

*Children with Specific Language  
Impairment: Bridging the Genetic and  
Developmental Perspectives*

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It is widely understood that children's language acquisition is a remarkably robust phenomenon. A valuable scientific generalization is that youngsters share a nearly universal aptitude for the acquisition of their native language during the first few years of life. As documented in this volume, there is now a large literature that provides descriptive benchmarks, pegged to chronological age, that describe children's language development across different linguistic dimensions and social contexts of use; the ways in which children may or may not draw upon one dimension, such as semantic development, to build another, such as syntax; and the ways in which children's social contexts contribute to their development.

Some years ago, Steven Pinker succinctly captured a predominant view of the robustness of children's language acquisition abilities: "In general, language acquisition is a stubbornly robust process; from what we can tell there is virtually no way to prevent it from happening short of raising a child in a barrel" (Pinker, 1984, p. 29). Opposition to his perspective came from scholars who argued for more influence attributable to variations in environmental exposure that are less extreme than total deprivation (cf. Snow, 1996). From either perspective, a corollary was implied: If a child did not develop language readily, there must have been something significantly deficit about the child's environment (barring no obvious concomitant conditions such as severe mental retardation or hearing loss that would impair a youngster's abilities to acquire language).

Children with specific language impairment (SLI) are significant exceptions to this assumption. Children with SLI do not keep up with their age peers in their language development, although there is no obvious reason for them to fall behind. To understand these youngsters, the perspective shifts from central tendencies across all children to individual differences characteristic of a subgroup of children; from an assumption of

tightly intertwined dimensions of language to consideration of possible disruptions in synchrony; and from an emphasis on shared or nonshared social environments to possible inherited limitations in language acquisition mechanisms.

These variations from the normative developmental assumptions are evident in the clinical, developmental, and genetic perspectives of SLI, each of which is summarized in this chapter. It is suggested that a bridge between the genetic and developmental perspectives is needed and the notion of maturational mechanisms could serve such a bridging function. There is a long-standing interest in the possible causes of SLI. The momentum of modern genetics inquiry is profoundly shaping the fundamental work of description of the symptoms of language impairment in SLI and interpretive frameworks. In many ways the new genetics investigations sharpen the need for solid empirical work across the wide range of language phenomena. This includes a need for developmental studies that capture the growth trajectories of language with careful attention to central tendencies within age levels and individual variations, and interpretive frameworks that aim to integrate growth mechanisms with etiological factors.

The chapter begins with conventional operational definitions of SLI, followed by a brief overview of the history of investigations of this condition. Then the clinical, genetic, and developmental perspectives are briefly explained, and why they are in need of bridging. This is followed by a review of the research that aims to describe the phenotype (behavioral symptoms thought to be related to genetic influences), with an emphasis on growth trajectories. The next section reviews the recent genetics advances. The following section lays out some basic parameters of a maturational perspective of inherited timing mechanisms and developmental change. The chapter concludes with a section on the implications of the SLI research for general models of language development.

## **Definition of Specific Language Impairment**

The commonly accepted research definition of SLI invokes inclusionary and exclusionary criteria. Inclusionary criteria are intended to establish that the affected youngsters have language impairments relative to age expectations. Typically, this is determined by performance on a standardized omnibus language assessment instrument, with “impairment” defined as performance of  $-1$  standard deviation or more below the age mean, or, roughly, at the 15th percentile or below of the child’s age group. Depending upon the purpose of a given investigation, the definition is sometimes further specified according to receptive versus expressive language abilities, and according to whether or not the children have accompanying speech impairments. For example, in studies of morphological impairments it is important to control for phonological/speech impairments, in order to avoid confounding estimates of morphological impairments with phonological or speech impairments. This is typically done by means of a pretest designed to elicit word final consonants in single words (cf. Rice & Wexler, 2001). Another example is the need to control for speech impairments in imitation tasks; without such control, it is not possible to tell if poor performance is attributable to poor speech skill or poor memory of speech/phonological units.

The exclusionary criteria are intended to select affected children whose developmental impairment is specific to language (see Rice & Warren, 2004, and Rice, Warren, & Betz, 2005, for information about language disorders across different clinical groups). Children with hearing loss are excluded. Children diagnosed with syndromic conditions, such as Williams syndrome, Down syndrome, and fragile X syndrome, are excluded. Conventionally, children diagnosed as autistic have been excluded, although in the recent shift to the broader clinical category of autism spectrum disorders (ASD) the diagnostic boundary between SLI and autism is somewhat blurred and is currently a matter of active investigation (see Tager-Flusberg, 2004, 2005, for overviews). The issue is the extent to which children with language impairments show overlap, either in genetic risk indicators (i.e., family aggregation) or in behavioral symptoms. Mental retardation is usually ruled out via exclusion of children whose nonverbal IQ performance levels are 85 or below. The range between 70 and 85 nonverbal IQ is sometimes invoked as acceptable for the label of SLI, although it is preferable to label this range as “nonspecific language impairment” (NLI) (Tomblin & Zhang, 1999) and treat children in this range as a separate clinical group, in order to avoid error attributable to too much heterogeneity (and unknown sources of error variance) in the grouping criteria (e.g., Miller, Kail, Leonard, & Tomblin, 2001; Rice, Tomblin, Hoffman, Richman, & Marquis, 2004). Finally, children with known neurological conditions, such as epilepsy, are routinely excluded from the SLI research groups. In actual clinical practice the generic label of “language impairment” is sometimes used to collapse across the exclusionary criteria to create an inclusive clinical group that is more diffuse than the controlled research groupings.

There is long-standing recognition of the need to avoid confusing dialectal differences in linguistic systems with linguistic differences indicative of language impairment. Conventional standardized language assessments have been widely criticized as inappropriate for dialect speakers. With support from the National Institute of Deafness and Communication Disorders, investigators have developed new assessment systems designed to differentiate dialectal differences from language disorders in young children (Seymour, Roeper, & de Villiers, 2003), or have adapted existing measures and methods to use as culturally valid language screenings (Oetting & McDonald, 2002; Washington & Craig, 2004). Research standards are moving toward explicit control of dialectal differences in studies of children with SLI, that is, to treat dialect as an exclusionary variable for studies of the linguistic dimensions affected by the dialect or to use dialect as a grouping variable.

A recent epidemiological investigation generated an estimate of 7% of children (8% for boys and 6% for girls) aged 5 to 6 years as meeting the definition of SLI (Tomblin et al., 1997). This investigation also established that in the general population clinically significant speech impairments are orthogonal to language impairments (Shriberg, Tomblin, & McSweeney, 1999). This outcome was surprising, because children with speech impairments are more likely to be identified for clinical services. Thus, children with speech and language problems are over-represented in clinical caseloads, creating the impression of considerable overlap. It is worth noting that because children with SLI who participate in research studies are often recruited out of clinical caseloads, children who participate in scientific studies may be likely to have speech as well as language impairments, although it is often difficult to establish this because speech status is not

always described. These sampling issues are important to keep in mind because the clinical samples may not be representative of the general population of children with language impairments; speech and language impairments are not intrinsically linked in the condition of language impairment; and the co-occurrence of speech and language impairments can introduce confounds in some tasks.

Another important outcome of the epidemiological study was that affected children were unlikely to be identified and enrolled in clinical services. Tomblin et al. (1997) report that the parents of 29% of the affected children reported that they had previously been informed that their child had a speech or language problem. Thus, our best current information suggests that about 70% of affected children are not identified and enrolled in appropriate intervention during their kindergarten year of school.

Collectively, these observations point toward a clinical condition of unexpected variation in children's language acquisition aptitude that is: (1) more prevalent in the general population than widely assumed; (2) likely to be confused with speech impairments and/or possible concomitant developmental deficits such as low cognitive ability, social deficits, or reading impairment; and (3) often undiagnosed. These facts certainly contribute to the challenges involved in careful investigation of this clinical condition, and perhaps to the inconsistencies in the scientific literature insofar as the difficulties conspire to increase the likelihood of heterogeneity in the affected groups and an imprecise calibration across studies of children labeled as SLI. This becomes especially relevant to etiological investigations, as discussed below. At the same time, there are areas of convergent and replicated findings that contribute to a strong sense of momentum.

## **History of Studies of SLI**

The study of SLI has roots at least as far back as the beginnings of modern descriptive medicine and psychology, to the early years of the 1800s. A case description by Gall in 1822 is widely cited as the earliest description in the modern literature (Gall, 1835, English translation). Subsequent entries in the literature generated a variety of labels for the condition, ranging from "congenital word deafness" to the widespread extensions of the term "aphasia" in the 1900s. "Developmental aphasia" (Eisenson, 1968) and "developmental dysphasia" (Clahsen, 1989) are immediate antecedents of the current term. By the 1980s, researchers began to adopt "specific language impairment" as an etiologically neutral term, without assumption of neurological impairments of the same sort as associated with aphasia or cortical lesions (e.g., Leonard, 1981). In the current literature, "specific language impairment" is in widespread use by investigators, although a variety of diagnostic labels appear in clinical manuals and across different clinical settings (cf. Leonard, 1998, pp. 5–8).

In the context of this chapter, two points are of note. One is that the unexpected variation in children's language acquisition has been documented for a significant length of time. The other is that, historically, the labels vary as a consequence of the scholarly context of the observer. The condition of SLI falls at the margin of the focal points of studies of child development, child health/disease, and children with disabilities. The

disciplines of psychology, medicine, education, and, more recently, speech/language pathology, linguistics, and genetics have contributed to the literature that bears on SLI. It remains to be seen if the label stays constant or if new labels appear for new distinctions. What is essential is the need to explicate the core phenomenon, that is, how it is that children who seem to have all the prerequisite abilities and environmental resources in place nevertheless do not develop language in the expected robust fashion.

### **Clinical, Genetic, and Developmental Perspectives: A Need for Bridging**

From the outset there has been a great interest in the factors that cause SLI. This is apparent in early studies of the genetics of language, dating from the beginning of the 1900s (e.g., Orton, 1930). Much of the early period of investigation established that individuals with speech and language impairments tended to cluster in families. Lenneberg (1967) foresaw the current program of investigation in his description of “congenital language disability” involving “delayed onset of speech, protracted articulatory difficulties in childhood, congenital expressive disorders” (p. 349).

With the advent of the modern explosion in behavioral and molecular genetics, the search for a genetic contribution to individual variation in language aptitude has taken on new momentum. Studies of the genetic contributions to SLI are moving on multiple fronts, with the most striking advances in DNA analyses of affected cases and family members (for an overview of current methods and research directions, see Rice & Smolik, *in press*, Smith, 2004, and Smith & Morris, 2005). Twin studies are also contributing valuable new information. There is growing consensus that genetic factors contribute to SLI, although it is thought that the genetics underlying SLI are complex, involving several genes that are likely to interact with each other and with a child’s linguistic environment (cf. SLI Consortium, 2004). Although it is early in this line of investigation, there is reason to suspect that the genetic contributions are probably not uniform across linguistic dimensions or related cognitive variables.

A vital element of the emerging genetics studies is the characterization of the behavioral phenotype that is thought to be a core deficit of SLI (cf. Smith, 2004; Smith & Morris, 2005). Measures to identify affected individuals must be sensitive to the disorder (i.e., able to identify a high percentage of affected individuals), and they must be specific (i.e., able to identify a high percentage of unaffected individuals). Further, a good phenotype has validity for interpretation of the nature of the disorder. Operationally, this amounts to a search for clinical markers that yield little overlap of affected and unaffected groups of children.

The prevailing methods have relied on samples of affected children identified on the basis of comparison with age-level peers. Such methods are essential for establishing appropriate levels of sensitivity and selectivity. At the same time, they do not capture well the ways in which the linguistic system of affected children is similar to or different from that of unaffected children over a developmental trajectory, nor the ways in which an affected child’s linguistic system does or does not fully approximate the

expected end-state grammar. Recent studies of the acquisition of finite verb morphology show how a developmental perspective of SLI can bring together the accounts of unaffected, typically developing children with accounts of the nature of language impairment. A further advantage of the developmental perspective is that it could provide a developmentally calibrated phenotype, such that a person's affectedness could be described quantitatively as a slope of the acquisition trajectory and/or age of asymptote (cf. Francis, Shaywitz, Stuebing, Shaywitz, & Fletcher, 1996, for a study of reading disability with a similar developmental perspective). This possible refinement of phenotype definition has yet to be formally evaluated, but it is clearly on the horizon for future work.

### **Tracking the Developmental Course of SLI Relative to Normative Development: Delay versus Disruptions in Language Acquisition**

An important distinction in the study of children with SLI is the notion of delayed language acquisition versus deviant or disrupted acquisition. It is possible that affected children are like younger typically developing children, that is, that there is a general immaturity in the language acquisition system. This model is a conservative model, in that it assumes that the mechanisms of language acquisition, once activated, are very similar in affected and unaffected children. Experimentally, a delay model is evaluated by means of a control group of younger children at equivalent levels of general language acquisition, most often benchmarked during the preschool years as equivalent levels of mean length of utterance (MLU). This sets up a three-group design in studies of SLI that has proven to be very informative, comprised of an affected group, an age comparison group, and a language equivalent group. If the SLI group is at lower levels than the age comparison group but equivalent to the younger language equivalent group, this is generally viewed as a pattern attributable to the generally lower language competencies of the affected group, more like that of younger children.

By contrast, there are two versions of a nondelay model. One proposes that the language of affected children is "deviant," in the sense that it is qualitatively different from that of unaffected children (cf. Karmiloff & Karmiloff-Smith, 2001). The deviant model, while very interesting, will not be discussed further here, because of space limitations. The alternative nondelay model is one of "disruption" (referred to as a "delay within a delay" model in Rice, 2003, and updated to a "disruption" notion in Rice, 2004a). In this model, some elements of language are out of harmony, or disrupted, relative to others, leading to a lack of synchrony in the overall linguistic system. Evidence for this possibility is lower performance of the SLI group than either of the control groups on a given linguistic dimension, indicating that affected children's low performance extends beyond that expected for a general immaturity relative to age expectations. Such an asynchrony is a good candidate for a clinical marker, because the affected children's performance is not likely to overlap with unaffected age peers (cf. Rice, 2000). Areas of disrupted synchrony are of theoretical interest, because they show how the linguistic dimensions that are tightly intertwined in typically developing children are to some

extent independent elements that can be selectively affected and fall behind in acquisition.

### **Delayed Onset: Late Talkers**

Some otherwise healthy children are slow to begin talking, as is evident in smaller vocabularies than their peers at 24 months of age. These children are referred to as “late talkers.” Late onset of language is a hallmark characteristic of children with language impairments. In the case of children with SLI, late talking can be the first diagnostic symptom. Tager-Flusberg and Cooper (1999) called for studies of early identification of SLI, “with particular emphasis on predicting which late talkers develop SLI” (p. 1277). Estimates of proportions of 2-year-olds who are late talkers have been hampered by limitations of ascertainment. Until recently, the available evidence was drawn from self-selected, predominantly middle class families. A new investigation by Zubrick, Taylor, Rice, and Slegers (under review) reports outcomes from an epidemiologically ascertained sample of 1,766 children of 24 months old for whom there were parent reports of vocabulary and first word combinations, as well as a six-item rating scale for general communication abilities, including early receptive language. In this large sample, 13% of the children met the definition of “late talker.” Further, there was extensive information about maternal, family, and child characteristics. It is noteworthy that in this large sample, variables associated with home resources (cf. Entwistle & Astone, 1994), including mother’s education, family income, socio-economic status, parental mental health, parenting style, and family functioning, did not predict late talker status. The significant predictors were only a handful out of a large number of variables: gender (2.74 times the risk for boys than girls); the family history of speech and language delays (2.11 times the risk for families with a positive history); number of children in the family (double the risk for families with two or more children); perinatal status (1.8 times the risk for children with a low percentage of expected birth weight or gestation age less than 37 weeks); and the child’s early neuromotor skills (more than double the risk for children somewhat late in developing motor skills, although it must be emphasized that the late talker children’s motor development is within normative expectations). These outcomes suggest that a child’s genetic make-up and certain constitutional attributes are associated with the timing of language onset.

The available evidence about the longitudinal outcomes for children who are late talkers is comprised of very small samples of children followed over time, drawn from a restricted range of mothers who agreed to participate: Rescorla (2002) followed 34 children; Paul (1996), 31; Whitehurst and Fischel (1994), 37; Thal, Tobias, and Morrison (1991), 10 children. The data are complicated further by some differences in the arbitrary means of identifying affected children. Keeping the limitations in mind, the best estimate for children with a history of late talker status who are classified as SLI at age 6 years seems to be in the range of 17–25% (cf. Paul, 1996; Rescorla, 2002).

The other approach to exploring the full growth trajectory of children with SLI is to ascertain children in the 5- to 6-year age range, follow them over time, and project the

obtained growth curves to an earlier period of development. In the following section, we see that this method also points toward delayed onset as part of the overall picture of SLI, and in addition it reveals important areas of disrupted language growth, out of sync with the rest of the language system.

### **Delays in the Language Growth Trajectories of SLI**

Evidence of a consistent pattern of language delay is evident in the growth of MLU and receptive vocabulary in children with SLI. Rice, Redmond, and Hoffman (in press) followed a group of 21 five-year-old children with expressive/receptive SLI (screened for speech impairment) and 20 MLU equivalent children who were two years younger. The children were assessed at 6-month intervals for MLU, for a total of nine data points over 5 years. The two groups showed remarkable parallels in MLU growth. They were at equivalent levels of MLU at each time of measurement, ranging from  $\sim 3.7$  to  $\sim 5.2$  from the first to last time of measurement. Growth curve modeling showed that there were no group differences in the growth trajectories; each group showed linear and quadratic growth, with negatively accelerating growth such that at the later times of measurement there was less of an increase in the MLU between times of measurement. It is as if the mechanisms that guide increased utterance length are working in the same way in the two groups over the observed time, even though the affected children are two years older than the controls, were enrolled in language intervention at the outset, and were at higher levels of formal education. Projecting the growth trajectories downward, and assuming the continued parallel growth patterns early on, there is strong implication of a delayed onset of the system of combining words into phrases and clauses for affected children.

Growth in receptive vocabulary was tracked in the two groups, as well. Receptive vocabulary was measured annually by the raw score on the Peabody Picture Vocabulary Test-Revised (Dunn & Dunn, 1981). The groups were not initially selected for equivalency on receptive vocabulary. At the outset, the affected group had a small but statistically significant numerical advantage ( $M = 32$  raw score vs. 25 for the MLU equivalent group). At the end, the affected group had a small but statistically significant numerical disadvantage ( $M = 80$  for the SLI group vs. 89 for the MLU equivalent group). The groups did not differ in the intervening times of measurement. In the growth model, there were significant linear and quadratic growth parameters, with group differences at the intercept (outset) and in linear rates, such that the MLU equivalent group overcame the initial lower level of performance with a greater degree of linear change subsequently. It is as if the affected children benefited from the two years' age difference at the outset, in the experience needed to acquire new words, but this advantage was overcome by a slightly better rate of learning new words in the younger group.

So the picture of synchrony/asynchrony is mixed during this observation period – receptive vocabulary and MLU growth are not exactly aligned in the same ways across the two groups in the observed period of acquisition, and yet for the middle two years the groups remain equivalent for vocabulary as well as MLU. It seems overly strong to characterize the general vocabulary development of affected children during this time



as “disrupted” from the general growth in language competency as indicated by MLU, given this mid period of alignment. At the same time, there are some indications that younger, MLU equivalent children may be better than affected children in quick incidental learning of new vocabulary items, a learning advantage that could accumulate over time to generate the higher receptive vocabulary levels at the last time of measurement. MLU equivalent children, as a group, are somewhat better than affected children in using syntactic cues in their acquisition of new words (cf. Rice, Cleave, & Oetting, 2000), although they are similar in their need for frequent exposures to novel words in order to store them in memory (cf. Rice, Oetting, Marquis, Bode, & Pae, 1994). Oetting (1999) found that 6-year-old affected children used cues to interpret verb meaning as well as younger MLU equivalent controls, but the affected children were less able to retain new verbs than the younger group. Other studies report mixed outcomes for the SLI/MLU group comparison (cf. Hoff-Ginsberg, Kelly, & Buhr, 1996; O’Hara & Johnston, 1997; Van der Lely, 1994), which Oetting (1999) suggests may be attributable to task differences and memory demands. Overall, the cross-sectional outcomes are consistent with the longitudinal outcomes in suggesting that if there are differences between affected and younger children in the synthesis of general language growth, as indexed by MLU and lexical acquisition, then the differences are relatively subtle, and not yet well replicated.

### **Disruptions in the Language Growth of Children with SLI: Finiteness Marking**

In contrast to the lexical picture, there is robust documentation of notable disruption in the linguistic system of affected children for the grammatical function of finiteness marking. For some time there had been accumulating evidence that morphology associated with verbs was especially problematic for children with SLI, although morphology was widely viewed as a problem of lexical stem + affix, and surface characteristics of morphology, such as perceptual salience, were accorded a strong role in accounting for affected children’s limitations (cf. Leonard, 1998). Recent advances in linguistic theory allowed for a more precise characterization of the nature of the impairments. Finiteness involves tense and agreement features on verbs in main clauses, features that interact with syntactic requirements of clause structures, hence the term “morphosyntax” to capture the close connection of morphology and syntax.

A thumbnail sketch of the adult grammar from this theoretical perspective can lay out some features of interest in children’s grammars. For a more complete description, see Haegemann (1994); for the theory as applied to child grammar, see Guasti (2002); for a short but technically sound synopsis, see Schütze (2004). It is hypothesized that features such as *tense*, *agreement*, and *case marking* are tightly interrelated in the syntax of clause structure. Essentially, the term “finiteness” involves “tense” and “agreement.” Note that the term “tense” is used in two ways: it can refer to the semantics of reference to temporal dimensions (as in “present” vs. “past” tense), and it also has a second sense of a required grammatical property which is not so tightly linked to temporal dimensions

(e.g., the need to insert auxiliary *DO* in questions). Further, agreement involves person and number marking on nouns, markings which are “copied over” onto verbs, where they do not add additional meanings to the verbs. Such features are distinguished from other properties of the underlying syntax.

This framework captures some of the following facts about English grammar. Finiteness is marked by the following morphemes: Third person singular present tense *-s* as in “Patsy runs home every day”; past tense *-ed* or irregular past tense, as in “Patsy walked/ran home yesterday”; copula or auxiliary *BE* as in “Patsy is happy” or “Patsy is running”; and auxiliary *DO*, as in “Does Patsy like to run?” In a simple clause there is only one site for finiteness marking and no more than one finiteness marker can appear, as shown in the following examples where an asterisk is inserted to indicate ungrammatical clauses: \*Runs Patsy home every day; \*Does Patsy likes to run?; \*Patsy is runs home every day; \*Does Patsy is happy? Note that the set of morphemes is not limited to verbal affixes but instead includes irregular stem-internal morphophonological variants and free-standing morphemes as well.

Subject–verb agreement requires agreement of the person/number features on the noun and verb. These sentences violate that requirement: \*Patsy are happy; \*I runs home every day. English requires an overt subject for a well-formed clause, as shown by this ungrammatical utterance: \*runs home every day. Pronoun case assignment differentiates nominative and accusative case, which is determined by syntactic position, as in “She likes him,” and not “\*Her likes he.” It is thought that the tense feature of English is linked to the requirement of overt subjects (in contrast to languages that do not require overt subjects, such as Italian), and the agreement feature is linked to the requirement of nominative case, although the precise technical details of these interpretations are under investigation (cf. Schütze, 2004).

This perspective allows for some fine-grained distinctions to be applied to child grammars, and to interpretations of the locus of disruptions in the grammars of children with SLI. As expressed very succinctly by Schütze (2004, p. 355), “. . . it is possible for children with normal syntactic structures to sound very unlike adults, because in their lexicon certain morphemes either are missing or have incorrect features associated with them.” Finiteness came to the attention of scholars with the observation that in many languages children show an acquisition period in which they produce infinitival forms of verbs where finite forms are required in the adult grammar. At the same early period of word combinations, English-speaking children produce uninflected verbal forms, such as “\*Patsy go home” and “\*Patsy happy.” Wexler and others recognized that the uninflected verbal forms of English were the English variants of infinitives in other early child grammars, thereby unifying the observations across languages and relating the child grammars to the end-state adult grammar.

Wexler initially labeled this an *optional infinitive stage* (Wexler, 1991, 1994, 1996) which was later amended to an *agreement tense omission model* (ATOM) (Schütze & Wexler, 1996; Wexler, Schütze, & Rice, 1998) and then to a *unique checking constraint model* (Wexler, 1998) as the theory evolved to account for a wider range of phenomena across languages. The basic claims about finiteness in English-speaking children, however, remained the same as the theory evolved. The fundamental notion is that, in some languages, young children go through a period in which they seem to treat finiteness

marking as optional, although it is obligatory in the adult grammar; at the same time they know many other properties of clausal construction. In the normative literature, this phenomenon is widely accepted as a general description of young children's grammars. There are ongoing discussions and debates about the nature of the underlying linguistic representations, reasons this period is evident in some but not all languages, and the way in which finiteness is linked to null subjects and case marking.

The theory was extended to children with SLI in the prediction that their long delay in the acquisition of verbal morphology is an extension of a phase that is part of younger children's grammatical development (Rice, Wexler, & Cleave, 1995). Early on, it was pointed out that this is, in effect, an enriched *extended development model* (cf. Rice & Wexler, 1996a), which recognizes the many ways in which the language of children with SLI is similar to younger unaffected children, but with a greatly protracted period of incomplete acquisition of grammatical tense marking.

The theory offers some explicit predictions of particular relevance here. One is that the domain of finiteness marking could be uncoupled from other semantic and syntactic properties, in that under this theoretical perspective the computational requirements of tense and agreement checking are distinct from the lexical/semantic elements of the grammar and also are distinct from other syntactic dimensions (cf. Chomsky, 1995; Schütze, 2004). In the context of the discussion here of delay versus disruption of linguistic synchronies, this model allows for a disruption as well as delays. Another prediction is that a set of surface morphemes will cluster together in their performance levels in children, based on their shared underlying function in the adult grammar. This set includes bound as well as free morphemes. Further, at an empirical level, the association among morphemes allows for the calculation of composite variables that enhance psychometric robustness. A third prediction is that although weakness in the finiteness domain can be evident, at the same time other syntactic mechanisms can be unaffected. This translates to the expectation that affected children should be unlikely to make errors indicative of basic syntactic limitations.

Let us consider the first two predictions. These are testable within the three-group design for studies of SLI. Further, longitudinal observations pose an empirically rigorous test of the extent to which the expected associations within the morpheme set are observed, and the extent to which the disruption persists. The children who participated in the longitudinal study of MLU and receptive vocabulary described above also received tasks to measure finiteness marking. The results revealed multiple ways in which the affected group did not perform as well as the MLU equivalent group. Each of the target morphemes showed such a deficit at almost each and every measurement point. Further, the set of morphemes showed strong associations among the items. Finally, the difference was evident across tasks: spontaneous language samples, elicited production tasks, and grammatical judgment tasks yielded the same pattern of outcome. The effect sizes for the group differences are relatively robust, that is, eta square values in the range of 28–54% relative to the younger group, and 31–85% for the age comparison group (Rice, Wexler, & Hershberger, 1998). Models of growth were the same for the two groups, indicating linear and quadratic components for both groups. The predictor relationships were also the same across groups: growth was not predicted by a child's nonverbal intelligence, mother's education, or PPVT-R vocabulary scores at the outset, although a

child's initial MLU did predict rate of acquisition. The findings replicated for irregular past tense, when irregular accuracy was calculated as finiteness marking by regarding over-regularizations as finiteness markers (albeit ones in which the phonological requirements of morphology were not fully worked out) (Rice, Wexler, Marquis, & Hershberger, 2000). Further, the replicated findings with judgment tasks establish that the effects are not restricted to production demands but also are evident in children's likelihood to accept utterances as well formed with the same kinds of omissions that they produce (Rice, Wexler, & Redmond, 1999).

There are also cross-sectional replications from other labs of the basic finding that children with SLI, as a group, are likely to perform less accurately than younger controls on morphemes associated with the finiteness marker (cf. Bedore & Leonard, 1998; Conti-Ramsden, Botting, & Faragher, 2001; Eadie, Fey, Douglas, & Parsons, 2002; Grela & Leonard, 2000; Joseph, Serratrice, & Conti-Ramsden, 2002; Leonard, Eyer, Bedore, & Grela, 1997; Marchman, Wulfeck, & Ellis Weismer, 1999; Oetting & Horohov, 1997). Thus the empirical phenomenon is well established, although there is a lively and flourishing dialog about the interpretation.

To return to the predictions, the first two are well supported by available empirical evidence, to the effect that the grammars of children with SLI show a protracted period of delayed acquisition of finiteness marking relative to their age peers and, more remarkable, relative to younger children. There is strong reason to consider this a disruption rather than a general immaturity, in that performance in this area lags behind general indices of language acquisition such as MLU and receptive vocabulary (at least in some portions of the period between 3 and 7 years of typical development). This part of the grammar is not in full synchrony with other dimensions of language growth in affected children.

Let us return to the third prediction. As noted by Schütze (2004), the theory carries further requirements of clause-by-clause inspection to determine if there are errors of morpheme use that would indicate underlying syntactic deficiencies. Such analyses are laborious and often not done, but in the studies of English-speaking children that carefully code for such possibilities, the general finding is that there are very few errors of usage. More particularly, errors of subject-verb agreement are at best only a very small proportion out of all possible occurrences; errors of word order placement are rare or nonexistent as are violations of the requirement that there can be only a single instance of finiteness marking within a main clause. In short, in the studies carried out in the Rice lab, errors are overwhelmingly likely to be constrained to omissions in obligatory contexts. Leonard, Camarata, Brown, and Camarata (2004) report a similar outcome for the observed error patterns in an intensive training study.

Further, the prediction that there should be an association of pronoun case marking with finiteness marking is also evident in children with SLI, such that "she runs home" is much more likely than "\*her runs home" (cf. Wexler et al., 1998; also see Charest & Leonard, 2004, for a detailed analysis of tense vs. agreement as the source of case assignment in affected children). Collectively, such observations indicate that large portions of the underlying syntactic system must be operating in an unimpaired way in children with SLI. This does not necessarily imply that all syntactic dimensions are robust (cf. the arguments of van der Lely and others that a condition described as "grammatical

SLI,” thought to be a subset of the generic SLI clinical group, can include syntactic limitations).

There are many complexities yet to be addressed. One is the extent to which similar linguistic symptoms and developmental benchmarks appear in different languages. Although the bulk of the available SLI literature involves English-speaking children, studies of other languages are entering the literature, including French, German, Italian, Spanish, Hebrew, Swedish, Greek, and Cantonese. It is beyond the scope of this chapter to address the outcomes in detail. In general, as expected, there are differences across languages in which elements of the linguistic system are vulnerable, although there is also a growing understanding of how the underlying finiteness system is affected even if the surface manifestations are not the same. A most interesting example is the work on bilingual French/English children with SLI who have been studied in Canada (Paradis, Crago, Genesee, & Rice, 2003). An elegant design feature is the use of balanced bilingual children as participants. This means that the language comparisons are not confounded with extraneous variables that are difficult to control when comparing groups across languages. These French/English bilingual children showed grammatical impairments in both languages, and a problem with finiteness marking in both languages, although the precise symptoms varied according to the rules for English and French. Furthermore, the age benchmarks for affected children were similar across the two languages; 8-year-old French-speaking children continue to show deficits in their levels of finiteness marking as do English-speaking children. This is interesting because unaffected French-speaking children master finiteness marking at an earlier age than English-speaking unaffected children. So the delay of finiteness is actually greater in the French-speaking children with SLI, relative to normative expectations. Another important outcome was that bilingual French/English children with SLI do not show a deficit attributable to the bilingualism; their performance was comparable to monolingual French- or English-speaking children with SLI.

As these examples attest, there is much to be gained from cross-linguistic studies and investigations of bi- or multilingual children with SLI. Early findings are yielding some surprising indications of unexpected robustness as well as important documentation of how the symptoms will interact with the structure of the child’s native language.

## **Genetics of SLI: Focus on the Phenotype**

The recent reports of DNA analyses of affected cases and family members are harbingers of an oncoming wave of genetic information. At this early stage of investigation, the findings are mixed but promising. As a higher order cognitive trait, language acquisition is complex, and there are multiple possible ways of defining affectedness. Part of the inconsistency in the current findings is likely to be related to the diversity in definitions. A descriptive review of the evidence is beyond the scope of this paper (see Rice & Smolik, in press, for further details).

A brief summary is as follows. New findings link sites on chromosome 16q and 19q to SLI (SLI Consortium, 2004). The findings are complicated by a lack of replication

across subsamples of the participants, inconsistencies between nonword repetition and an omnibus expressive language measure, and possible age effects, such that the genetic influences are more detectable in children older than 8 years than in younger children. These sites are different from the well-reported FOXP2 gene discovery on chromosome 7, first documented in a large extended family in English, referred to as the KE family (see Fisher, 2005, and Vargha-Khadem, Gadian, Copp, & Mishkin, 2005, for clear overviews). Although the ways in which speech and language are affected in the KE family are still debated, the one way in which the affected members of the family are different from unaffected members is a developmental dyspraxia of the orofacial system, such that all affected members have difficulty in controlling the complex mouth movements that generate speech. Thus, dyspraxia is established as a core deficit, although candidates for additional, independent core deficits include “rule-based learning, lexical acquisition and retrieval, and non-verbal cognition” (Vargha-Khadem et al., 2005). It is worth noting that dyspraxia is ruled out in the definition of SLI, although the other candidate core deficits are implicated in the condition of SLI.

A recent twin study (Bishop, Adams, & Norbury, 2005) evaluated the heritability of tense marking and nonword repetition ability (as an index of phonological short-term memory) in a sample of 173 six-year-old twin pairs. Disrupted development in the finiteness-marking element of grammar is predicted to be related to underlying genetic factors (Rice, 1996, 2000, 2003, 2004a, 2004b; Rice & Wexler, 1996b; Wexler, 1996, 1998, 2003). Nonword repetition is an index of phonological short-term memory that is of interest because it is associated with language impairments and is a candidate clinical marker for SLI (Bishop, North, & Conlan, 1996; Gathercole, Willis, Baddeley, & Emslie, 1994; Tager-Flusberg & Cooper, 1999). Bishop et al. (2005) found that impairments in both areas were significantly heritable, although there was minimal phenotypic and etiological overlap between the two deficits. They conclude: “Our findings are also in agreement with predictions made by Rice and colleagues, in confirming that deficits in use of verb inflections commonly persist beyond the age of 4 years in children with language impairments and are heritable. Most crucially, this study reveals that impairments in use of verb inflections have distinctive genetic origins and cannot be explained away as secondary consequences of limitations of phonological short term memory.” Thus, although it is early on in the investigation of the inherited elements of SLI, these new discoveries point toward the viability of this line of study, and the promise of such investigations.

### **Putting it Together: A Maturational Perspective of Inherited Timing Mechanisms and Developmental Change**

As matters now stand, the genetics studies employ a wide range of language phenotypes, most of which define affectedness by reference to age peers. Although this approach has psychometric value and is beginning to uncover potential genetic linkages, it does not capture well the ways in which the linguistic systems of children with SLI share fundamental growth properties with the linguistic systems of unaffected children, nor the ways

in which the linguistic system can be less synchronized in affected youngsters, nor the ways in which inherited mechanisms can be influencing the growth. An alternative framework is a maturational perspective, suggested by the delays and disruptions within the grammatical system of children with SLI.

A maturational account posits that there are powerful timing mechanisms that activate the onset of language that can be delayed in onset for affected children (cf. Rice, 2003, 2004a, 2004b; Wexler, 2003). Once language onset is activated, there is an expected synchrony in the emerging system that unfolds in typically developing children, in a close interaction with environmental input, including the child's native language and the ways in which adults and familiar people in a child's home environment interact with him or her. Twin studies indicate that the relative contribution of genetics and environment loads more heavily toward genetics than environment for children at the lower levels of language performance during the 2- to 4-year age period, implicating genetic contributions to onset mechanisms that are especially significant for children who start late (Dale et al., 1998; Viding et al., 2004). Under a maturational model, it is predicted that environmental factors would show weaker relationships to onset timing in affected children insofar as the delayed onset is more driven by constitutional weaknesses than insufficiencies in the environment (assuming no gross violation of environmental resources).

For children with SLI, the finiteness-marking component seems to be under different timing mechanisms such that the clock for this linguistic element is running out of sync with the other elements. Bishop et al. (2005) are the first to document a positive genetic contribution to this particular phenotype, in a cross-sectional study of 6-year-old twins. A recent training study provides evidence that if children have not yet started to use the finiteness markers, the effects of intensive training are modest at best. After 48 individual intervention sessions for 31 children with SLI ages 3;0 to 4;4, as a group, the treated children did not move beyond optional use of third person singular present tense *-s* or auxiliary *BE* (Leonard et al., 2004). The maturational model would predict that the mechanisms guiding components of the linguistic system are not necessarily in sync, such that some elements can lag behind, although the general pattern of change mirrors that of unaffected children. The environmental manipulations involved in language intervention would not be likely to reset the acquisition curves if intervention antedates the expected deflection points in the acquisition curve that mark a transition in aptitude for change. As noted by Leonard et al. (2004), language training is not likely to be effective if the children are not ready for the targeted grammatical forms.

Although maturational models have been out of favor as a developmental account, the condition of SLI is calling for a reconsideration of the feasibility of such models and how they can be carefully evaluated. Much of modern genetics is moving toward explicit investigations of the timing mechanisms, that is, the internal clock that is intrinsic to cellular growth and the timing of genetic effects (cf. Purnell, 2003; Rice, 2004a, 2004b). Although studies of language acquisition have been highly sensitive to the need for age-referenced benchmarks in language acquisition, there has been surprisingly little serious attention given to the ways these benchmarks are tied into children's biological guidance mechanisms in interaction with the environmental influences.

Among the benefits of a developmental perspective on language impairment of this sort is that it can be applied more broadly to language impairments in other clinical conditions. Rice (2004a, 2004b) argues that consideration of the delay-with-disruption model focuses attention on the onset, growth trajectories, and possible plateaus of language acquisition. These three elements in turn can be explored across conditions. It may well be the case that a common feature of language impairment is a delayed onset, which in turn may be vulnerable to multiple sources of genetic dysfunction. Delayed development may be more characteristic of some conditions whereas disruption may be more operative in others. Comparison with a language-equivalent, as well as an age-equivalent, control group could help clarify language impairments in disorders beyond SLI.

## Implications for Models of Language Development

Perhaps one of the biggest paradoxes of the study of children with SLI is that the research focus on the ways in which these youngsters are different from unaffected children has also led to an increased appreciation of the many ways in which these youngsters are also attuned to the developmental mechanisms that guide language acquisition in unaffected children. This suggests that the scientific problem is not so much about how to develop different theories for different groups of children, but how to develop theories robust enough to capture loci of possible delay and disruption within the linguistic system, as well as the exquisitely finely integrated system of language acquisition in general. Contemporary lines of investigation promise to generate progress toward this goal.

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