
Growth Models of Developmental Language Disorders

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From cells to whole organisms, there is a time to grow and a time to proliferate; a time to keep silent and a time to express; a time to change and a time to refrain from transformation. But where are the cellular and organismal timepieces and how do they mark off time and keep the myriad physiological events in sync?

—Purnell (2003, p. 325)

The prior quote is from a recent issue of *Science* with a special section on the topic of developmental timing. The temporal events that guide development are key parts of the puzzle of contemporary molecular genetics. Genes are known to turn on at certain times in development; microRNAs show temporal- and tissue-specific patterns of gene expression (cf. Carrington & Ambros, 2003); and genes activate to trigger downstream developmental events removed in time from the triggering event (cf. Gehring & Ikeo, 1999; Marcus & Fisher, 2003). Timing mechanisms are fundamental to the way that inherited elements direct an individual's growth, whether at the cellular or organism level.

The fact that language acquisition is a developmental phenomenon that emerges in the early childhood period is a commonplace observation that is known to parents and scholars alike. The literature is full of debates about the extent to which growth in language early on is attributable to inborn, language-specialized mechanisms; inborn, generalized learning mechanisms that are heavily influenced by environmental input; or fundamental learning

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algorithms that are largely acquired by experience. As the contents of this volume attest, there is a growing consensus that language acquisition is guided by an exquisitely elegant interface of inherited individual aptitudes and personal experiences with other language-users. In other words, genetic influences are modulated by environmental influences. A parallel conclusion is that faulty genetic mechanisms are implicated in language impairments and characterized by breakdowns in the expected development of language.

At this early stage of formal investigation of the genetics of language (cf. Rice, 1996a), although the developmental dimension of language acquisition is so salient, there is surprisingly little in the literature about the developmental dimension of the phenotype and underlying timing mechanisms. Recently the first wave of studies established genetic influences on the basis of one-time, static classification of the phenotype, either as a categorical or quantitative phenotype. Given the current state of inquiry, this approach to phenotyping is of vital importance and is essential to progress in the identification of genotype/phenotype correlations. Current attention focuses on the need for greater precision in the language phenotype, for improved accuracy in the identification of affected children, and more precise description of the dimensions of affectedness.

In this chapter, I direct attention to the timing elements of language impairment as potentially of great import in moving us forward in our understanding of causal mechanisms and genotype/phenotype relationships. I draw on longitudinal outcomes in ongoing studies of children with specific language impairment (SLI) to demonstrate what appear to be powerful timing mechanisms at work in language impairment. Just as the developmental program of gene expression in the cells of plants and animals shows timing differences across structures and tissues, the developmental trajectory of language growth in children shows different timing mechanisms across various elements of the linguistic system. Although children with SLI can be identified on the basis of weaknesses within the linguistic system, at the same time their growth trajectories show strikingly robust parallels with the trajectories of unaffected children. It is as if the timing mechanisms are set to unfold in the same way once activated. Thus, there is a puzzling combination of developmental robustness in language acquisition coexisting with developmental weaknesses in the linguistic system for children affected with SLI. To make sense of the full picture, one must consider the following elements of linguistic growth: onset timing, configuration of the linguistic system with delineated subcomponents, acceleration rate, and points of change in the acceleration. Consideration of these elements will lead to further specificity of the language phenotype and to phenotypes that apply across different clinical conditions. It will also help with the investigation of possible etiologic subtypes attributable to genetically based timing mechanisms.

The conclusions of the chapter require some preliminary sections. First, to provide a background on language and language delays, and the design rationale for control groups, a nontechnical metaphor of language acquisition and impairments is provided. This is followed by a section describing the condition of SLI and the genetics of SLI to serve as background for the illustrative growth curves to follow and to establish the plausible relevance for genetics of language. The section on illustrative growth outcomes begins with a description of the groups of children who participated in the studies, as well as the design and interpretive rationale for observed group differences. The motivation for the language measures is laid out next, to establish a focus on finiteness and the expectation that in English-speaking children the acquisition of finiteness-marking is delayed, in the form of an Optional Infinitive period, and that this delayed period is even more extended in children with SLI (an Extended Optional Infinitive period), which leads to measures that serve as a “grammatical tense marker” that can differentiate affected from unaffected children. This section ends with a summary of what is known about the grammatical tense marker in children with autism, Down syndrome, and Williams syndrome. The core section on growth appears next, which reports on patterns of change over time in children with SLI compared with unaffected younger children. The picture that emerges is one of robust maturationally governed growth for the affected children as well as unaffected children. Yet the strengths, delays, and disruptions are referenced to linguistic distinctions, and the affected children may never reach fully adultlike competencies. A section then compares growth outcomes for children with language impairment above and below the 85 nonverbal IQ level. Although a lower IQ does not necessarily lead to low performance on the grammatical marker, in concert with language impairment it protracts growth for a longer time. Next is a discussion of timing and possible maturational mechanisms, followed by a summary of future directions for growth-focused research across clinical conditions. The chapter concludes with some implications for genetics of language impairments and future directions.

ELEMENTS OF LANGUAGE GROWTH: ONSET, CONFIGURATION OF THE LANGUAGE SYSTEM, ACCELERATION, AND RATE OF CHANGE

Just as a train has an expected configuration of engine, cars, and tracks, so does the language system. Lexical, syntactic, morphological, and computational elements must mesh together in a tightly synchronized way, roughly analogous to the alignment of cars, wheels, connections between

cars, engine, drive shafts, and so on. These elements are constrained to follow a developmental trajectory, again roughly analogous to the tracks of the train that constrain the route of forward motion. As described by Phillips (chap. 11, this volume), linguists conclude that there is a common cognitive infrastructure available to children—an aptitude that enables them to acquire any of the world’s languages during the early period of childhood. From this perspective, the configuration and constraints of the train’s structure and path of acceleration can be thought of as this common infrastructure. The fundamental operating system and constraints are thought to be the same, although details of the train’s configuration vary across languages.

Trains have an expected time of departure. This is when the acceleration mechanisms are activated. When the language train leaves on time, as in Panel A of Fig. 10.1, parents are pleased that their youngster is beginning to understand and use words; the language system is likely to emerge on the expected subsequent trajectory. For unknown reasons, however, the language train can be *delayed at departure*, as in Panel B of Fig. 10.1. Although the startup time is delayed, the train may follow the expected acceleration patterns afterward, in which case the youngster never catches up with the child whose train leaves on time. Another possibility is that the late-starting child has an unknown factor that resets the acceleration rate to catch up to the expected rate, with a subsequent deceleration to follow the expected rate. This popular model of “catch up” is in fact a complex model of growth with little known about the mechanisms that readjust rate. Intervention seems to adjust acceleration rates for at least some elements of language, as described by Warren (chap. 9, this volume), but it is not clear what the exact mechanisms would be or how or why the rates would subsequently stabilize at the normal rate following intervention.

Another scenario is shown in Panel C of Fig. 10.1, in which the language system is not only *delayed*, but also *disrupted* in the expected configuration. In this terminology, *disrupted* is the dictionary sense of “impeding the usual course or harmony of.”¹ In this case, although the overall momentum of language growth is forward, certain elements are slowed, thereby disrupting the overall harmony. This can appear in localized elements of the language system, where perhaps coupling or computational relations between elements are not the same as the expected alignments. Such a language system could start late, and perhaps some elements might eventually catch up to those of unaffected children, or perhaps the

¹In an earlier publication (Rice, 2003), I referred to this scenario as a “delay-within-a-delay” outcome. Here I adopt the *disruption* terminology as a way to encompass a broader range of phenomena that may hold beyond the classic SLI situation.

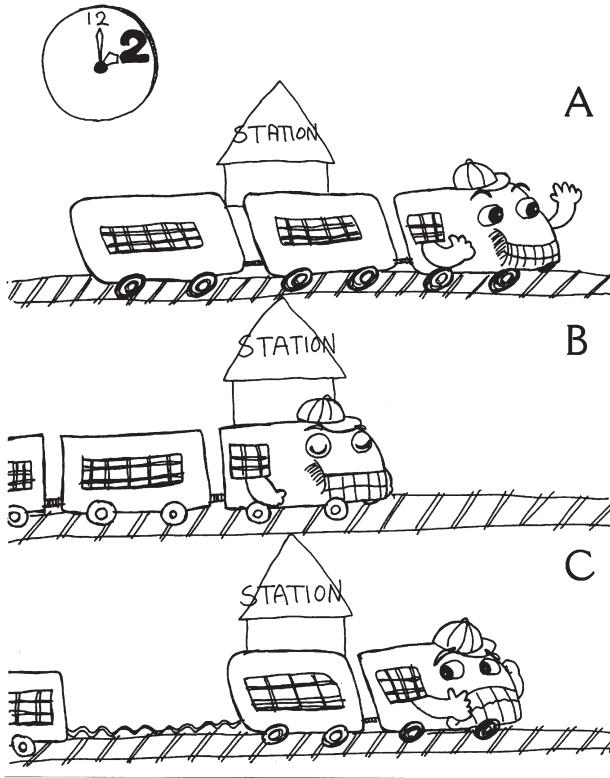


FIG. 10.1. A train metaphor of language acquisition, delays, and disruptions.

disrupted element is never fully resolved and remains out of sync for protracted periods, even into adulthood as is suggested by current evidence. In the *disrupted* scenario, it is essential to have the details about different elements of the linguistic system to capture where the system is robust, where it is delayed, and where it is disrupted. Whereas in the *delayed* system it is possible to predict one element of the linguistic system from other elements in the system according to the relationships evident in the unaffected language system, in the *disrupted* system this language-internal prediction may vary from the expected relationships.

Specific Language Impairment: Clinical Definitions

The condition of SLI is conventionally defined according to inclusionary and exclusionary criteria (cf. Leonard, 1998; also see de Villiers, 2003, for a thoughtful examination of the criteria). The inclusionary criteria establish

that a child has a level of language growth that is lower than expected for his or her age. Typically, this is determined by performance on an omnibus measure of language performance, which includes a variety of language tasks, item selection by psychometric criteria (as opposed to linguistically motivated item sets), blocking of tasks by performance versus comprehension mode of response, and differentiation of tasks by major elements of language such as semantics, syntax, and/or narrative tasks. The dividing line between “normal” and “affected” is roughly at the 10th to 15th percentile of the chronological age comparison group, often defined as one standard deviation below the age group mean or a somewhat lower cutoff of -1.25 standard deviations below the age mean, which has been identified as a level that corresponds to practitioner clinical judgments (Tomblin, Records, & Zhang, 1996).

Recently, new instruments report outcomes in terms of sensitivity and specificity. Sensitivity is the likelihood of correct identification of affected children; specificity is the likelihood of correct identification of unaffected children. Sensitivity and specificity estimates require the usual reference group of a normative distribution of children of the same age and, in addition, a group of children of the same age who are identified as language impaired (cf. Rice & Wexler, 2001; see Mervis, chap. 8, this volume, for an extension of these concepts to profiling of relative cognitive strengths and weaknesses). The psychometric approach to assessment is also being supplemented by newer linguistically motivated instruments intended to target particular areas of linguistic competence that can be meaningfully compared to the adult linguistic system (cf. Rice & Wexler, 2001; Seymour, Roeper, & de Villiers, 2003; Zukowski, chap. 6, this volume). A strong advocacy for linguistically motivated instruments is put forth by de Villiers (2003), who argued for substantive advantages for defining children as affected on the basis of precisely defined linguistic deficits. All things considered, the inclusionary criteria are evolving so that we have more precise information to identify the phenotype of language impairment.

The exclusionary criteria have conventionally ruled out hearing loss, intellectual impairment, known neurological conditions, and the condition of autism. The precise purpose of the exclusionary criteria in research investigations is sometimes unclear for a particular investigation. In general, the criteria have served as a way to document the existence of language impairments in children for whom other developmental competencies are at levels thought to be sufficient for language acquisition, thereby establishing the existence of language impairment without obvious concomitant contributing conditions. Another research benefit of the exclusionary criteria is that the criteria help restrict unknown sources of error or variability within a given clinical sample, which enhances replicability and generalization of findings across studies. These exclusionary criteria

are now under reexamination to determine whether there are areas of overlap between: SLI and autism, SLI and mild hearing impairment, or SLI and borderline levels of nonverbal intellectual impairment (Conti-Ramsden, Botting, & Faragher, 2001; McGuckian & Henry, 2003; Norbury, Bishop, & Briscoe, 2001; Plante, 1998; Rice et al., under review; Tager-Flusberg, chap. 3, this volume; Tager-Flusberg & Cooper, 1999).

Finally, another area of possible overlap in the clinical symptoms of SLI is the likelihood of speech impairments coexisting with language impairments. Although it was long assumed that problems with speech production and related unintelligibility of speech should be expected for children with language impairments, recent epidemiological findings establish that by 5 to 6 years of age, the estimated co-occurrence of speech and language impairments is less than 2%. For children with SLI, speech disorders were evident in approximately 5% to 8% of the children (Shriberg, Tomblin, & McSweeney, 1999; cf. Tager-Flusberg, chap. 3, this volume, for similar findings for children with autism).

Genetics of SLI

Putting aside the details of definitional issues, there is a growing body of evidence establishing a strong likelihood of genetic contributions to SLI. Case-control familial aggregation studies document aggregation for SLI probands (Rice, Haney, & Wexler, 1998; Tallal et al., 2001; Tallal, Ross, & Curtiss, 1989; Tomblin, 1989; van der Lely & Stollwerck, 1996). New findings provide explicit evidence of possible genetic influences on verb morphology deficits in 6-year-old twins. Using the experimental tasks for elicitation of third-person singular present tense *-s* and past tense developed by Rice and colleagues (described in further detail later), Adams and Bishop (2002) used the DeFries–Fulker analysis to investigate MZ and DZ twins. They found a shared genetic influence on these grammatical markers as predicted by Rice and Wexler (1996a), as well as evidence of heritability of nonword repetition, although the genetic influence on this trait was not shared with tense marking. In personal communication, Bishop cautioned that the bivariate analyses should be considered preliminary. The twin data join the recent identification of the *FOXP2* gene on Chromosome 7 for a large extended family in London known as “KE” (Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001). Although the *FOXP2* gene discovery is surely an important one, the phenotype of the affected individuals is complex, with severe oral dyspraxia, language impairments, and some individuals with limited nonverbal intelligence. Attempts to identify this gene as affected in individuals with SLI or autism have so far been unsuccessful (Meaburn, Dale, Craig, & Plomin, 2002; Newbury et al., 2002; SLI Consortium, 2002), although it is too early to

rule out a possible role for *FOXP2*. A new study by O'Brien et al. (2003) reported positive association of SLI to the region of 7q31. Although no mutations were found in *FOXP2*, strong association was found on markers adjacent to *FOXP2* (one on the *CFTR* gene and one on 7q31, D7S3052). Sample sizes ranged from 58 to 164 family triads. The language phenotype was based on performance on standardized language tests at second grade, and the performance IQ level was broadened beyond the conventional level of 85 to include children at 70 or above. This raises the possibility that the inherited elements may involve the overlap of lowered IQ with language impairments. At the same time, inclusion of children with low nonverbal IQ may contribute confounding variance that obscures genetic contributions to language impairments (although Marcus & Fisher, 2003, argued this is not the case for the KE family). Meaburn et al. (2002) included low IQ children and found no connection to *FOXP2*, whereas the SLI Consortium (2002) and Newbury et al. (2002) excluded children whose nonverbal IQ was less than 80 and also found no connection.

In addition, Bartlett et al. (2002) reported a major susceptibility locus for SLI on 13q21 in a study of five Canadian pedigrees. They used three categorical phenotypic classifications: clinical diagnosis (via family history), language impairment (below normal range on a standardized omnibus language test), and reading discrepancy (nonverbal IQ minus reading level). The locus was identified using the reading phenotype, and the authors noted the difficulty in differentiating the possible role of the SLI language impairment in the reading phenotype. It is well known that there is an increased likelihood relative to controls for young children with SLI to subsequently encounter problems with reading (Catts, Fey, Tomblin, & Zhang, 2002).

At this early stage of inquiry, it continues to be important to aim for precise measurement of the phenotype for the identification of affected individuals (Smith, chap. 13, this volume). Family history methods can be inconsistent with current testing (cf. Tallal et al., 2001), language impairments that are outgrown can be missed in older children and adults, omnibus language assessments are broadly configured and may obscure important details of linguistic performance, and the current identification methods do not include developmental information.

Illustrative Growth: Description of the Child Participants, Design, and Interpretive Rationale

The following illustrative growth data are drawn from an ongoing longitudinal study initiated in 1993 with funding from the National Institute of Deafness and Communicative Disorders (NIDCD, Award #R01 DC01803). This section provides a general description of the child participants, de-

sign, and interpretive rationale of the design. More detailed descriptions are available in Redmond and Rice (2001), Rice and Wexler (1996b), Rice, Wexler, and Cleave (1995), Rice, Wexler, and Hershberger (1998), Rice, Wexler, Marquis, and Hershberger (2000), Rice, Wexler, and Redmond (1999), Rice (2003), Rice (2000), and Rice (1997).

Child Participants. The participants include children identified as SLI and typically developing comparison groups of children. The affected children were ascertained from clinical caseloads of speech/language pathologists. The children in the SLI group met the following exclusionary criteria at intake: no hearing loss, nonverbal IQ of 85 or above, no diagnosis of autism, no intelligibility problems with speech, a passing score on a screening test of word-final sounds used in grammatical morphology, and no known neurological impairments.

The inclusionary criteria identified children with receptive and expressive language impairments defined as follows: performance one standard deviation or more below the age group mean on the Peabody Picture Vocabulary Test–Revised (PPVT–R; Dunn & Dunn, 1981), on the Test of Language Development–Primary (TOLD–P:2; Newcomer & Hammill, 1988), and on the mean length of utterance (MLU) obtained from a spontaneous speech sample (Leadholm & Miller, 1992).

The figures reported here are from a longitudinal sample of 23 affected children followed for more than 10 years. The children were in the age range of 52 to 62 months ($M = 56$ months) at the initial assessment. An additional 60 children with SLI who meet the same criteria (and who do not differ from the original sample in mean levels of performance on the PPVT, TOLD–P:2, and Goldman–Fristoe Test of Articulation; Goldman & Fristoe, 1986) have subsequently been recruited and are followed longitudinally. The additional recruits to the longitudinal study have verified that the growth outcomes are characteristic of the full sample of 83 affected children, suggesting that the illustrative growth curves are likely to generalize to other groups of similarly defined children. Furthermore, the growth curves of the affected children are highly similar to those obtained from an epidemiologically ascertained group of 130 children with SLI recruited at kindergarten, which included children with only expressive or receptive disorders as well as those with expressive and receptive disorders (Rice et al., in press). Although definitive conclusions await more extensive data collection and analyses, the working conclusion here is that the illustrative growth curves of affected children (in Figs. 10.2– 10.5) are likely to be representative of many, if not most, of the children who meet a conventional definition of SLI.

Two groups of typically developing children were recruited as control groups: One was the same chronological age (age range of 52–67 months,

$M = 59$) at initial assessment, and the other was the same MLU (indexed by MLU in morphemes, although the same outcomes hold for MLU in words; range of 2.25–4.64 in the affected group, $M = 3.49$; 2.75–4.81 in the control group, $M = 3.66$). The MLU comparison group was selected on the basis of MLU, which yielded a mean age about 2 years younger than the affected group (with an age range of 30–44 months, $M = 35$ months).

A family study (Rice, Haney, & Wexler, 1998) found evidence of family aggregation in this sample. The rate of reported affectedness was higher in the SLI group (probands) than in the control children's families. About 22% of nuclear proband family members reported speech and/or language difficulties as compared with 7% of nuclear control family members; at the level of extended family (including aunts, uncles, cousins, and grandparents), the rate is 15% of proband family members versus 6% of control family members.

Design and Interpretations. Following a widely used design in the study of SLI (cf. Leonard, 1998), the longitudinal study compared the growth of three groups: SLI, chronological age (CA) match, and language (MLU) match. The CA group allowed for documentation of: (a) the expected age-referenced growth in the target areas of language; (b) the lower level of performance of the affected group relative to age expectations on the experimental dependent outcome variables as well as the initial grouping variables; and (c) the age-referenced levels for possible catch-up for the affected group as growth occurs. To return to the train example, the CA group describes the outcome of Panel A, with a normally configured train following the usual acceleration mechanisms.

The MLU group allowed for evaluation of the train scenarios in Panels B and C. As noted earlier, if the affected group performed at levels of language similar to the younger children at the same general language levels, it is presumed that the group's performance is consistent with a *delay* in language acquisition. That is, the children have not yet overcome a late start in language acquisition, leaving them with a generally immature language system that does not show particular areas of deficit once the late start is taken into consideration (see Panel B).

In many ways, the generally immature system is the default expectation, consistent with the idea that the affected children are following the same mechanisms of language acquisition as other children, but they are slow in getting started. Under this model, the onset timing mechanisms are carrying the bulk of the interpretive weight. It is widely assumed that children with a late start will relatively quickly catch up to their age peers. About 75% of children whose first words appear later than expected—sometimes known as “late talkers”—recover from their initial delays by 6 years of age (cf. Paul, 1996; Rescorla, 1993, 2002), although the available

evidence is limited to a few studies of small numbers of children, and long-term outcomes at ages 8 to 9 years are not robust. As noted earlier, such a recovery would involve a relatively complex pattern of developmental change that requires some resetting of the expected acceleration rates. The initial rate is slow, then it accelerates greater than usual, then it decelerates to the normal rate after the age-expected level is reached (otherwise the late talkers would shoot up into the range of verbally gifted, and that is not attested to by the outcome data). The timing mechanisms or environmental effects for accomplishing such adjustments in rate are not known.

The other possible scenario is depicted in Panel C of Fig. 10.1, in which some elements of language acquisition in the SLI group lag behind that of the younger MLU-equivalent group in a way that shows *disruption* of the expected language configuration. Three implications follow from this scenario. If some elements of language show even lower levels of performance than expected by a general delay, then those elements must correspond to some potential lines of cleavage in the linguistic system. Growth in unaffected children is so fast and well synchronized that it may obscure the different systems coming on-line and changing in tandem. Second, an element that lags behind other language components would be a good candidate for a clinical marker because the low levels of performance are less likely to overlap with the lower end of the normative range, allowing for clearer differentiation of affected and unaffected children (as indexed by sensitivity and specificity). Third, the genotype/phenotype correlation may be especially detectable in this element because the identification of affected individuals is more accurate.

Illustrative Growth: Grammatical Tense as a Clinical Marker

By the early 1990s, it was generally recognized that children with SLI have difficulties with morphology and verbal morphology, 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). The Extended Optional Infinitive account (cf. Rice & Wexler, 1995; Rice, Wexler, & Cleave, 1995) was developed as an alternative interpretation based on developments in theoretical linguistics and the Optional Infinitive model of a stage of acquisition observed in young unaffected children (cf. Wexler, 1991, 1994, 2003). This model focuses on the grammatical features of tense and subject/verb agreement, which are involved in finiteness marking on verbs and the way these features are operative in clausal structures. Roughly speaking, each main clause in English (and

many other languages) has functional projections for tense and agreement marking that are related to movement operations among clausal elements, hence the term *morphosyntax* (cf. Haegeman, 1994). In many languages, children show an acquisition period in which they produce infinitival forms of verbs where finite forms (i.e., those marked for tense and/or subject/verb agreement) are required in the adult grammar. At the same early period of word combinations, English-speaking children produce uninflected verbal forms such as “Papa have it,” “Cromer wear glasses,” and “Marie go.” 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 (cf. Guasti, 2002). Wexler referred to this period as *optional infinitive*; an alternative term is *root infinitive*. Both have come to be used descriptively without any theoretical differentiation between them. 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, while they know many other properties of clausal construction. In the normative literature, the phenomenon of optional (root) infinitives has become an accepted general description of young children’s grammars. It has spawned a rich and ongoing debate 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 other properties of clausal structure, such as null subjects and case marking (cf. Phillips, chap. 11, this volume).

The Extended Optional Infinitive (EOI) model hypothesized that the long delay in acquisition of verbal morphology by children with SLI is an extension of a phase that is part of younger children’s grammatical development. It can be viewed as an enriched Extended Development Model (cf. Rice & Wexler, 1996b), 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. More recently, Rice (2003) reexamined the notion of language delay to note that the extended difficulties with finiteness-marking (the grammatical tense marker) have implications for explicating the nature of language impairment across clinical conditions. This in turn helps sort out the ways in which genetic and environmental etiological factors contribute to language impairments in children. In this chapter, these arguments are extended to the issue of timing mechanisms.

The EOI model focuses on a small set of morphemes in English that mark tense and agreement and are obligatory in clausal structure. They are: third-person singular present tense *-s* (e.g., *Patsy walks*); regular past tense *-ed* (e.g., *Patsy walked*); irregular past tense (e.g., *Patsy ran*); copular

BE (e.g., *Patsy is happy*); and auxiliary *BE* and *DO* (e.g., *Patsy is walking*; *Does Patsy walk?*). Note that the set of morphemes varies in that some are affixes and some are free-standing morphemes; some can move in questions (i.e., *BE* copula and auxiliary), but others cannot (i.e., third-person singular present tense; past tense) and one is uniquely inserted in questions (i.e., *DO* auxiliary). Although past tense has a salient semantic component, the other morphemes carry little or no apparent semantic weight in a clause. Instead the group of morphemes is thought to be part of a grammatical computational system that is more about grammatical well formedness than the expression of meaning.

A series of research reports from the longitudinal study documented that, as predicted individually and collectively, the finiteness-marking morphemes differentiated the performance of children with SLI from younger MLU-matched children in: spontaneous language samples, elicited production tasks, and grammaticality judgment tasks—across multiple times of measurement (cf. Redmond & Rice, 2001; Rice & Wexler, 1996b; Rice, Wexler, & Cleave, 1995; Rice, Wexler, & Hershberger, 1998; Rice, Wexler, Marquis, & Hershberger, 2000; Rice, Wexler, & Redmond, 1999). These outcomes support the theoretical assumption that this part of the grammar is to some extent a discrete component in the overall linguistic system.

These investigations are now part of a lively scientific dialogue about the EOI model and other models of SLI, children's acquisition of the set of target morphemes, and interpretation of the grammatical marker. In general, the basic finding replicated across labs is that, as a group, children with SLI perform less accurately than younger controls on morphemes associated with the grammatical marker of the EOI model, although the details vary depending on the ages of participants and the level of linguistic detail. Reports from other investigators include studies of the acquisition of copula and auxiliary *BE* and possible constraints on optionality (Grela & Leonard, 2000; Leonard, Eyer, Bedore, & Grela, 1997; Joseph, Serratrice, & Conti-Ramsden, 2002); past tense (Marchman, Wulfeck, & Ellis Weismer, 1999; Oetting & Horohov, 1997); and third-person singular present *-s* and past tense (Bedore & Leonard, 1998; Conti-Ramsden, Botting, & Faragher, 2001; Eadie, Fey, Douglas, & Parsons, 2002). Rescorla and Roberts (2002) reported that children with a history of late talking at 3 and 4 years of age show deficits relative to age controls on all four of the tense-marking morphemes they investigated in spontaneous speech samples (i.e., copula and auxiliary *BE*, auxiliary *DO*, and third-person singular present *-s*). Oetting and McDonald (2001) found that the grammatical marker is evident in children speaking various dialects of English.

These findings, and the newly available normative data from a nationally standardized assessment instrument (Rice & Wexler, 2001), demonstrate that the grammatical marker of the EOI model is evident in young

children with SLI, and it appears as a delay even beyond that of a generally immature language system. As proposed early on (cf. Rice & Wexler, 1996a; Tager-Flusberg & Cooper, 1999), the grammatical marker of the EOI is shaping up as a solid clinical marker of language impairments in children with SLI and a good candidate for a phenotype as demonstrated by the findings of Adams and Bishop (2002).

Grammatical Tense Marker in Other Clinical Populations. Studies of the grammatical tense marker in other clinical conditions show patterns of relative strength and limitations. Tager-Flusberg and colleagues explored the marker in children with autism and found that language-impaired children with autism performed below nonlanguage-impaired children with autism on the same elicitation tasks used in the Rice et al. studies for past tense and third-person singular *-s* (Roberts, Rice, & Tager-Flusberg, in press). In a comparison study of nonlanguage-impaired children with autism, language-impaired children with autism, and children with SLI matched for nonverbal IQ and MLU, they found that the two language-impaired groups were below normal levels on the third-person singular *-s* and not below normal on other morphemes (tense-marking and nontense-marking) although the children were as old as 13 years (Condouris, Evancie, & Tager-Flusberg, 2002).

Studies of children with Down syndrome have documented that these youngsters are likely to perform below mental age on general language tests and more poorly on the morphology and syntax subtests than on vocabulary subtests (cf. Abbeduto & Murphy, chap. 5, this volume). Eadie et al. (2002) examined the grammatical tense marker and control morphemes in spontaneous language samples in 10 children with Down syndrome, 10 children with SLI, and 10 normal controls matched for MLU. They concluded that the SLI profile is similar to that of children with Down syndrome. Unexpectedly, the performance on the tense-marking morphemes did not differ from the control morphemes for either group—an outcome attributable to somewhat anomalous findings for certain morphemes, which in turn are possibly attributable to problems of sampling validity in the spontaneous language samples. Although the study needs to be replicated, it is well motivated and points in the direction of appropriate future studies.

Children with Williams syndrome show a contrasting profile. Rice, Mervis, Klein, and Rice (1999; also reported in Rice, 2003) compared the grammatical marker in samples of children with Williams syndrome matched for MLU to the longitudinal SLI children and younger control children studied by Rice and colleagues as reported in the growth curves in this chapter. The samples were compared at the first time of measurement when the SLI children were 5 years of age, younger controls were 3 years, and the WMS group was a mean of 7.7 years. The outcomes show

that the performance of the WMS group was at higher levels than the SLI and younger controls and, in fact, were at levels similar to the 5-year-old unaffected group—near ceiling. In short, the WMS group’s mean level of performance on the grammatical marker was near adult levels of competence, although their MLU was like unaffected children 3 years of age.

Although comparison of performance on the grammatical tense marker across clinical conditions is at an early stage of investigation, these early findings clearly show that this part of the grammar can be selectively spared or impaired relative to other levels of language growth. Many details remain to be explored regarding the ways in which morphosyntax is acquired across different conditions of language impairment.

Illustrative Growth of Grammatical Tense Marking: Patterns of Change Over Time of Affected and Unaffected Younger Children

Let us begin the discussion of growth in the age range of 3 to 8 years, which corresponds to the time in which English-speaking children move from optional to obligatory when they use the morphemes of interest in the clinical grammatical marker as part of simple clauses, including the extended period for the children with SLI. In this age range, growth in the grammatical marker is essentially completed in unaffected English-speaking children by ages 4 to 4;6. Recall that the affected children and chronological age controls were ages 4;6 to 5;6 at the start of the study. Because the age control group was, as expected, at adultlike ceiling levels, no growth is apparent. Therefore, the groups of interest for growth are the affected group and the younger MLU-equivalent group.

Delays in Utterance Length and Vocabulary Growth. A delay model predicts that the affected children would track alongside the younger children in general indexes of language acquisition. This is apparent in the utterance length of spontaneous utterances and semantic development. Figure 10.2 shows a schematized rendering of the group means as a function of age. The semantic measure is a child’s raw score on the PPVT-R (a measure of receptive vocabulary on a picture-pointing task), which was administered annually; the utterance length (MLU-morph) was measured in 6-month intervals (see Figs. 2.14 and 2.15 of Rice, 2003, for actual means and range of performance per group per time of measurement). In Fig. 10.2, there is no scale on the Y axis because the actual levels of performance on the two variables are in different ranges (mean PPVT-raw scores are from 25–90 in this time period, and mean MLU levels are 3.5–4.8). Two empirical generalizations are of interest here: (a) For both variables, the two groups start at equivalent levels and remain at equivalent

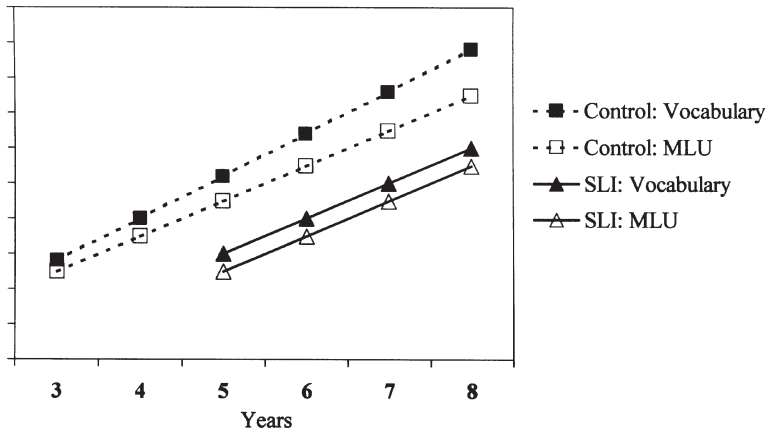


FIG. 10.2. Growth in receptive vocabulary and MLU for children with SLI and unaffected children.

lent levels throughout this 3-year period; and (b) the trajectories of growth for the two variables are highly similar and generally linear in nature.

With regard to the first generalization, recall that the MLU group was selected for equivalence to the affected group on the MLU measure, so it is not surprising that at the initial time of measurement the MLUs are in fact equivalent. The age of the younger group was not controlled beyond the constraint that children below 2;6 were not likely to complete the experimental tasks, thereby excluding very young children at equivalent MLUs. Empirically, the MLU-equivalent group turned out to be almost exactly 2 years younger than the SLI group—an outcome that is replicated repeatedly in my lab and in other investigators' studies.

There is controversy regarding the use of MLU as a matching variable because it is thought to be unstable (cf. Eisenberg, Fersko, & Lundgren, 2001). On the contrary, our outcome data show that it is in fact quite stable. As illustrated in Fig. 10.2, it remains at equivalent levels of performance with the affected group for years. Rice, Rice, and Redmond (2000) and Redmond, Rice, Haney, and Hoffman (under review) reported that at each time of measurement the group means do not differ statistically. Furthermore, the groups do not differ in growth trajectories. Redmond, Rice, Haney, and Hoffman (under review) reported neither group effects nor group interactions in the growth terms of individual growth curve modeling, which shows a strong linear component and a relatively weak quadratic component attributable to a slight leveling effect at the upper levels for the affected group.

Methodological diligence may have contributed to the documentation of stable growth in MLU (cf. Mervis, chap. 8, this volume). A relatively

stringent equivalency criterion was used for grouping (i.e., a candidate child for the MLU group had to obtain MLU-morph within .10 of at least one child in the affected group). The same spontaneous sample elicitation contexts were maintained over time, involving interactive play with age-appropriate toy materials. Examiners were carefully trained to eliminate possible sources of error in data collection (such as too many yes/no questions on the part of examiners; cf. Johnston, 2001). Transcription and coding conventions were explicitly documented and maintained. Intercoder reliability was consistently monitored to minimize coding errors.

Overall, the MLU growth outcomes can be summarized as follows: The SLI group is 2 years behind the expected growth levels in utterance length and remains 2 years behind during the period of 5 to 8 years. Furthermore, although displaced by 2 years, the growth in the affected children follows the same generally linear trajectory as the younger unaffected children.

Turning our attention to semantic growth, another unplanned empirical outcome of the matching design is that the groups' performance on the PPVT-R raw scores at time of entry, as shown in Fig. 10.2, is equivalent and remains so at each time of measurement (Rice, Rice, & Redmond, 2000). Models of individual growth curves (Redmond, Rice, Haney, & Hoffman, under review) differ somewhat from the outcomes for MLU, with a group \times linear interaction such that there is a slight tilt in the growth trajectory for the younger children; they start a bit lower and end a bit higher than the affected children. The major growth elements, however, are linear for both groups.

Taking into consideration the two variables, my working conclusions are: Utterance length and semantic development are in close alignment throughout this age period, and the SLI group is 2 years behind the expected growth levels and remains 2 years behind during the period of 5 to 8 years. Furthermore, although displaced by 2 years, growth in the affected children follows the same generally linear trajectory as the younger unaffected children (with a small adjustment in the slope of the receptive vocabulary growth to show slightly greater growth in the younger group). Thus, growth in the SLI group clearly follows a language delay trajectory in these two important indicators of language growth, with no indication of catch up during this period.

Growth Complexities in Morphosyntax and Morphology. Consideration of other elements of language growth shows that more is involved than a simple delay of an inherently highly synchronized language system. Consider Fig. 10.3, which plots the growth in grammatical tense marking in simple clauses for the two groups in terms of percentage correct use and contrasts growth in tense marking with that in regular plural -s during the same time period. The regular plural affix (e.g., *hats*) has

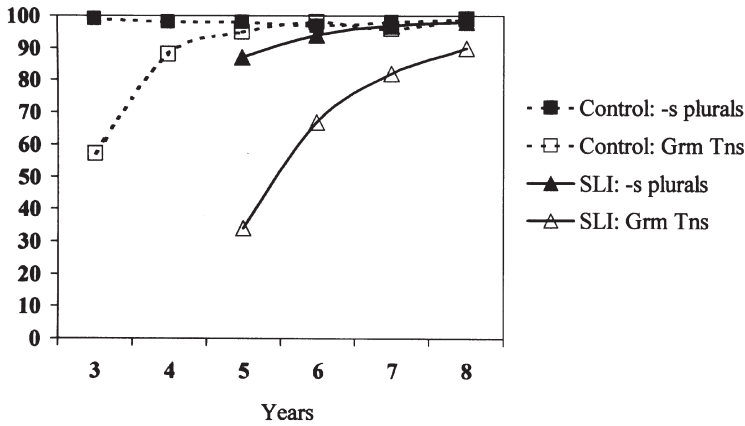


FIG. 10.3. Growth in grammatical tense marking in simple clauses for children with SLI and unaffected children compared with -s plurals.

been of interest because it is phonetically similar to the third-person singular present tense *-s*, and presumably similar learning mechanisms would be involved in acquisition (cf. Leonard, 1998). If growth in the two morphemes followed similar trajectories, it would be consistent with the idea that affected children had problems learning morphological rules for affixation, in general, and/or they had selective difficulties with certain morphemes that could be less salient such as *-s*. Although as an explanation there are many appealing elements of this interpretation, the empirical outcomes are not supportive.

As shown in Fig. 10.3, at the same time that the affected children have mastered plural *-s* (at levels of 90% or above accuracy), they continue to sometimes use finiteness markers in obligatory contexts in simple clauses and sometimes omit them. The variable plotted for grammatical tense marking is the proportion of finiteness markers collapsed across the full set of morphemes that carry out this function (i.e., third-person singular present tense *-s*, past-tense regular and irregular, *BE* and *DO*). The same general growth curve holds for each of the individual morphemes (cf. Rice, Wexler, & Hershberger, 1998), including third-person singular present tense *-s*.

It is clear that the growth of plural *-s* and third-person singular present tense *-s* is not synchronized (although the morphemes marking finiteness are correlated throughout this period). Of course it is possible that growth of plural *-s* for the affected children follows a delay pattern linked with the younger children at an earlier period of development; this delay is resolved by the observed age levels. As suggested by Leonard (1998), there could be a semantic advantage for plural marking, which makes it conceptually useful or salient for a child to master. In this case, plural marking

might follow the general delay pattern associated with utterance length and early semantic development, although the delay would only be detectable at earlier ages. However, the most obvious elements that are shared by the two different *-s* affixes (i.e., similar phonology and learned patterns of affixation) do not account for the markedly different growth pattern for the grammatical tense marker.

It is clear from Fig. 10.3 that, as expected by the Extended Optional Infinitive account, growth of finiteness marking for unaffected English-speaking children is relatively delayed, and it is even more delayed in the SLI group. Of interest here is that the growth trajectories are strikingly similar for the two groups, involving relatively strong quadratic elements with shifts in acceleration.

At the same time, the grammatical marker's growth curve of the affected children lags below that of the younger children and traces lower levels of performance throughout (see Rice, Wexler, & Hershberger, 1998; Rice, Wexler, Marquis, & Hershberger, 2000, for detailed reports of Hierarchical Linear Modeling of Growth). The bends in the curve are not marked by chronological age, but by relative position in the growth curve. It is as if the train left the station late for the affected children, but a particular component was delayed even further; once activated, it followed the same plan for change as the younger children.

This is really quite amazing given that the affected children are considerably older and have experiences that are meaningfully different from the controls. All the affected children were enrolled in intervention at the initial time of measurement and had therefore been identified for special services. Presumably the intervention activities would have greatly enriched a child's language experiences even if the targeted morphemes were not explicit intervention goals. The children were enrolled in kindergarten to third grade during this time, when the younger children were in preschool experiences and lagging behind 2 years in the grades. Affected children are more likely to encounter social rejection from peers (Gertner, Rice, & Hadley, 1994), be somewhat introverted (Redmond & Rice, 2002) or somewhat aggressive (Tomblin, Zhang, Buckwalter, & Catts, 2000), and encounter difficulties learning to read (the mean levels of reading performance for this group of affected children also lag below that of their age comparison group throughout the elementary grades). The children in our study were not drawn from the same schools and attendance centers, but rather were distributed across a wide array of school settings. A wide range of socioeconomic circumstances is represented in the sample. All in all, there are no apparent experiential similarities that can account for the robust mirroring of the unaffected growth pattern in affected children. Instead there would be reason to think that the affected children would either catch up (as an unspecified default outcome) or labor along

in a compensated learning approach to the problem presumably manifested in a mostly linear trajectory, one step at a time. Neither of those plausibly assumed outcomes proved to be true.

It would be misleading to conclude that the disrupted delay evident in growth toward obligatory use of finiteness markers means that the affected children have a generally weak understanding of morphosyntax. At the same time that they persist in omitting the targeted forms, it is rare that they make errors involving misuse of the forms. They are very unlikely to make mistakes like: "tomorrow I am going to walked my dog," "I walks home," "he do wants a cookie," "is he want a cookie?," "where is he want a cookie?," "does they happy?," or "he not is going." Laborious documentation of the children's errors reveals that although errors of this sort occasionally appear, such errors are rare (<1%) given the number of opportunities for error—a generalization that holds for both groups. Grammaticality judgment data provide further evidence of selective grammatical weakness combined with robustness. Although the affected children are likely to accept as okay the statement "he eat toast" (an utterance they are likely to produce), they are likely to reject "I drinks milk" (an utterance they are unlikely to produce; Rice, Wexler, & Redmond, 1999). Just as the train in the acquisition metaphor does not wander off on all possible trajectories, it is as if the children are constrained to a limited set of possibilities. As they are working out grammatical tense marking, their utterances conform to this set of constraints.

The list of linguistic constraints is surprisingly long and substantive. It includes word order, knowledge of finiteness constraints (such as one site per main clause with no more than one occupant of the site), relationships among functional elements (such as the constraint in English that will not allow a lexical verb to move forward to form a question in the way that copular and auxiliary *BE* can move from the clause internal position in a declarative, and instead *DO* must be inserted, but *DO* is not used in questions with predicate adjectives or with the progressive ending on the lexical verb), and knowledge of subject-verb agreement (such as knowing that third-person singular *-s* cannot be applied to subjects carrying first- or second-person or plurals, and the forms of *BE* and *DO* must conform to the person/number markings on the subject). The affected children seem to be as adept in avoiding these kinds of errors as are the unaffected children. It is difficult to measure this kind of growth, but there is obviously a great deal of growth in morphosyntax at the same time that the grammatical tense marker is apparent. To be complete, any proposed models of language impairment in children with SLI must account for not only the selectivity of grammatical tense marking, but also the apparent sparing of related fundamental properties of morphosyntax.

Growth Plateaus Beyond 8 Years. One possibility is that the affected children do eventually catch up to younger children, somewhere beyond 8 years of age. The tasks for the children younger than 8 years have focused on simple declaratives, such as “he is sleepy” or “a dentist fixes teeth.” To explore later growth, we turned our attention to simple questions where finiteness marking appears in the *BE* and *DO* forms that precede the subject (Rice & Wexler, 2000). The EOI model predicts that just as the children regard the use of *BE* copula and auxiliary as optional in declaratives, they will also regard them as optional in questions, where there is the additional requirement of movement to the left of the subject. This prediction was evaluated in judgment tasks, with items such as “what do you like to eat/what you like to eat?” and “what is she saying?/what she saying?” The outcomes are presented in Fig. 10.4 in terms of A' values, which are adjusted for a tendency of children to say “yes” and are roughly interpretable as percentage correct. It is apparent that well before 8 years almost all growth in this judgment has occurred for unaffected children, although the affected children are not at ceiling and instead level off in a plateau some 10 to 15 points lower than the control children—a gap that persists through 14 years of age.

Following the assumption that the question data are an upward extension of the earlier finiteness-marking growth curve, the picture suggests that the affected children may never reach fully robust competencies in this area of the grammar, although they now show many areas of linguistic competency and adherence to subtle linguistic constraints. It is as if the

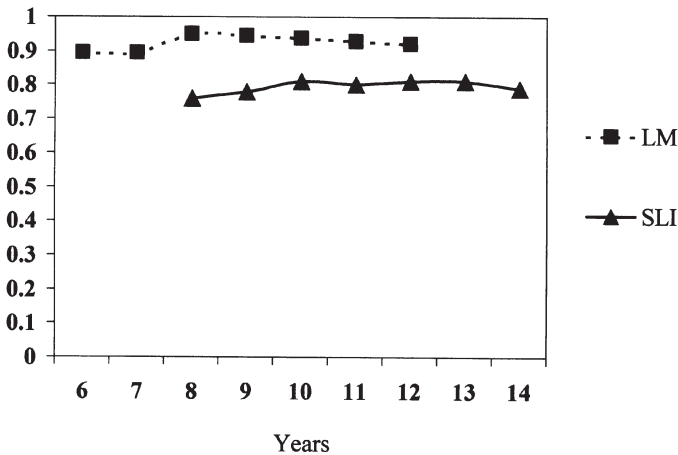


FIG. 10.4. Growth in grammatical tense marking in questions for children with SLI and unaffected language-matched children.

growth mechanisms are programmed to follow the expected track and rate, but the train never quite makes it to the destination.

Growth With Language Impairment Above and Below 85 Nonverbal IQ

As noted earlier, there is great interest in examining the ways in which the condition of SLI is manifest if the exclusionary criteria are relaxed to include children in the borderline level of nonverbal intelligence. Figure 10.5 illustrates data from a recent study by Rice et al. (in press), which followed children epidemiologically ascertained at kindergarten (Tomblin et al., 1997). The children were grouped into categories of: unaffected age controls ($N = 117$ at kindergarten; 24 in the longitudinal sample); SLI, defined as having receptive or expressive language deficits with a criterion of 1.25 standard deviations below the mean ($N = 130$ at kindergarten, 57 longitudinal); nonspecific language impairments (NLI), defined as language impaired with nonverbal intelligence levels below 85 (mostly in the 70–85 range with a few children below 70; $N = 100$ at kindergarten, 54 longitudinal); and low cognition (LC; $N = 73$ at kindergarten, 16 longitudinal), or children who performed below 85 nonverbal IQ but whose language performance was above the criteria for impairment.

The children were assessed annually for 5 years on two picture-elicitation tasks to estimate the grammatical tense marker—one for regular and irregular past tense and the other for third-person singular present tense -s. Figure 10.5 reports the composite of the tasks for each time of measurement. Several things are immediately clear. One is that the growth curve for the SLI group is similar to that of the Kansas sample of Fig. 10.3, suggesting that the pattern of growth is evident across modest

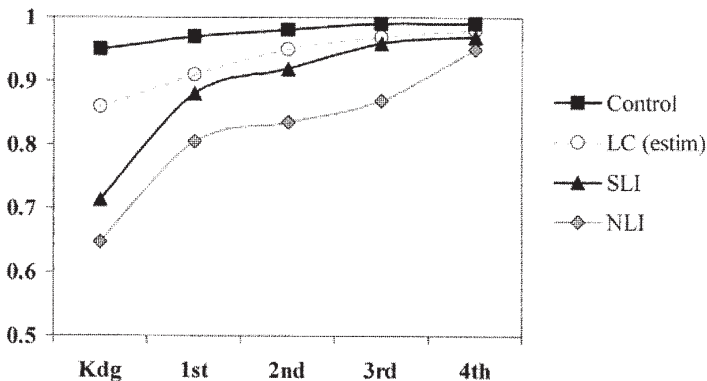


FIG. 10.5. Growth in grammatical tense marking for control, SLI, NLI, and LC groups.

variations in definition of the grouping criteria. Second, as expected, the control group stays at ceiling levels throughout. Third, the NLI children, with lower levels of nonverbal IQ, follow a growth trajectory similar to that of the SLI group; growth curve modeling reveals linear, quadratic, and cubic elements for the composite score for both groups.² Finally, the LC group shows a much higher level of performance. Because so many of the LC children were at or near ceiling on the tasks in kindergarten, only the low-scoring members of the group were followed longitudinally. What is displayed in Fig. 10.5 is a best estimate of the full trajectory for the full group.

These group comparisons suggest that, just as the SLI group presumably starts later and lags below their age peers, the NLI group is even further behind. It is tempting to attribute the lower language performance to the lower nonverbal IQ levels of the NLI group, but the performance of the LC group provides counterevidence to that interpretation. The LC group demonstrates that some children with low nonverbal IQs nevertheless perform well on the grammatical tense marker. Thus, low IQ is neither necessary nor sufficient for language impairment or, more particularly, low performance on the grammatical tense marker (cf. Tager-Flusberg, chap. 3, this volume; Mervis, chap. 8, this volume; Zukowski, chap. 6, this volume). Instead it seems that deficits in grammatical tense marking and low nonverbal IQ are additive independent elements, such that if both are present growth is slower. The NLI group requires 2 years more than the SLI group to come to ceiling levels on the grammatical marker. Yet the way growth unfolds is remarkably similar, with the addition of an apparent acceleration burst around 9 to 10 years of age for the NLI group.

Timing and Possible Maturation Mechanisms

There is a strong impression of maturational mechanisms at work in these observed growth patterns for typical development and in nonsyndromic language impairment (i.e., SLI and NLI). In the age ranges observed, the timing mechanisms differ by linguistic subcomponents, with obvious differences between the general dimensions of growth in utterance length and semantic/vocabulary acquisition, and that of the grammatical marker in the morphosyntax domain. In these areas, timing patterns show invariance once activated, involving a change in acceleration leading to

²At the level of linguistic details, there are indications of differences between the SLI and NLI groups in growth for irregular past tense, with only a linear element for the NLI group and an additional small quadratic element for the SLI group. This suggests some qualitative differences may apply to certain elements of linguistic growth in the NLI group, particularly in the phonological learning needed to master exceptions to phonologically regular morphemes (see Rice et al., under review, for discussion).

relatively rapid growth, followed by a final phase of deceleration to arrive at asymptote. As noted earlier in the chapter, this story requires consideration of onset, configuration of the language system, acceleration, points of change, and slope (rate of change). A full account is not complete without considering the ways in which the linguistic system is robust, especially in the form of subtle but powerful morphosyntactic constraints that block errors and inform judgments.

There are precedents for maturational models of language acquisition, most notably that of Lenneberg (1967), a biological model ahead of its time. Wexler (2003) proposed a contemporary maturational model in which he argued that children are genetically prepared to acquire the configurational properties and associated principles of linguistic structure. He followed a slightly modified full competency model, in which children are presumed to have the same set of fundamental linguistic principles and constraints as adults. This assumption avoids the problem of how a child system later switches over to an adult system (cf. Phillips, chap. 11, this volume). Wexler's model also assumes learning components that are operative to allow a child to deduce properties of the input language at the level of parameters of possible cross-linguistic variation, and with regard to the phonological ways in which a given language represents words and morphemes. His assumptions, and those of other full competency theorists, account for the strong role of constraints in the acquisition of morphosyntax (and other elements of language).

Wexler (2003) proposed that the particular delay in finiteness marking is attributable to a discrete constraint that children follow, the Unique Checking Constraint (UCC), formulated within a generative framework of grammar (Chomsky, 1995). Essentially, the notion is that early in acquisition children employ a single computational operation where two operations are required. Under maturational control, the UCC withers away leaving the two-operation checking procedure of the adult grammar. The UCC and its associated theoretical framework accounts for a variety of linguistic phenomena related to finiteness marking and the ways that this system emerges differently across different languages.³ The UCC model is the topic of lively discussion, debate, and challenge from counterproposals (cf. Borer & Rohrbacher, 2002; Guasti, 2002; Phillips, chap. 11, this volume).

³In languages such as French and Spanish, young unaffected children do not display the protracted period of acquisition that is evident in English, but it appears that French-speaking SLI children do follow a prolonged growth trajectory that makes their development even more behind that of their age peers than English-speaking SLI children (cf. Paradis, Crago, Genesee, & Rice, 2003; see de Villiers, 2003, for discussion of cross-linguistic issues).

Although many of the technical linguistic details are yet to be worked out, a maturational model of this sort accounts for the major findings in the growth curves presented here (i.e., why general elements of language acquisition can show a language delay in children with SLI, but some other elements shear off for a more protracted acquisition period). I have three observations in this regard. First, once activated, growth seems to be invariant in trajectory. Second, robust elements of morphosyntax are evident at the same time that other elements are greatly delayed. Third, a particular element of morphosyntax can be relatively robust in individuals with low levels of nonverbal IQ while it can be combined with a low level of nonverbal IQ to create a double burden in development. These observations align if we assume that children are equipped with powerful language insights that are comprised of differentiable linguistic elements, and such elements can play out according to different onset triggers and growth trajectories. This does not eliminate a role for environmental modulations or general learning mechanisms, but it does suggest that genetically guided language-specific onset and growth mechanisms play a powerful role.

Relevant maturational models also exist in the area of reading disabilities, where growth issues similar to those raised in this chapter have been addressed. A big issue has been whether poor readers eventually catch up in the form of a developmental lag or whether they demonstrate a deficit, which leads to a plateau level below age expectations from which they do not recover. Francis et al. (1996) studied this question with longitudinal reading outcomes data. With growth curves not greatly different from that of the grammatical tense marker illustrated earlier, they concluded that the developmental deficit model held, characterized by quadratic growth, to a plateau. They operationalized the difference between the two models as follows:

the notion of developmental lag could be operationalized as a difference in the age at which two groups reach their final level of performance. In contrast, the notion of developmental deficit was operationalized as a difference in the final level of performance and may or may not be associated with any lag in development. (p. 14)

In analyses of individual growth curves assuming a start point of 8 years, they noted that there were no differences between the groups in the age at which scores reached a plateau thereby supporting the developmental deficit model. In contrast, the grammatical tense growth curves reported here show strong indications of developmental lag prior to 8 years, and probable developmental deficit beyond 8 years of age. Francis et al. (1996) called for the incorporation of longitudinal assessments into the diagnos-

tic process for identifying children with poor reading skills—a suggestion I return to later.

Of course growth in reading and in the morphosyntax of language have obvious differences in that reading is explicitly taught in formal educational settings, whereas morphosyntax is spontaneously acquired by young children without explicit instruction. Reading impairments have known genetic contributions (cf. Smith, chap. 13, this volume), and reading impairments are also known to be closely associated with language impairments. As suggested by Bartlett et al. (2002), perhaps SLI and dyslexia share the same gene loci as indicated in the region of 13q (see Smith, chap. 13, this volume). If particular genes affect both phenotypes, perhaps these genes are also involved in the timing mechanisms that the phenotypes share.

GROWTH TRAJECTORIES FOR LANGUAGE IMPAIRMENTS ACROSS CLINICAL CONDITIONS: FUTURE DIRECTIONS FOR RESEARCH

These examples of language growth in children with SLI and NLI point toward possibly fruitful approaches to determine the ways in which language impairment can be manifest across a wide range of clinical conditions. What we should aim for is an enhanced understanding of the exact nature of language impairment, in the form of knowing about the elements of language growth that can be compromised, and the pattern of growth. As the chapters in this volume attest, there is no doubt that various elements of language can be spared while others are affected. What we need to know is the way the linguistic system comes on-line, the extent to which the “linguistic train” components are well synchronized, and the expected end state relative to the adult grammar.

The observations of developmental growth and linguistic issues raised in this chapter suggest a series of questions to be answered about the way that language impairment is manifest across different clinical conditions and if the symptoms hold across individuals within a particular diagnostic category. These questions lay out as follows:

1. Is onset delayed?
2. Is speech/phonology affected?
3. Is vocabulary growth in line with age expectations or at lower levels?
4. Do nonfiniteness-marking morphemes appear when expected, such as plurals and progressives?

5. Does the grammatical tense marker appear when expected?
6. Are there signs of disrupted grammatical systems, such as errors of word order, errors of subject–verb agreement, or errors of form choice (such as confusion of *BE* and *DO*)?
7. Is growth following the expected trajectory for given elements of the language system?
8. Does growth plateau before expected levels of acquisition?
9. Is growth synchronized across elements of language, or are there unexpected areas of growth delay or disruption?
10. Are the predictors of growth as expected?
11. Does intervention lead to a change in acceleration of growth in a given domain?

This list is not meant to be exhaustive, but answers to these questions allow us to:

1. Confirm that a delayed onset is characteristic of language impairment in general, although a delayed onset is not diagnostic of subsequent language impairment (as shown by the Late Talker outcomes). Of course it is important that measures of delayed onset are obtained at the time of the delay. As noted by Lord et al. (chap. 2, this volume), retrospective reports by parents are subject to error.

2. Determine the co-morbidity of language and speech impairments at the time that language emerges. This information is much needed for the early period, across clinical conditions, to further sort out the speech and language phenotypes (cf. the KE family and the work on *FOXP2*).

3. Determine whether a general language delay is the default condition for language impairments. This is at least a plausible first hypothesis that entails the assumption that the language acquisition mechanisms are robust in human children, but they can be slow in activation.

4. Determine whether there are particular weaknesses or strengths in vocabulary development, general morphology, and/or grammatical tense marking (or other linguistic properties). An EOI period could be characteristic of some, but not all, forms of language impairment, or it could be characteristic of relatively mild forms of language impairment such as SLI, in which the semantic and general morphological components of language are developing but the grammatical tense-marking system is much more delayed. At the same time, it is clearly the case that in some forms of language impairment, such as Williams syndrome, the morphosyntactic system is relatively robust.

5. Determine whether there is a disrupted grammatical system as manifest by unexpected grammatical errors. Such errors imply that the under-

lying linguistic constraints and principles are affected. Fundamental disruptions to grammatical systems are of considerable theoretical interest, although seldom documented (cf. van der Lely, 2003, and the diagnosis of a grammatical subtype of SLI characterized by a representational deficit for dependent relations in a selected sample of adolescents).

6. Determine whether linguistic growth is well synchronized and the pattern of growth for different elements of the linguistic system. If growth is flat or decelerating to earlier levels, this would be a red flag for serious concerns about neurocortical integrity because it is a profound violation of the powerful growth mechanisms at work in language acquisition (cf. Lord et al., chap. 2, this volume). Further study is needed to confirm the growth patterns reported in this chapter and the apparent differences in growth trajectories across the different linguistic domains. To echo the suggestion of Francis et al. (1996), growth indexes would be powerful identifiers of language impairment. We need to know whether children with a given clinical condition are likely to start late and then show a strong acceleration, as seems to be the case for children with Williams syndrome, or if they start late and instead follow a slow rate of change without subsequent changes in acceleration—a profile with a guarded prognosis for arriving at the adult grammar. We need to know what is triggering the points of change (i.e., the biochemical, neurocortical, or environmental events associated with these points of change). Without detailed longitudinal records we will not be able to identify these crucially important components of language impairment and possible ways to reset delayed or defective growth.

7. Determine the ways in which intervention leads to an acceleration of linguistic growth and optimal timing for targeted intervention. Knowledge of the expected growth trajectory in various areas of language are immensely helpful in planning for intervention. The fact that grammatical tense marking is a relatively late acquisition in English-speaking children, and is even more protracted in children with SLI, means that it is probably not an appropriate early goal for language intervention in young children with language impairments. A fruitful intervention strategy would be to look for maturational change points, the time at which the obligatory use begins to increase, and then build on this point of change to further enhance acquisition.

Implications for Genetics of Language Impairments

The chapter opened with reference to current studies of genetically controlled timing mechanisms at the cellular level as well as for organs such as eye structure and even entire organisms. Although the maturational mechanisms advocated by Lenneberg (1967) seemed rather far-fetched at

the time, the existence of such mechanisms is now well attested by modern genetics. Marcus and Fisher (2003) discussed how the *FOXP2* gene regulates a wide range of cellular development and is one element of a complex pathway involving multiple genes that, among other things, controls the development of brain structures. They described a gene with pervasive developmental effects that is linked to a relatively specific phenotype of higher order cognitive functioning in the form of speech and language impairment. Marcus and Fisher speculated that the consequences of disruption to a gene are expressed in separate systems, and the gene provides “an entry-point into the relevant neural pathway (or pathways), by pointing to the downstream targets which it regulates or the proteins with which it interacts” (p. 262).

As shown by Bailey et al. (chap. 7, this volume), investigation of the genetics of some of the clinical conditions associated with language impairment, such as fragile X, is already focused on the proteomics of genetic expression. It is not at all far-fetched to imagine that within the foreseeable future more will be known about regulatory genes and protein expression in various conditions of language impairment.

Although specification of the phenotype is important in determining genetic effects—a specific phenotype does not entail a discrete gene/phenotype connection. Just as the genetic contributions to reading impairments are thought to be at the level of underlying neurocognitive structures essential for reading skill, so the genetic contributions to particular linguistic elements, such as grammatical tense marking, surely are at more abstract levels than a child’s knowledge of when to say *walked* instead of *walk*. Instead the omitted finiteness markers are thought to indicate fundamental properties of underlying linguistic representations consistent with universal properties of linguistic structures and principles, which in turn are expressed in neurocognitive pathways (cf. Rice, 1996b; Wexler, 1996; Phillips, chap. 11, this volume).

Inclusion of growth indicators in the phenotype of language impairment, and evaluation of possible phenotype/genotype correlations for different growth patterns, would be useful for adding greater precision to estimates of genetic contributions and for inferring time-controlled gene expression that may operate across different elements of language in different ways. In turn, this would greatly enhance our understanding of environmental influences, the points in development when such influences might be most powerful, and how to design intervention programs to make maximal use of developmental trajectory. The search for this information will be greatly facilitated by cooperative study across the wide range of clinical conditions in which language impairments appear, just as the benefits of improved intervention strategies would be applicable to a wide range of affected children.

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