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1. Introduction

This symposium has, from the very beginning, had an emphasis on the "development" part of children's language. In keeping with this tradition, I, too, will focus on the developmental dimension of children's language. My talk is going to pick up on several of the themes from keynote/lecture talks. In 1995 Lydia White (1996) demonstrated how second language acquisition research has come of age, in terms of theoretical significance for linguistics. In 1997 Annette Karmiloff-Smith (1998) concluded that atypical language development is not necessarily a window on normal language acquisition. In 1999 Elissa Newport (Newport & Aslin, 2000) described a program of research intended to clarify the role of statistical learning in language acquisition and how innate components of language acquisition may arise from computational constraints. In 2000 Lois Bloom (2001) emphasized the effortful nature of children's language acquisition, the value of performance data, and language as an interrelated part of children's development. In the same year, Nina Hyams (2001) examined optionality in child grammars and argued that optionality is a product of the interface of semantics and inflectional classes.

I borrow from Lydia the notion that the area of research I will report on has "come of age" in the sense of moving from an unnoticed area of investigation to one with theoretical relevance for students of linguistics and children's language development. It is obvious in the topics on the program here, and in previous meetings of the BUCLD, that studies of children with specific language impairment (SLI) are recognized as theoretically substantive. In many ways the BUCLD community of scholars was at the forefront of recognizing the significance of the contemporary studies of children with SLI, and for that I am most appreciative. I especially want to recognize the important contributions of my colleague, Ken Wexler, to the program of investigation that I will be describing.

My conclusion of relevance is opposite that of Karmiloff-Smith—as I will describe, the parallels between unaffected children and children with SLI are powerful and highly informative to our understanding of the wayschildren's language changes over time.

Like Elissa Newport, I am involved in a program of investigation that is largely empirical, in the early stages of a long enterprise, and fundamentally grounded in questions about the role of innate language acquisition mechanisms. More specifically, the program of investigation aims to clarify why some children have significantly less aptitude than expected for acquisition of their native language, and if those differences are related to the robustness of unaffected children's language development. I share Lois Bloom's sense that language acquisition can be effortful, but from my perspective the extended efforts of children with SLI make the efforts of unaffected children seem relatively minor. Furthermore, "effort" is intrinsically linked to timing mechanisms—more effortful acquisition requires longer time—and we must understand change over time in order to sort out relative amounts of effort. I also share Dr. Bloom's appreciation of performance data, and will, I hope, demonstrate that the growth story from performance data can be very informative. Also, I will highlight that language acquisition can be fundamentally out of sync with the rest of a child's development, much to the child's and parents' dismay.

Finally, Nina Hyam's investigations of morphosyntax and the phenomenon of optionality are classic examples of the value of focusing on linguistic details within and across languages, the centrality of notions of finiteness in current models of clause structure, and the perplexing nature of the optional and obligatory features of clause structure. In short, theoretical models of linguistics are proving to be crucially important in helping us develop more precise measurement systems to better understand patterns of individual difference and growth in children's language.

1.1 Growth as a Biological Phenomenon—the Genetics Perspective

My topic here is language growth. In the modern study of genetics, growth is a defining biological phenomenon. This is expressed eloquently in a research issue of Science:

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, 2000)
Although the biological perspective can seem to be far from linguistic notions such as finiteness, the gap is narrowing as early genetic discoveries appear and as we consider the timing mechanisms inherent in linguistic growth. Questions can now be framed as phenotype/genotype relationships, and the need to understand growth in these terms. The ultimate question is how to account for varying amounts of individual aptitude for native language acquisition. The story of how language grows over time is proving to be a crucial part of the picture if we are to ultimately understand the ways that genes and environments interact in language acquisition.

1.2 Preview

In this paper I report the longitudinal outcomes of language acquisition of a group of children with SLI and unaffected control children. I begin with general characterizations of the condition of SLI and a brief review of recent genetic findings. Next I describe longitudinal growth outcomes of children with SLI and comparison groups of unaffected children, to demonstrate the following: 1) The condition of SLI is a model system for the description of timing effects; 2) Timing mechanisms can vary across different elements of the linguistic system; 3) Full explanations of timing must consider onset timing, configuration of the linguistic system with delineated subcomponents, acceleration rate, and points of change in the acceleration; and 4) Faulty timing mechanisms are implicated in SLI. I conclude with implications for models of language acquisition/impairment and studies of the genetics of language impairments.

2. SLI as a Condition of Language Delay or Language Disruption

The condition of SLI is defined relative to age expectations for language, cognitive, and social development. The diagnosis is determined by inclusionary and exclusionary criteria. The inclusionary criterion is that a child’s language development is below what is expected for his or her age level. This is conventionally set at 1 or more standard deviation below the mean for age level, and conventionally is determined by an omnibus assessment instrument that collapses across multiple dimensions of language. The exclusionary criteria rule out mental retardation, hearing impairment, and clinically significant neurological impairment. Traditionally, children with a diagnosis of autism also would be excluded from samples of children with SLI, although the overlap in these conditions is a matter of current inquiry (Tager-Flusberg, in press; Roberts, Rice & Tager-Flusberg, in press).

Much of the literature has tried to determine the nature of the children’s language impairment (cf. Leonard, 1998). At a global level of language acquisition, it is possible that the youngsters show a simple language delay. Under this model, the age-referenced deficit is attributed to a late start of a synchronized language system. Essentially, the notion is that a 5-year-old child with SLI will have the language system of a younger child. This often also brings the assumption that the child will “outgrow” the problem. This is not entirely implausible, as the evidence suggests that perhaps 75% of children who are slow at first word acquisition subsequently “catch up” with other children. Note that this is in fact a complex growth pattern, such that there is a slow onset, followed by accelerated acquisition that is somehow reset to the expected rate. If the rate is not reset the children would shoot up and pass their age peers, and there is no indication that happens. Elsewhere I suggest that a language delay may be the default form that unifies the symptoms of language impairment across a number of clinical conditions (Rice, 2003, in press).

Another possible form of language impairment is that of language disruption. In this form, the overall momentum is forward, but certain elements are slowed, thereby disrupting the overall harmony. When compared to children of the same general language level, localized elements of the language system can be at even lower levels than predicted by a fully synchronized language system. This could be because coupling or computational relations between linguistic elements are not as expected. Perhaps the disrupted element is never fully resolved and remains out of sync for protracted periods.

Finally, it is possible to have a combination of Language Delay and Disruption, such that the overall language system is slow to emerge and follows a slower growth pattern than expected, and some elements of the linguistic system are even more greatly delayed. Although it is possible to predict general features of the linguistic system by adjusting to lower age expectations, it is not possible to predict certain elements from the other elements in the system according to the expected relationships of a fully integrated linguistic system.

The identification of disrupted elements of linguistic growth has been a long-sought empirical outcome of studies of children with SLI. Under the guidance of current theoretical models of children’s grammar, such elements have been identified and are proving to be valuable as clinical markers of affectedness. But before moving to the empirical evidence, a few more generalizations about SLI must be established.

2.2 SLI as a Condition of Language Impairment with Low Risk for Speech Impairment

Although it was long assumed that children with language impairments were very likely to have speech impairments as well, a recent epidemiological study of kindergarten children establishes that the likelihood of speech impairments with language impairments is actually very low. Instead there is minimal overlap of the two forms of impairment. It is estimated that by 3 to 6 years of age, only 5-8% of children with SLI have clinically significant speech problems (Shriberg, Tomblin, & McSweeney, 1999). This suggests a significant degree of orthogonality in the two interrelated systems of speech production and language competence and their respective timing mechanisms. It is important to
note that clinical caseloads are weighted toward children who have significant speech problems. This poses a possible methodological problem for studies of morphosyntax, such that it is important to ensure that SLI participants ascertained from clinical caseloads do not have speech deficits that can interfere with morphological productions.

3. Genetics of SLI

A growing body of evidence establishes the very strong likelihood of genetic contributions to the condition of SLI. Definitive linkage studies do not yet exist, but the emerging evidence is pointing consistently toward the strong probability that eventually such linkages will be discovered although they may well be complicated by multiple gene effects or interactions. Four kinds of evidence point in that direction. One is familial aggregation for SLI probands. A reasonable working estimate now is about 25% of nuclear family members of affected children are likely to be affected, as compared to population estimates of 5-7% (cf. Rice, Haney & Wexler, 1998; Tallal, et al., 2001). Second, twin studies establish heritability for SLI. A recent study (Adams & Bishop, 2002) shows heritability of grammatical tense-marking deficit in 6-year-old twins. I will return to this symptom in more detail later on. Third, positive linkage outcomes are established for reading disorders that are likely to be related to SLI; for example, there is documented linkage of dyslexia to 13q21 and 6p21 (cf. Smith, in press). Fourth, Anthony Monaco, Simon Fisher and colleagues identified the FOXP2 gene on Chromosome 7 as linked to the speech and language symptoms of one extended family known as “KE” (Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001), but this linkage has not been replicated in other samples of SLI (cf. SLI Consortium, 2002). The affected family members have distinctive speech as well as language impairments, and the problems apparently arose from a mutation in the grandfather’s DNA. Fisher and colleagues (Fisher, Lai, & Monaco, 2003; Marcus & Fisher, 2003) argue that this mutation is probably not among the common risk alleles for SLI; the most common form of SLI is probably not linked to this site. Perhaps of most import for future genetic models of SLI are the regulatory properties of FOXP2. This gene is part of a family of genes that control the operation of other genes that operate “down-stream,” such that the mechanisms of FOXP2 “turn on” or “block” the operation of other genes at subsequent points in time. That is to say that the operation of the regulatory gene influences the behavior of other genes and this influence is apparent after elapsed time. In effect, the gene is about timing as well as expression of directions. One of the “downstream” effects of FOXP2 is the development of level 6, or the innermost, level of the cortex. The gene is also implicated in the development of cerebellar structures, as well as lung and liver tissue.

Thus, FOXP2 is not the “speech gene” or the “language gene” in the sense of a single gene that has a direct effect on speech or language. Instead, it provides a glimpse into the exquisite timing mechanisms and interrelatedness of genetically guided development. The gap between such mechanisms and higher cognitive functions such as language is surely wide, but we can begin to see how the timing elements may be related, even if mediated by a long string of intervening events.

4. Language Patterns of SLI
4.1 Evidence of Growth: Classic Protracted Delay of MLU and Receptive Vocabulary

Let us turn now to evidence of growth of language in children affected with SLI as compared to unaffected children. The experimental design includes two control groups, one of children of the same age and another of children matched for MLU as a general index of language development. The affected children meet the conventional inclusionary and exclusionary criteria, and are screened for speech production sufficient for the target morphological contrasts. In these studies and in the investigations of other researchers, for affected children in the age range of 4 to 8 years, the MLU matched children are 2 years younger, thereby establishing a 2-year offset in language growth for the affected children. The representative data displays reported here show age-related levels of performance for affected and unaffected children. The empirical evidence is replicated across more than 125 SLI children for whom there are longitudinal data, about 85 controls, and up to 10 times of measurement per child. Corroborative evidence is available in the normative data for the Rice/Wexler Test of Early Grammatical Impairment (Rice & Wexler, 2001), with cross-sectional evidence from about 600 affected children and 550 controls, ages 3-9 years.

Let us begin with the way that MLU and Receptive Vocabulary performance changes over time. Consider Figure 1. Note the boxes for unaffected children, and the triangles for affected children. Between the ages of 3 and 8 years, unaffected children gain steadily in vocabulary performance, as measured by the Peabody Picture Vocabulary Test, a test that requires pointing to pictures and is a psychometrically robust way to measure vocabulary growth. For both groups, growth is linear; the SLI group is at levels of performance commensurate with children two years younger throughout this period, but their growth trajectory mirrors that of the unaffected children. Now consider growth in MLU, where the control group is indicated by an open square and the affected children by an open triangle. It is obvious that MLU tracks beside vocabulary development during this period for each of the groups, and the affected children are about two years behind the controls on both indexes. In fact, there is a statistically significant positive correlation between the two measures during this period in the range of about .4. This fits the classic protracted delay model—children with SLI start later, with the language system of younger unaffected children. Yet the slopes of the children’s growth suggest that the children with SLI are not likely to catch up. As long as they follow the rates and trajectories of younger children, they maintain the gap between actual and expected growth parameters.
performance. There is no adjustment of acceleration for “catch up” but instead the acceleration remains faithful to the expected rate.

![Graph showing growth in receptive vocabulary and MLU for children with SLI and unaffected children.](image)

**Figure 1: Growth in receptive vocabulary and MLU for children with SLI and unaffected children**

Keep in mind that although the SLI group performs like the younger children, their life experiences, and their social and cognitive development, are indexed by their chronological age. That is to say that they are not actually younger children; they are in most ways more like their age peers than their language peers. Also, the affected children in the Kansas samples and in the Rice/Wexler test (TEGI) norms are drawn from clinical caseloads, so these youngsters have experienced intensive language tutorial experiences that are intended to enrich their language experiences beyond age expectations.

4.2 Evidence of Disrupted Growth: Grammatical Tense as a Clinical Marker

Not all elements of language follow the linear trajectories of growth shown by the vocabulary and MLU data. Some required elements of clausal structure in the morphosyntactic domain do not grow in a linear way. This outcome emerged from our interest in finiteness marking in affected children that was motivated by theoretical models of language acquisition that in turn drew upon new models of adult clausal representations.

A number of scholars contributed to theoretical breakthroughs that are relevant to the data I will describe, and it is not my intent here to evaluate the goodness of fit of the data for opposing theoretical accounts. Instead I wish to emphasize how this general view of morphosyntax, and its focus on linguistic particulars, has contributed substantially to our understanding of what is happening in the grammars of unaffected children and children with SLI and how they develop.

This work was informed by Ken Wexler’s model of Optional Infinitives (Wexler, 1994; 1996; 2003), and several key constructs advocated by him (and later extended to the Agr/Infl Omission Model (ATOM) in collaboration with Carson Schütze (Schütze & Wexler, 1996; Wexler, Schütze & Rice, 1998). Wexler argued that the documented root infinitive or optional infinitive stage for some languages with overt infinitival morphemes was evident in English speaking children in the omission of the following set of verbal morphemes: Third person singular present tense -s, past tense -ed, copula and auxiliary BE and auxiliary DO. Examples follow, where a possible omission is indicated by parentheses:

- Patsy paint(s) the fence.
- Patsy painted(ed) her house.
- Patsy (is) painting the house.
- Patsy (is) happy.
- (Does) Patsy paint other things?

Wexler argued that unaffected English-speaking children show a protracted period of Optional Infinitives. At this time it was known that children with SLI experienced problems with verbal morphology, including work carried out in my lab, but the full import of these problems was not well understood and such problems were generally attributed to problems learning affixes or small unstressed parts of the morphological system. The morphosyntactic models, such as Optional Infinitives, allowed for better understanding of the underlying continuities between the end state adult clausal grammar and children’s incomplete clauses, generated more precise predictions of relationships among affected morphemes and generated more precise predictions of the relationship between morphology and syntax. The Optional Infinitive model was well suited to the possibility that what was happening with affected children was the protraction of a natural area of delay in unaffected children for an even greater period of time in affected children. The notion of an “Extended Optional Infinitive” stage was formulated in late 1990. In effect, from the outset this has been a developmental model, with the prediction that consistency of finiteness marking will be out of sync with the rest of the language system for affected children, combined with the assumption that the same general language

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1. Irregular past tense conforms to the pattern as well, with bare stems for irregular past forms (cf. Rice, Wexler, Marquis, & Hershtberger, 2000).
acquisition mechanisms are available to affected as well as unaffected children. From this perspective, the locus of impairment is not generally about surface phonology, nor is it about stem + affix learning, although obviously those systems are essential parts of the emerging morphosyntactic system. For descriptive purposes, we have referred to the set of finiteness morphemes as a grammatical tense composite, and the predicted lower performance of the affected group as a grammatical tense marker of language impairments. The findings reported here are drawn from Rice, 2003; Rice, Wexler & Cleave, 1995; Rice & Wexler, 1996; Rice, Wexler & Hershberger, 1998; and Rice, Wexler, Marquis, & Hershberger, 2000).

Let us consider the evidence from a developmental perspective. Figure 2 pulls together key pieces of the developmental outcomes. Consider first the open boxes (control children) and open triangles (affected children). The variable here can be interpreted as the growth in percentage correct use in obligatory contexts of third person present singular −s, or the growth in a composite measure of percentage correct in obligatory contexts of the entire set of finiteness-marking morphemes. Within this set of morphemes the growth trajectories are highly similar. As expected, although there are grammatical and empirically sound, as well as theoretically motivated, to consider the depicted trajectories as finiteness marking and this is the underlying common property that links the surface morphology. Several features of the growth of finiteness are strikingly different from the growth in MLU and receptive vocabulary. First, it has nonlinear as well as linear components—it includes a definite change in acceleration, a quadratic component as well as linear. Second, the affected children perform at lower levels of performance throughout the period of measurement. They are more than two years behind in this domain. Yet the growth trajectories of the affected children mirror those of the younger unaffected children. It is as if once the system begins to grow, it is very likely to grow in the same way, even if the onset is delayed relative to the onset of other dimensions of language. The points of acceleration changes are benchmarked to the same amount of elapsed time for the affected children as well as the unaffected children, although these points of change are at different ages and different levels of MLU/receptive vocabulary, and certainly at different levels of accumulated life experiences. Also note that the affected children seem to asymptote at levels lower than those of the unaffected children, suggesting that perhaps they never fully master finiteness marking.

Now consider the filled boxes and filled triangles. This is the level of performance on plural −s during this same time period. Plural −s is an affix with similar surface properties to third person singular present tense, and has been widely used as a comparison morpheme for evaluating the extent to which morphological delays of children with SLI are attributable to more general problems with affix learning that could be related to incomplete or inefficient processing of incoming speech. Note that the affected group is near ceiling performance on this morpheme throughout this period, indicating that they can learn phonologically similar morphemes at the same time that finiteness acquisition is weak.

4.3 Nonpredictors of Growth in Grammatical Tense Marking in the Younger Children and Children with SLI

In a series of studies we have investigated possible predictors of growth in grammatical tense marking, using three predictor variables: Children's nonverbal IQ, levels of receptive vocabulary, and mother's education (as an index of the richness of linguistic input in the home). Outcomes repeatedly show that not one of these variables predicts change over time for this part of the grammar, findings that have been replicated by other labs. The lack of prediction is true for the affected children as well as the unaffected control. More precisely, one cannot use a child's general cognitive level, or general levels of receptive vocabulary, or mother's education, at the onset of these times of measurement to predict which children will show fast growth and which ones will show slow growth. It seems instead that this part of the grammatical system is following a design for growth that is not fully synchronized with other dimensions of development, nor is it the case that th
attributes that a mother with higher levels of education brings to child rearing are instrumental in accelerating growth in this part of the grammar.

What accounts for the similarity in growth between the 2-year-old affected children and the younger unaffected children? This is indeed rather puzzling, insofar as there are many apparent differences between the affected children and the unaffected children of younger ages. The affected children have been in treatment (although many drop out over time). The affected children are older and therefore have different accumulated life experiences and social and cognitive development relative to the younger children. Finally, the children are drawn from a wide array of school attendance center and home circumstances.

The overall impression is that very robust timing mechanisms are at work, programmed to unfold in time-locked ways once activated. In terms of delayed onset, growth takes one track for MLU and receptive vocabulary—a steady increase in levels—and a different track for grammatical tense marking—with sharp shifts in acceleration that are adjusted for a delayed onset, i.e., points of change that are indexed to elapsed time from onset not accumulated years of experience with the grammatical system. To return to the earlier descriptions of the nature of the language impairment of children with SLI, it is clear that both delay and disruption are involved, although in different dimensions of the language system.

4.4 Areas of Robust Growth in Morphosyntax for Children with SLI

One possibility is that the observed weakness in the grammatical tense marker is but one symptom of a more pervasive weakness in the morphosyntactic system of affected children. Omissions of functional material, such as the grammatical tense marker, are sometimes interpreted as attributable to missing or deficient functional structure in the underlying grammar, or to faulty learning mechanisms. Crucial to such interpretations are the distribution of observed errors or limited levels of performance on related grammatical tasks.

In a large amount of detailed empirical data collection, at the same time that affected children persist in omitting finiteness markers, they are very unlikely to commit overt errors in use of the forms. Errors such as the following are rare:

*I walks home
*he do wants a cookie
*is he want a cookie?
*why is he want a cookie?
*does they happy?
*he not is going
*tomorrow I am going to washed my dog
*he is happy, does he?

The avoidance of errors such as these indicate that the affected children know a great deal about the sites in clauses where finiteness is expressed, the constraint on multiple marking of finiteness in a single clause, the need for subject/verb agreement marking and the associated spell out rules, and the uses of BE and DO form classes.

Furthermore, 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 very unlikely to produce) (Rice, Wexler, & Redmond, 1999)

It is as if the affected children do indeed have full access to most of the language acquisition mechanisms of unaffected children, including syntactic constraints on morphological forms, the need for subject/verb agreement marking and the distributional requirements of correct form use, and the complex ways in which BE and DO trade off finiteness marking in various clausal contexts.

Empirically it is difficult to describe nearly errorless growth, but presumably the affected children's avoidance of errors such as these is also indexed to onset time, in the same way as younger children. Unfortunately, carefully documented prospective growth of very young affected children is not yet available in order to determine if this prediction holds.

4.5 Lower Asymptote of Growth in Affected Children

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 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. Just as the children regard the use of BE copula and auxiliary as optional in declaratives, they should also regard them as optional in questions, and this should apply to DO auxiliary forms in questions also inserted to carry finiteness marking. 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 the saying/*what she saying?” Data to age 11 years was reported in Rice and Wexler (2000). The growth outcomes to age 14 years are presented in Figure 3.

In this figure, A' values are plotted, which are adjusted for a tendency of children to say “yes” and are roughly interpretable as percentage correct on a choice forced alternative task configuration. It is apparent that well before 8 years of age almost all growth in this judgment has occurred for unaffected children, although the affected children are not at ceiling and instead level off at a plateau some 10-15 points lower than the control children, a gap that persists through 14 years of age. Following the assumption that the question data is an upward extension of the earlier finiteness-marking growth curve, the picture suggests that the affected children may never reach fully robust competencies.
this area of the grammar, even though they now show many areas of linguistic competency and adherence to subtle linguistic constraints. It is as if the growth mechanisms are programmed to follow the expected trajectory, but the end state grammar still carries some of the weakness of the earlier stages.

![Figure 3: Growth in grammatical tense marking in questions for children with SLI and control children](image)

4.6 Grammatical Tense Marking Independent of Nonverbal IQ: Further Evidence

Among some investigators there is a strong assumption that grammatical growth and levels of nonverbal intelligence are intrinsically aligned, notwithstanding the earlier evidence that nonverbal IQ levels do not predict growth in grammatical tense marking. Recent evidence from a large epidemiological study provides further details about this issue (Rice, Tomblin, Hoffman, Richman, Marquis, in press). This evidence is drawn from a large-scale study based at the lab of Bruce Tomblin at the University of Iowa. It is an important sample of children with language impairments because these children were not identified from clinical case loads, but instead were identified in a large scale program of testing kindergarten children. Measures of nonverbal IQ were collected, as well as general language measures. Children were grouped into the following categories: Unaffected age controls (language and nonverbal levels as expected for age); SLI; Nonspecific Language Impairments (NLI) (language impaired with nonverbal intelligence levels below 85); Low Cognition (LC) (children who performed below 85 nonverbal IQ but did not meet the criteria for language impairment).

The children were assessed annually for five 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 4 reports the composite of the tasks for each time of measurement. Several things are immediately clear. One is that, as expected, the control group stays at ceiling levels throughout. Second, the growth curve for the SLI group is very similar to that of the Kansas sample of Figure 2. This is important because it shows that the pattern of growth is generalizable to the common form of SLI, whether or not the children are clinically identified. Third, the NLI children, with lower levels of nonverbal IQ, follow a growth trajectory very similar to that of the SLI group; growth curve modeling reveals linear, quadratic, and cubic elements for the composite score for both groups. Finally, the LC group shows a much higher level of performance. In fact, the children in this group were at such high levels of performance at kindergarten that their growth had already reached ceiling levels. The growth line in the figure is estimated for the lower ones in this group.

So we see that the conclusions for the role of nonverbal IQ require consideration of both the NLI and the LC groups. The conclusion is that a low level of nonverbal IQ is neither necessary nor sufficient for language impairment, or, more particularly, low performance on the grammatical tense marker. 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 two 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 9-10 years of age for the NLI group.

4.7 Delayed Growth in French-speaking Children with SLI: Late Mastery of Grammatical Tense in a Highly Inflected Language

The growth outcomes for English-speaking children with SLI cause one to wonder if similar outcomes hold for languages that differ from English. Although this is a very important question, there is very little available evidence to describe long-term growth. A team of Canadian scholars, Johanne Paradis, Martha Crago, and Fred Genesee, are conducting an interesting program of study to examine how the grammatical marker færes in French-speaking children with SLI (Paradis, Crago, Genesee, & Rice, 2003). Early rounds of evidence show some surprising outcomes. From what we know of the normative literature, young French-speaking children early on use finite functional projections in their use of finite lexical verb forms and their use of copula être, être/avoir auxiliaries in passé composé, aller auxilary in future proche. This use is presumed to be near ceiling for percentage correct use at age 3 years, in contrast to the much lower percentage for English-speaking children at that age. We know that the growth for English-speaking children is between 3 and 5 years of age.

The findings from French-speaking children with SLI at 7 years of age indicate that the affected children reach, on average, only about 75% accuracy, a level very similar to that of English-speaking children with SLI. Furthermore, this pattern holds in bilingual English/French speaking children as well as in monolingual children. That is to say that the child with SLI who speaks both languages shows striking similarity in the likelihood of using grammatical tense marking in French as well as English, although the elapsed time from expected mastery is much greater in French than English.

This is a very interesting outcome because it suggests that perhaps the growth mechanisms are referenced to age across different languages. Another possibility is that the lower level at 7 years of age for French is accounted for by a lower asymptote, as seems to be true for the English-speaking children. In this case, the earlier growth trajectories may mirror the younger children, with an offset of several years. This is an empirical question that requires further evidence to help us sort out the story.

4.8 Time-Referenced Growth as Maturation?

A number of theorists have argued for maturational mechanisms that guide language acquisition. Eric Lenneberg (1967), perhaps ahead of his time, argued for such mechanisms. Elisa Newport and colleagues have argued for age constraints in language acquisition (Johnson & Newport, 1989). Borer and Wexler (1987; 1992) put forth more detailed arguments for maturation of grammar. More recently, Wexler (2003) has argued for a Unique Checking Constraint in which children are initially constrained to use one checking operation where two are required, a constraint that fades with age.

The evidence I have laid out here is certainly consistent with the spirit of maturational arguments, although the linguistic details can seem to be a far remove from the known biological mechanisms involved in the central nervous system and higher cortical functioning. If we are ever going to be able to understand the timing mechanisms described in the opening quote from Purcell, we will need better models of the linking levels between brain and linguistic behavior. Colin Phillips (in press) argues that such hypotheses will be enhanced if grammatical knowledge is viewed as a real-time system for constructing sentences. His perspective is at the level of sentence processing. At the genetic level, Marcus and Fisher (2003) argue that the import of the FOXP2 gene is that it provides an entry point into neural pathways implicated in language by pointing to downstream targets which it regulates or the proteins with which it interacts. What I wish to suggest here is that within a larger, whole organism developmental time perspective, the condition of SLI provides a revealing model of how growth plays out in the language system. The rapid and highly synchronized growth of unaffected children can obscure the relative discreteness of the dimensions of language, and the ways in which timing mechanisms are robust even when the onset of a given element of the grammar is delayed.

5. Concluding Remarks

To recapitulate, the main points here are the following: The protracted period of language acquisition of children with SLI displays growth patterns surprisingly parallel to unaffected younger children, accompanied by an out-of-sync greater delay in the domain of finiteness, in a delay + Disruption pattern. The partitioned greater delay in finiteness (i.e., the disrupted growth area) nevertheless also tracks with the growth patterns of unaffected children albeit at an even greater age discrepancy. The general impression is of robust acquisition mechanisms shared by unaffected and affected children, with striking similarities in timing mechanisms. The assumption that the affected children have access to the same mechanisms of unaffected children is strengthened further by a similar, very low error rate in the morphosyntactic structures under investigation and similar performance on grammatical judgment tasks requiring children to detect errors of usage of the sort that do not appear in their spontaneous productions, such as overt subject/verb agreement errors. Growth in finiteness, which is characterized as overcoming an optionality option, is not predicted by a child's nonverbal intelligence, vocabulary development or mother's education, thereby suggesting a meaningful dissociation of the obligatory properties of finiteness from the learning mechanisms central to general vocabulary development or performance on nonverbal intelligence tests.
The parenting habits of better educated mothers also evidently do not hasten a child’s resolution of the optional phase of finiteness marking.

Let me elaborate a bit more on some key points. One is that the current theoretical interest in the central role of finiteness in morphosyntax is most felicitous for adding to our understanding of the nature of SLI, and the way that growth plays out. Empirically, the outcomes provide strong support for the notion of finiteness as a unifying element among a small set of morphemes that vary in other ways. The lexical affixes of third person singular present tense -s and regular past tense -ed are linked in growth with the copula BE, auxiliary BE and DO, and even stem-internal vowel changes of irregular past tense morphology. While this may seem self-evident in the context of the current literature, a bit more than a decade ago there was no clear direction as to how to differentiate among morphemes that might show a disrupted pattern of growth from those that did not. Without this clear separation, delayed morphemes get lumped together with disrupted morphemes, and the correct generalizations are obscured. Instead, we see that an abstractly unified set of morphemes grows (or remains greatly delayed) as a set. These linkages pose a challenge for models of language acquisition that posit that children acquire language in a piecemeal fashion, in localized rules, given the many differences within the set of morphemes.

Does this mean that finiteness-marking, as a grammatical tense marker, constitutes the only possible marker of delay + disruption? As Rice and Waxler have repeatedly stated, detection of one such marker does not rule out the possibility of other markers. A number of such candidates are under careful investigation now, in English and other languages. It is hoped that such inquiries will be fruitful, because this would contribute to our collective understanding of the possible points of vulnerability in the grammars of affected children. At the same time, there are theoretical advantages to the operating hypothesis that, by and large, affected children share the same underlying language acquisition mechanisms, as do unaffected children. This reduces the need to posit faulty acquisition mechanisms that then must be reconciled with the extensive evidence of relatively robust acquisition (including the striking evidence of parallels in growth and timing) and general avoidance of grammatical errors. Put another way, the big challenge for models of faulty learning or faulty grammar is to account for what the children with SLI do know, as well as their apparent limitations, in an extension of the well-known learnability problem.

One possible caveat is that relatively little is known of the late childhood-adolescent period of language impairment in children with SLI. The question judgment data reported here, along with unreported data collection underway, strongly points toward possible plateaus, and the likelihood that the earlier period of optional use of finiteness markers may not fully resolve into the expected adult grammar. This may be part of the apparent discrepancy between the characterization of SLI children presented here and those older children described by Heather van der Lely as “grammatical SLI,” which she argues display core deficits in the syntactic system in the form of a deficit in dependent relations, evident in passives and complex sentence structures (cf. van der Lely, 2003). In contrast, the SLI children reported here at 10 years of age understood reversible full passives as well as age controls and they were equally likely as age controls to detect semantic errors of agent assignment on grammatical judgment tasks (Rice, Waxler & Francois, 2001). Van der Lely attributes the differences to different subtypes within the SLI population. At present, direct comparisons across studies are confounded by several factors. One factor is the different developmental levels under study. As is evident in Figures 2 and 3, characterizations of differences between affected children and age controls or even vocabulary-matched controls will be greatly affected by the time of comparisons, i.e., the points in the developmental curves, or possible plateaus, of observations. The other factor is the type of task that is administered. Tasks intended for older participants can differ from those developed for younger participants in multiple ways, and at least some of those ways may also contribute to differences in outcomes. All of which is to say that although there are significant theoretical issues involved in the different interpretations, much of the ultimate resolution of apparent differences depends on more extensive empirical evidence.

To return to the import of the timing differences, let us consider the implications of delay and disruption of language. The delay is evident in the vocabulary development of children with SLI, as well as the increase in utterance length that is characteristic of the early childhood period. The fact that the two dimensions are correlated at each time sample, seem to follow highly similar growth trajectories, and show strong similarities between control and affected children all suggest that these two indicators are part of the same general language acquisition system. The puzzle here is why are the affected children behind when they apparently are following the same growth trajectories? Obviously, the answer to the puzzle depends on evidence prior to the growth period documented here, ostensibly in the onset period, assuming that the linear lines project in a linear fashion to the period below 3 years of age for the controls and below 5 years of age for the affected children. The best guess is that the affected children suffer a delay in the onset of language acquisition, an observation consistent with the well-documented Late Talker phenomenon and clinical cases which are typically referred on the basis of parental concern about delayed onset.

Although the earliest stages of language acquisition have been extensively studied and there is good documentation of the expected rate of vocabulary development, there is surprisingly little known about the exact mechanisms that activate the language system, and even less is known about why this activation does not start at the expected time for a given child. Clearly this is a very important issue for scientific inquiry.

Now to the topic of disruption. It is obvious in the differences between affected and younger control children that the timing mechanisms for language acquisition are not intrinsically fully synchronized across all dimensions of language. Grammatical tense marking appears to be controlled by timing
mechanisms not fully articulated with the timing of other dimensions, such as MLU or receptive vocabulary, or even other elements of morphosyntax, such as configurational knowledge. In this study, essentially it boils down to the observation that the early period of optionality persists for an even longer period for affected children, but once it begins to fade and obligatory use of finiteness morphology increases, then the growth unfolds in the same way regardless of affectedness status. The question here is a variant of the one above: Why are the affected children even further behind in this property of grammar, and why would they manifest the same growth path? The interpretation is that they are stuck for a longer time before they move beyond optionality, or, under Waxler’s model, before they resolve the Unique Checking Constraint. Out of possible change agents, none of the three studied, i.e., child vocabulary, nonverbal IQ or mother’s education, seem to play a role, leaving unknown the reasons.

It bears repeating that the timing mechanisms involve linear growth, and some elements, such as grammatical tense marking, also involve quadratic and cubic growth elements involving points where the acceleration rate increases or decreases. These change points are referenced to elapsed time from onset, not chronological age set points. Consider, for example, that it could have been that if an age-referenced change point was missed a youngster might never continue in the expected growth curve, but would instead plod along in a steady upward rate that falls further and further behind the expected growth pattern. That is clearly not the case, however. What triggers the points of change of acceleration and what accounts for the consistent time lapse between points of change, and what accounts for a lower plateau of performance by the affected children? These factors remain unknown.

Issues of timing are not unlike those now becoming the focus of biological inquiry, and issues that are moving to the forefront of genetics investigations. The regulatory properties of the FOXP2 gene locus of the KE family are inherently time-referenced, linked to instructions for the development of cortical tissue. Conventionally, we have thought of genotype/phenotype investigations as involving determination of affectedness and then comparing the DNA of affected with unaffected individuals. Although this approach is still the central one for inquiry, the evidence reported here suggests that perhaps we can benefit by asking if the definition of affectedness can be improved by identification of specific timing patterns associated with inherent mechanisms. This may allow greater precision in the evaluation of possible environmental issues and intervention effects, both of which will be essential in the emerging investigations of genetic influences, as well as greater precision in possible biological factors.

Let me conclude by expressing my deep appreciation to the families and children that agree to participate in our studies. The condition of SLI is a lasting individual difference, one that runs in families and contributes to an individual’s lifelong challenges in ways that go far beyond the grammatical limitations that we have been able to study. My colleagues and I are appreciative of the ways in which linguistic developments, and the community of scholars who participate in the BUCLD, have helped add to our understanding of the condition of SLI and our progress toward ways to reduce or ameliorate the insidious effects of this condition.

References


Purnell, Beverly (2003). To every thing there is a season. Science, 301, 325.


