular past tense deficit that is found not only in children with G-SLI, but also the vast majority of children with SLI, has been explored most thoroughly. We have already discussed the impact of syntactic and morphological deficits on G-SLI children's realization of tense. Here we complete the picture by considering the effects of phonological complexity. Using an elicitation task we manipulated the phonological complexity of the inflected verb end, and found that, as predicted, phonological complexity impacted on suffixation. G-SLI children were less likely to inflect stems when the suffixed form would end in a consonant cluster, for example *jumped* and *hugged*, compared to when no cluster would result, for example, *weighed.* Furthermore, stems ending in a cluster (*jump*) were less likely to be suffixed than stems ending in a single consonant (*hug*). In contrast, typically developing controls showed no effect of phonological complexity on inflection (Marshall & van der Lely, 2007b).

In another study, we found that morphological and phonological deficits impact even on an aspect of language that is not traditionally noted as being problematic for English-speaking children with SLI—derivational morphology (Marshall & van der Lely, 2007a). We elicited two types of derived forms—adjectives derived from nouns by the addition of *-y* (*sand* → *sandy*, *rocks* → *rocky*), and comparative and superlative adjectives (*happy* → *happier, happiest*). In the former case, the stimulus was either a singular or a regular plural noun, while in the latter the adjectival stem was either one or two syllables long. G-SLI children almost invariably supplied the *-y*, *-er,* and *-est* suffixes, in stark contrast to their high omission of the past tense suffix. Moreover, increasing the morphological or phonological complexity of the stimulus did not trigger suffix omission, but did result in nontarget forms that were uncharacteristic of typically developing children. Some G-SLI children included *-s* inside *-y* when presented with a plural stimulus, producing forms such as *holesy, rocksy,* and *frillsy,* whereas typically developing children very rarely did so (*holey, rocky, frilly*). In forming comparative and superlative adjectives, both G-SLI and typically developing children reduced three-syllable outputs (e.g., *happier, narrowest*) to two-syllable outputs, providing evidence of a maximal word effect on derivation; that is, pressure to limit the output to the size and shape of a trochaic (strong-weak) foot, a constraint that is characteristic of

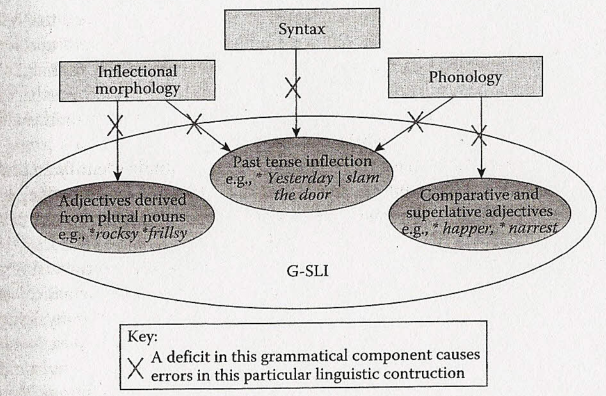

FIGURE 20.2 The computational grammatical complexity model of G-SLI: The impact of component deficits on different linguistic constructions.

young English-speaking children. However, the groups responded differently—G-SLI children's favored strategy was to truncate the stem and retain the suffix (*happer, narrest*), whereas for typically developing children the favored strategy was to omit the suffix and retain the stem-final weak syllable (*happy, narrow*).

The cumulative impact of independent component deficits in the grammatical computational system is illustrated in Figure 20.2.

DO AUDITORY DEFICITS MAINTAIN G-SLI?

One possibility is that an auditory deficit causes and maintains the grammatical impairments found in G-SLI. Evidence of subtle auditory deficits would conflict with domain-specific hypotheses and, instead, support domain-general perspectives. However, neither behavioral nor electrophysiological data have revealed any consistent auditory deficit in individuals with G-SLI that is independent of their language deficit. First, we explored G-SLI children's auditory perception for speech and nonspeech sounds, at varying presentation rates, and controlling for the effects of age and language on performance (van der Lely et al., 2004). For nonspeech formant transitions, 69% of the G-SLI children showed normal auditory processing, whereas for the same acoustic information in speech, only 31% did so. For rapidly presented tones, 46% of the G-SLI children performed normally. Auditory performance with speech and nonspeech sounds differentiated the G-SLI children from their age-matched controls, whereas speed of processing did not. A further set of experiments looked at "backward masking"; that is, their ability to detect a brief tone in quiet, and in the presence of a following noise (Rosen, Adlard, & van der Lely, 2009). Here group analyses showed that mean thresholds for the G-SLI group were never worse than those obtained for the two younger language control groups, but were higher in both backward and simultaneous masking compared to age-matched controls. However, more than half of the G-SLI group (8/14) were within normal limits for all thresholds. Furthermore, the G-SLI children consistently evinced no relationship between their auditory and phonological/grammatical abilities.

A further possibility is that a more sensitive measure might identify an impairment. To test this we explored the neural correlates to auditory processing using event related potential techniques. ERPs can measure brain responses with a millisecond precision, and therefore have the time resolution to detect delayed or deviant brain responses. We recorded Auditory Evoked Potentials (AEPs) of G-SLI and age matched control participants to pure tones in a classical auditory oddball paradigm. Auditory processing elicits early electrophysiological responses known as the N100/P200 (or N1/P2) complex. This complex is associated with perceptual detection of a discrete change in the auditory environment. In addition, they elicit a later P300 component that reflects attentional control processes to detect and categorize a specific event. We discovered that children with G-SLI have age-appropriate waveforms for the N100, P200, and the P300 components (latency, amplitude, distribution on the scalp; Fonteneau & van der Lely, 2008). Our results reveal that G-SLI children have normal auditory processing during the discrimination of pure tones.

These findings, along with those investigating nonverbal cognitive abilities thought to possibly co-occur with, but not cause SLI (or G-SLI; see van der Lely et al., 1998), are consistent with a number of other researchers in the field investigating other SLI subgroups (Bishop et al., 2000; Bishop, Carlyon, Deeks, & Bishop, 1999) and dyslexia (Ramus, 2003). Auditory and nonverbal deficits are more prevalent in the SLI population than in typically developing populations, but no consistent nonverbal deficit has yet been found to co-occur or cause any form of SLI. This of course does not mean that such deficits, when they do co-occur, do not have an impact on language development: it is likely that they do to some extent. We set out in the discussion of our model just why and how we see different components of language affecting performance.

THE CGC MODEL OF LANGUAGE IMPAIRMENT IN THE CONTEXT OF A COMPONENT MODEL OF LANGUAGE ACQUISITION

Our discussion above provides details of the nature of component deficits within the CGC model, and specifically the nature of the hierarchical deficits within each grammatical component. This model has led to clear predictions with respect to precisely those structures in syntax, morphology, and phonology that will be impaired. In addition, the model has enabled us to predict how component deficits would individually impact on processing of linguistic forms, such as wh-questions, and tense marking. The model provides a parsimonious explanation

for the pattern of impairments in G-SLI: cumulative effects of component deficits, alongside compensation by components that potentially function normally. Although the CGC model was originally developed to account for G-SLI, the component nature of this model and the detail within the grammatical components enables it to be applied to other forms of SLI, in both English and other languages, and indeed to typical language acquisition. For example, the model predicts that an auditory speech deficit will impact on phonology (specifically at the segmental level) due to mapping relations between the two, and thereby impact on language. It will not, however, directly *cause* the basic mechanisms and representations of phonology to be impaired. The CGC differs from domain-general perspectives in this respect, and this is, of course, an empirical issue. However, the specification of the CGC goes some way to helping understand *how* different components can affect language processing and performance. There is still much to understand about the role of mapping relations between components in normal and impaired acquisition, but we hope that this model will contribute to clarifying these relations.

CONCLUSION

This chapter has focused on the G-SLI subgroup. It has shown the existence of a relatively pure grammatical impairment that affects syntax, morphology, and phonology, but spares other cognitive abilities. More specifically, the deficit affects particular aspects of the grammatical system: complex hierarchical structure. This deficit in the CGC system manifests itself as an impairment in the computation of syntactic dependencies at the level of clause structure, complex morphological words, involving abstract rules, and complex phonological forms involving, for example, clusters and unfooted syllables. All these aspects of language are those learned through development and vary in interesting ways from language to language. Our data challenge views denying that some forms of language impairment are caused by underlying deficits to highly specialized domain-specific mechanisms that normally develop in the young child to facilitate language learning. The fact that other subgroups of SLI show deficits that have similar characteristics in syntax, morphology, and phonology, regardless of any co-occurring problems (Norbury et al., 2002), points to multiple genetic causes impacting on these components, with some genetic causes being more discrete than others.

Finally, the CGC model explains how processing in many different components of language—some within and some outside the grammatical system—in addition to factors pertinent to language such as phonological memory, contributes to language performance. In other words, processing is a reflection of "multiple processing systems." Our model clarifies how each component impacts on language performance in highly predictable ways. Thus, language performance will depend on both the linguistic characteristics of the material with respect to its complexity in each component, and the basic functioning of each component itself. We have shown how G-SLI children appear to use their strengths in certain components to compensate, at least partially, for deficits in others. Thus, for G-SLI children and perhaps other SLI subgroups too, a relative strength in semantic processing could be targeted to help compensate for their syntactic impairment.

In conclusion, our data from the G-SLI subgroup show how some components in grammar can be selectively impaired, supporting a domain-specific view of at least some cognitive systems. These findings provide a valuable window onto the functional architecture of the brain and the development of uniquely human and specialized higher cognitive systems.

ACKNOWLEDGMENTS

We thank the children, parents, schools, and speech and language therapists who have contributed so much to the work reported here. The work was supported by the Wellcome Trust Grant number 063713, the ESRC Grant number RES-000-23-0575, and a Leverhulme Trust Visiting Fellowship to H. van der Lely. Particular thanks are due to the insightful comments and discussion from participants of the Biocomp Workshop on "Specific language impairment and the language faculty" at Harvard University, February 20, 2008 where many of the above issues were discussed.

REFERENCES

Archonti, A. (2003). Wh-Question formation in typically developing children and children with Grammatical SLI. *Human communication science.* London, UK: UCL.

Bishop, D. V. M. (1982). Comprehension of spoken, written and signed sentences in childhood language disorders. *Journal of Child Psychology and Psychiatry, 23*, 1–20.

Bishop, D. V. M. (1997). *Uncommon understanding: Comprehension in specific language impairment*. Hove, UK: Psychology Press.

Bishop, D. V. M., Adams, C. V., & Norbury, C. F. (2006). Distinct genetic influences on grammar and phonological short-term memory deficits: Evidence from 6-year-old twins. *Genes Brain Behavior, 5*(2), 158–169.

Bishop, D. V. M., & Bishop, S. J. (1998). "Twin language": A risk factor for language impairment? *Journal of Speech, Language, and Hearing Research, 41*(1), 150–160.

Bishop, D. V. M., Bright, P., James, C., Bishop, S. J., & van der Lely, H. K. J. (2000). Grammatical SLI: A distinct subtype of developmental language disorder?" *Applied Psycholinguistics, 21*, 159–181.

Bishop, D. V. M., Carlyon, R. P., Deeks, J. M., & Bishop, S. J. (1999). Auditory temporal processing impairment: Neither necessary nor sufficient for causing language impairment in children. *Journal of Speech, Language, and Hearing Research, 42*(6), 1295–1310.

Bishop, D. V. M., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorder and specific language impairment: A study using standardised diagnostic instruments. *Journal of Child Psychology & Psychiatry, 43*(7), 917–929.

Bishop, D. V. M., & Snowling, M. J. (2004). Developmental dyslexia and specific language impairment: Same or different?" *Psychological Bulletin, 130*(6), 858–886.

Chomsky, N. (1998). *Minimalist inquiries: The framework Ms*. Cambridge, MA: MIT Press.

Chomsky, N. (2004). Beyond explanatory adequacy. In A. Belletti (Ed.), *Structures and beyond: The cartography of syntactic structures* (Vol. 3, pp. 104–131). Oxford, UK: Oxford University Press.

de Bree, E. (2007). *Dyslexia and phonology: A study of the phonological abilities of Dutch children at-risk of dyslexia*. University of Utrecht, The Netherlands: LOT.

Fisher, S. E., & DeFries, J. C. (2002). Developmental dyslexia: Genetic dissection of a complex cognitive trait. *Nature Reviews Neuroscience, 3*, 767–780.

Fisher, S. E., Lai, C. S., & Monaco, A. P. (2003). Deciphering the genetic basis of speech and language disorders. *Annual Review Neuroscience, 8*, 8.

Fonteneau, E., & van der Lely, H. K. J. (2008). Electrical brain responses in language-impaired children reveal grammar-specific deficits. *PLoS ONE, 3*(3), e1832.

Friederici, A. D. (2002). Toward a neural basis of auditory sentence processing. *Trends in Cognitive Sciences, 6*(2), 78–84.

Friedmann, N., & Gvion, A. (2002). Modularity in developmental disorders: Evidence from SLI and peripheral dyslexias. *Behavioral and Brain Sciences, 25*(6), 756–757.

Friedmann, N., & Novogrodsky, R. (2004). The acquisition of relative clause comprehension in Hebrew: A study of SLI and normal development. *Journal of Child Language, 31*(3), 661–681.

Friedmann, N., & Novogrodsky, R. (2007). Is the movement deficit in syntactic SLI related to traces or to thematic role transfer?" *Brain & Language, 101*(1), 50–63.

Gallon, N., Harris, J., & van der Lely, H. K. J. (2007). Non-word repetition: An investigation of phonological complexity in children with Grammatical SLI. *Clinical Linguistics & Phonetics, 21*(6), 435–455.

Gathercole, S. (2006). Nonword repetition and word learning: The nature of the relationship. *Applied Psycholinguistics, 27*, 513–543.

Gordon, P. (1985). Level-ordering in lexical development. *Cognition, 21*, 73–93.

Hauser, M. D. (2001). What"s so special about speech? In E. Dupoux (Ed.), *Language, brain and cognitive development: Essays in honor of Jacques Mehler* (pp. 417–433). Cambridge, MA: MIT Press.

Hauser, M. D., Chomsky, N., & Fitch, W. T. (2002). The faculty of language: What is it, who has it, and how did it evolve? *Science, 298*(5598), 1569–1579.

Jakubowicz, C., Nash, L., & van der Velde, M. (1999). *Inflection and past tense morphology in French SLI*. Somerville, MA: BUCLD, Cascadilla Press.

Jakubowicz, C., & Strik, N. (2008). Scope-marking strategies in the acquisition of long-distance wh-questions in French and Dutch. *Language and Speech: On Phonological, Lexical and Syntactic Components of Language Development, 51*, 101–132.

Joanisse, M. (2004). Specific language impairments in children: Phonology, semantics and the English past tense. *Current Directions in Psychological Science, 13*, 156–160.

Joanisse, M. (2007). Phonological deficits and developmental language impairments. In D. Mareschal, S. Sirois, & G. Westermann (Eds.), *Neuroconstructivism, Vol. 2: Perspectives and Prospects*. Oxford, UK: Oxford University Press.

Joanisse, M., & Seidenberg, M. (1998). Specific language impairment: A deficit in grammar or processing? *Trends in Cognitive Sciences, 2*, 240–247.

Leonard, L. (1998). *Children with specific language impairment.* Cambridge, MA: MIT Press.

Leonard, L. B., Dromi, E., Adam, G., & Zadunaisky-Ehrlich, S. (2000). Tense and finiteness in the speech of children with specific language impairment acquiring Hebrew. *International Journal of Language and Communication Disorders, 35*(3), 319–335.

Marcus, G. (2004). *The birth of the mind: How a tiny number of genes creates the complexities of human thought.* New York, NY: Basic Books.

Marcus, G. F., Pinker, S., Ullman, M., Hollander, M., Rosen, T. J., & Xu, F. (1992). *Overregularization in language acquisition. Monographs of the Society for Research in Child Development Series 228,* Chicago, IL: University of Chicago Press.
Marinis, T., & van der Lely, H. K. J. (2007). On-line processing of questions in children with G-SLI and typically developing children. *International Journal of Language and Communication Disorders, 42,* 557–582.
Marshall, C. (2004). *The morpho-phonological interface in specific language impairment.* PhD Thesis; DLDCN Centre. London: University College.
Marshall, C. R., Ebbels, S., Harris, J., & van der Lely, H. (2002). Investigating the impact of prosodic complexity on the speech of children with Specific Language Impairment. In R. Vermeulen & A. Neeleman (Eds.), UCL Working Papers in Linguistics (Vol. 14, pp. 43–68).
Marshall, C. R., Harcourt-Brown, S., Ramus, F., & van der Lely, H. K. J. (2009). The link between prosody and language skills in children with SLI and/or dyslexia. *International Journal of Language & Communication Disorders, 44,* 466–488.
Marshall, C. R., & van der Lely, H. K. J. (2006). A challenge to current models of past tense inflection: The impact of phonotactics. *Cognition, 100*, 302–320.
Marshall, C. R., & van der Lely, H. K. J. (2007a). Derivational morphology in children with grammatical-specific language impairment. *Clinical Linguistics & Phonetics, 21*(2), 71–91.
Marshall, C. R., & van der Lely, H. K. J. (2007b). The impact of phonological complexity on past tense inflection in children with Grammatical-SLI. *Advances in Speech Language Pathology, 9*, 191–203.
Marshall, C. R., & van der Lely, H. K. (2009). Effects of word position and stress on onset cluster production: Evidence from typical development, SLI and dyslexia. *Language, 85*, 39–57.
Messaoud-Galusi, S., & Marshall, C. R. (2010). Introduction to this special issue. Exploring the overlap between dyslexia and SLI: the role of phonology. *Scientific Studies of Reading*, 14, 1–7.
Montgomery, J. W. (2000). Verbal working memory and sentence comprehension in children with specific language impairment. *Journal of Speech Language Hearing Research, 43*(2), 293–308.
Norbury, C. F., Bishop, D. V. M., & Briscoe, J. (2002). Does impaired grammatical comprehension provide evidence for an innate grammar module? *Applied Psycholinguistics, 23*, 247–268.
O'Hara, M., & Johnston, J. (1997). Syntactic bootstrapping in children with specific language impairment. *European Journal of Disorders of Communication, 2*, 189–205.
Pinker, S. (1999). *Words and rules: The ingredients of language.* London, UK: Weidenfeld & Nicolson.
Pinker, S., & Jackendoff, R. (2005). The faculty of language: What's special about it? *Cognition*, *95*(2), 201–236.
Ramus, F. (2003). Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? *Current Opinion in Neurobiology, 13*(2), 212–218.
Ramus, F., & Szenkovits, G. (2008). What phonological deficit? *Quarterly Journal of Experimental Psychology, 61*(1), 129–141.
Rice, M. (2003). A unified model of specific and general language delay: Grammatical tense as a clinical marker of unexpected variation. In Y. Levy & J. Schaeffer (Eds.), *Language competence across populations: Toward a definition of Specific Language Impairment* (pp. 63–95). Mahwah, NJ: Lawrence Erlbaum.

Rice, M. (2004). Language growth of children with SLI and unaffected children: Timing mechanisms and linguistic distinctions. In A. Brugos, L. Micciulla, & C. Smith (Eds.), *Proceeding of the 28th Annual Boston University Conference on Language Development* (Vol. 1, pp. 28–49). Somerville, MA: Cascadilla Press.
Rice, M., Wexler, K., & Redmond, S. M. (1999). Grammaticality judgments of an extended optional infinitive grammar: Evidence from English-speaking children with specific language impairment. *Journal of Speech, Language, and Hearing Research, 42*, 943–961.
Rosen, S., Adlard, A., & van der Lely, H. K. J. (2009). Backward and simultaneous masking in children with grammatical specific language impairment: No simple link between auditory and language abilities. *Journal of Speech, Language, and Hearing Research, 52,* 396–411.
Selkirk, E. O. (1978). On prosodic structure and its relation to syntactic structure. In T. Fretheim (Ed.), *Nordic prosody* (Vol. 2, pp. 111–140). Trondheim, The Netherlands: TAPIR.
Snowling, M. (2000). *Dyslexia*, Second Edition. Oxford, UK: Blackwell.
Stavrakaki, S. (2001). Comprehension of reversible relative clauses in specifically language impaired and normally developing Greek children. *Brain and Language, 77*, 419–431.
Stavrakaki, S., & van der Lely, H. (2010). Production and comprehension of pronouns by Greek children with specific language impairment. *British Journal of Developmental Psychology*, *28*, 189–216.
Stromswold, K. (1998). Genetics of spoken language disorders. *Human Biology, 70*(2), 297–324.
Tallal, P. (2002). Experimental studies of language learning impairments: From research to remediation. In D. Bishop & L. Leonard (Eds.), *Speech and language impairments in children* (pp. 131–156). East Sussex, UK: Psychological Press..
Tallal, P., Miller, S. L., Bedi, G., Byma, G., Wang, X., Nagarajan, S. S., . . . Merzenich, M. M. (1996). Language comprehension in language-learning impaired children improved with acoustically modified speech. *Science, 271*, 81–83.
Thomas, M. S. C., Grant, J., Barham, Z., Gsödl, M., Laing, E., Lakusta, L., . . . Karmiloff-Smith, A.(2001). Past tense formation in Williams Syndrome. *Language and Cognitive Processes, 16*, 143–176.
Thornton, R. (1995). Referentiality and wh-movement in child English: Juvenile D-Linkuency *Language Acquisition, 1–2*(4), 139–175.
Tomblin, J. B., Records, N. L., Buckwalter, P., Zhang, X., Smith, E., & O'Brien, M. (1997). Prevalence of specific language impairment in kindergarten children. *Journal of Speech Language Hearing Research, 40*(6), 1245–1260.
van der Lely, H. K. J. (1994). Canonical linking rules: Forward versus reverse linking in normally developing and specifically language-impaired children. *Cognition, 51*(1), 29–72.
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.
van der Lely, H. K. J. (2005). Domain-specific cognitive systems: Insight from grammatical-specific language impairment. *Trends in Cognitive Sciences, 9*, 53–59.
van der Lely, H. K. J., & Battell, J. (2003). Wh-movement in children with grammatical SLI: A test of RDDR Hypothesis. *Language, 79*, 153–181.
van der Lely, H. K. J., & Christian, V. (2000). Lexical word formation in children with grammatical SLI: A grammar-specific versus an input-processing deficit? *Cognition, 75*(1), 33–63.

van der Lely, H. K. J., Jones, M., & Marshall, C. R. (in press). * Who did Buzz see someone? Grammaticality judgement of wh-questions in typically developing and children and children with Grammatical-SLI. Lingua.

van der Lely, H. K. J., Rosen, S., & Adlard, A. (2004). Grammatical language impairment and the specificity of cognitive domains: Relations between auditory and language abilities. *Cognition, 94*(2), 167–183.

van der Lely, H. K. J., Rosen, S., & McClelland, A. (1998). Evidence for a grammar-specific deficit in children. *Current Biology, 8*(23), 1253–1258.

van der Lely, H. K. J., & Stollwerk, L. (1997). Binding theory and grammatical specific language impairment in children. *Cognition, 62*, 245–290.

van der Lely, H. K. J., & Ullman, M. (1996). *The computation and representation of past-tense morphology in normally developing and specifically language impaired children.* The 20th Annual Boston University Conference on Language Development. Vol. 2, Somerville, MA: Cascadilla Press.

van der Lely, H. K. J., & Ullman, M. (2001). Past tense morphology in specifically language impaired children and normally developing children. *Language and Cognitive Processes, 16*, 113–336.

Vernes, S. C., Newbury, D. F., Abrahams, B. S., Winchester, L., Nicod, J., Groszer, M., et al. (2008). A functional genetic link between distinct developmental language disorders. *New England Journal of Medcine*, 359(22), 2337–2345.

Watkins, K. E., Dronkers, N. F., & Vargha-Khadem, F. (2002). Behavioural analysis of an inherited speech and language disorder: Comparison with acquired aphasia. *Brain, 125*(Pt 3), 452–464

21 The Developing Mental Lexicon of Children With Specific Language Impairment

Holly Storkel

INTRODUCTION

The mental lexicon refers to "the collection of words stored in the human mind" (Trask, 1997, p. 140) with each entry "detailing the properties of a single lexical item: its pronunciation, its meaning, its word class, its subcategorization behavior, any grammatical irregularities, and possibly other information" (Trask, 1997, p. 130). This chapter will focus on a subset of the properties of lexical items that are frequently incorporated in adult and child models of spoken language processing, namely phonological, lexical, and semantic representations (Dell, 1988; Gupta & MacWhinney, 1997; Levelt, 1989; Luce, Goldinger, Auer, & Vitevitch, 2000; Magnuson, Tanenhaus, Aslin, & Dahan, 2003; McClelland & Elman, 1986; Norris, 1994). The phonological representation includes information about individual sounds, with models varying in the specific information incorporated (e.g., phonetic features, context-specific allophones, phonemes). For simplicity of illustration, phoneme units will be used to illustrate the phonological representation in this chapter. Thus, the phonological representation of the word "cat" would consist of the individual phonemes /k/, /æ/, and /t/ (i.e., three separate units). The lexical representation includes information about the sound structure of the word as an integrated unit. Continuing the illustration, the lexical representation for "cat" would be /kæt/ (i.e., one unit). Lastly, the semantic representation consists of information about the meaning or referent of the word. Here, the semantic representation for "cat" would include, but not be limited to, information such as "four-legged furry pet that purrs."

For the developing mental lexicon, there are two processes of critical importance. The first process involves the actual creation of the mental lexicon. That is, children are not born knowing the words of their language. Instead, words must be learned through exposure to the language during every day interactions. The second process involves accessing the words in the mental lexicon for language production or comprehension. This is the process that allows children to use the words that they know to communicate. It is critical to understand the potential