How Different Is Disordered Language?

MABEL L. RICE

Studies of infant pathways to language have enlightened our appreciation of the richness of the neurocognitive resources available to babies and how they come prepared to acquire language. In a search for general properties that hold across many infants, the research has focused on healthy, robust babies who are comfortable with the experimental methods and have "normal" language aptitude. The full range of infants, however, includes some who are healthy but do not have strong aptitude for language. This chapter takes up what is known about these youngsters as they grow into the time of language use, the ways their language acquisition parallels or differs from that of other children, and the issues that arise when we try to account for language limitations as well as strengths. The ultimate goal is to arrive at interpretive models that encompass individual variation in language acquisition as well as the broad ways in which children share language acquisition abilities.

The chapter is organized as follows. The first section provides a brief overview of children with specific language impairment (SLI), a condition in which language acquisition does not meet normative expectations, although the children are healthy otherwise. The following sections describe dimensions of language acquisition from infancy onward, with an emphasis on the similarities and differences between affected and unaffected children. The second section begins with language onset, defined as the first use of words at 15–18 months and simple sentences at 24 months and the phenomenon of late language emergence (LLE) as the likely beginning period of SLI. The third section lays out the growth trajectories of children with SLI, from the preschool to the early elementary age range, with evidence of continued delays and disruptions in the grammar, and ways the dimensions of language are synchronous and asynchronous. The fourth section highlights that at the same time there are delays and disruptions there are also striking strengths in the grammar, similar to unaffected children.
The fifth section explores implications of the findings from children with SLI for models of infant pathways to language, and the final section provides brief concluding comments.

**CHILDREN WITH SPECIFIC LANGUAGE IMPAIRMENT**

Beginning with a clinical case history reported by Gall (1835), the scientific literature has documented the existence of children who are slow to acquire language, although there are no other apparent developmental disabilities and no obvious causal factors. In the modern literature, the commonly accepted research definition of SLI is based on both inclusionary and exclusionary criteria. The inclusionary criteria document that language acquisition does not meet normative expectations by comparing a child’s performance with levels expected for the child’s age. This is usually done by administering a general test of language acquisition composed of tasks that evaluate multiple dimensions of language in expressive and receptive language formats. The diagnosis of language impairment is defined as performance at the low end of the age level distribution, around the bottom 10th to 15th percentiles. The gold standard for research includes evaluation of speech performance as well to determine if children with poor intelligibility or word pronunciation are unable to demonstrate their vocabulary, morphological, or sentence formulation competencies because of speech limitations. In the full population of children with SLI, speech impairments are orthogonal to language impairments (Shriberg, Tomblin, & McSweeny, 1999), although children enrolled in clinical treatment are more likely to have both speech and language impairments. Children with SLI are likely to be overlooked for clinical services; Tomblin et al. (1997) report that only 29% of the children identified in their epidemiological study of kindergarten children had been identified for treatment.

The exclusionary criteria are intended to select affected children whose developmental impairment is limited to language (see Rice & Warren, 2004; Rice, Warren, & Betz, 2005, for information about language disorders across different clinical groups). Children with hearing loss are excluded. Children diagnosed with Williams syndrome, Down syndrome, and fragile X syndrome, are excluded, as are children with epilepsy and other neurological disorders. Conventionally, children with autism have been excluded, although in the recent shift to the broader clinical category of autism spectrum disorders (ASD) the diagnostic boundary between SLI and autism is somewhat blurred and is currently a matter of active investigation. It is generally accepted that social impairments form the core of ASD whereas language impairments form the core of SLI (see Tager-Flusberg, 2004, 2005, for overviews). Mental retardation is usually ruled out via exclusion of children whose nonverbal intelligence
quotient (IQ) performance levels are 85 or below. The range between 70 and 85 nonverbal IQ is sometimes invoked as acceptable for the label of SLI, although it is preferable to label this range as nonspecific language impairment (NLI) (Tomblin & Zhang, 1999) and treat children in this range as a separate clinical group (see Rice, Tomblin, Hoffman, Richman, & Marquis, 2004, for evidence of differences between the SLI and NLI groups in acquisition of past-tense morphology.) Finally, children with dialectal differences or bilingualism are usually excluded from experimental studies of SLI in order to avoid confounds in native language exposure.

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

The nondelay model of interest here is one of disruption—referred to as a delay within a delay model in Rice (2003) and updated to a disruption notion in Rice (2004). In this model, some elements of language are out of harmony, or disrupted, relative to others, leading to a lack of synchrony in the overall linguistic system. Evidence for this possibility is lower performance of the SLI group compared with either of the control groups on a given linguistic dimension, indicating that affected children's low performance extends beyond that expected for a general immaturity relative to age expectations. Areas of disrupted synchrony are of theoretical interest, because they show how the linguistic dimensions that are tightly intertwined in typically developing children are to some extent independent elements that can be selectively affected and fall behind in acquisition.

This chapter focuses on the comparison of unaffected children and children with SLI. Another perspective on language strengths and weaknesses is apparent when comparing language impairments across different clinical categories (see Rice et al., 2005; Rice & Smolik, 2007). Of interest here are three observations. One is that mental retardation does not fully predict language
impairments—that is, children with low nonverbal IQ levels can have language levels commensurate with age expectations. The second is that social impairment does not fully predict language impairment—that is, children with autism can have language levels commensurate with age expectations. Third, language impairment does not necessarily manifest as a global diminution of ability; instead, certain dimensions of language, such as vocabulary development, can be at or near age expectations whereas other dimensions, such as syntax, can lag further behind. Thus, clinical conditions beyond SLI demonstrate the language-specific properties of language impairment and the need for consideration of individual dimensions of language.

DELAYED ONSET AS THE BEGINNING OF SLI: LATE LANGUAGE EMERGENCE (LLE)

As the chapters in this volume attest, language emerges from the infancy period, in the way of early comprehension of spoken language, first words and first combinations of words into simple sentences. Parental report measures of children’s first words and word combinations indicate that 10%-20% of 24-month-old children show LLE, defined as using fewer than 50 words or no word combinations (Fenson et al., 1993; Klee et al., 1998; Rescorla, 1989; Rescorla & Achenbach, 2002; Rescorla & Alley, 2001; Rescorla, Hadick-Wiley, & Escarce, 1993). A recent epidemiologically ascertained sample of 1,766 24-month-old children yielded an estimate of 13% LLE, defined according to parental report of children’s comprehension as well as production of language (Zubrick, Taylor, Rice, & Slegers, 2007).

The phenomenon of LLE is linked to SLI via the fact that late onset is considered to be a hallmark characteristic of children with language impairments. LLE can be the first diagnostic symptom of a subsequent difficulty with language acquisition. Rice (2004) proposes that a late language start-up may be a unifying feature of language impairment across many different clinical conditions.

In spite of our great progress in investigations of the cognitive and perceptual capacities that infants bring to language onset, the picture does not yet reveal the exact mechanisms that determine when language appears and the interactions among multiple factors that contribute to the timing of language acquisition. An experimental way to approach this question is to investigate variables that could predict which 24-month-old children will show LLE (excluding children with obvious related medical conditions). In the largest study of its kind, Zubrick et al. (under review) examined a large range of possible predictors that describe maternal, home, and child characteristics. It is noteworthy that in this large sample, variables associated with home resources (cf. Entwhistle &
Astone, 1994), including mother's education, family income, socioeconomic status, parental mental health, parenting style, and family functioning, did not predict LLE status. The significant predictors were only a handful out of a large number of variables:

- Gender (2.74 times the risk for boys than girls)
- The family history of speech and language delays (2.11 times the risk for families with a positive history)
- Number of children in the family (double the risk for families with two or more children)
- Perinatal status (1.8 times the risk for children with a low percentage of expected birth weight or gestation age less than 37 weeks)
- The children's early neuromotor skills (more than double the risk for children somewhat late in developing motor skills, although it must be emphasized that the LLE children's motor development is within normative expectations)

One conclusion to be drawn from the outcomes is that across a very wide range of maternal and family differences, including the conventional ways of assessing socioeconomic resources, children can avoid LLE. Put another way, they are likely to share the expected time of language onset regardless of wide variation in family resources. On the other hand, independent of maternal and family factors, children whose relatives were LLE were at greater risk. This suggests that parents or siblings, when they were toddlers, were also likely to be LLE. Shared biological risk, evident at the same developmental period, is implicated, independent of other environmental resources or learning opportunities. Furthermore, neuromotor development at this time is predictive, such that children with LLE are more likely to be less robust in motor development at 24 months. All in all, it appears that individual differences in language onset are linked to individual differences in family members for the same developmental period and that these differences are part of a broader profile of slightly immature neurological development.

To return to the notion of language delay, it is clear that children with LLE are delayed in language onset and, at the same time, that they share many of the factors thought to enhance children's language learning. In turn, this suggests an interaction of child and environment, such that some children cannot benefit from the language learning opportunities to the same extent other children can, and this is evident from onset. Exactly what sets the stage for onset readiness is an important question for studies of infant pathways to language.
PROTRACTED GROWTH OF SLI—DELAYS AND DISRUPTIONS, SYNCHRONIES AND ASYNCHRONIES

Much of the literature on SLI examines language delay versus language difference in the classical three-group cross-sectional design (cf. Leonard, 1998). The evidence of delays versus nondelays is mixed in these cross-sectional comparisons, dependent upon the power to detect group differences (many of the studies have small sample size), the language dimension of interest, the ages of the participants, and other experimental issues. More recently, longitudinal studies provide stronger evidence of similarities and differences over time.

Growth trajectories clearly show that delays are characteristic of some dimensions of language growth whereas disruptions are characteristic of other dimensions. Delays are evident in the growth of MLU and receptive vocabulary in children with SLI. Rice, Redmond, and Hoffman (2006) followed a group of 21 5-year-old children with SLI and 20 MLU equivalent children who were two years younger. The children were assessed at 6-month intervals for MLU, for a total of nine data points over 5 years, encompassing the years 3–9. As depicted in Figure 5.1, the two groups showed remarkable parallels in MLU growth. They were at equivalent levels of MLU at each time of measurement. Growth-curve modeling showed that there were no group differences in the growth trajectories. Each group showed linear and quadratic growth, with negatively accelerating growth; that is, at the later ages there was less of an increase in the MLU between times of measurement. It is as if the mechanisms that guide increased utterance length are working in the same way in the two groups over the observed

![Figure 5.1](image-url)  
Figure 5.1  Growth in MLU of children with SLI and unaffected younger controls.
time, even though the affected children are two years older than the controls, were enrolled in language intervention at the outset, and were at higher levels of formal education. A further implication is that when the growth trajectories are projected downward to an earlier age, assuming the parallel growth patterns early on, affected children would be likely to have a delayed onset of combining words into phrases and clauses. Thus, the LLE roots of SLI are supported by the pattern of growth during late preschool and beyond.

The growth modeling also evaluated possible predictors of growth in MLU for the two groups. Neither nonverbal intelligence nor mother's education level predicted growth, although for each group nonverbal intelligence predicted initial status (the intercept) in levels of MLU.

Growth in receptive vocabulary was tracked in the two groups as well (Figure 5.2). Receptive vocabulary was measured annually using the Peabody Picture Vocabulary Test-Revised (PPVT-R; Dunn & Dunn, 1981). The groups were not initially selected for equivalency on receptive vocabulary. At the outset, the affected group had a small but statistically significant numerical advantage based on comparisons of PPVT-R raw scores. At the end, the affected group had a small but statistically significant numerical disadvantage. The groups did not differ in the intervening times of measurement. In the growth model, there were significant linear and quadratic growth parameters, with group differences at the intercept (outset) and in linear rates such that the MLU equivalent group overcame the initial lower level of performance with a greater degree of linear change subsequently. It is as if the affected children benefited from the two years' age difference at the outset in the experience needed to acquire new words, but

![Graph](image)

**Figure 5.2** Growth in PPVT-R raw score of children with SLI and unaffected younger controls.
this advantage was overcome by a slightly better rate of learning new words in 
the younger group.

The predictor relationships were the same as with the MLU outcomes: Mother's 
education did not predict growth in vocabulary for either group, and nonverbal intel-
ligence predicted initial status in vocabulary to the same extent in both groups.

Overall, although there are some minor ways in which growth in vocabulary 
is not exactly parallel across the two groups, the big picture is how strikingly sim-
ilar growth plays out during this time. It looks as if the dimensions of MLU and 
vocabulary are tracking in the same way across the groups, once adjusted for a 
delayed onset for the affected group. It appears that once the system starts, it will 
move forward with the same momentum for children with SLI as for younger 
children, and growth in MLU and vocabulary is synchronized, at least at a gen-
eral level, in the same way for both groups.

The picture is much different with regard to the grammatical function of 
finiteness marking. At the descriptive level, finiteness is a fundamental structural 
property of clauses that requires tense and agreement marking (usually in the 
form of overt morphemes) on the occupant of the main verb position in a clause 
(cf. Quirk, Greenbaum, Leech, & Svartvik, 1985). Recent theoretical advances in 
generative grammar (Chomsky, 1995) have linked finiteness (with a morphological 
requirement) to syntax in rules for movement of elements within a clause. The 
term morphosyntax is used to describe this interaction. Because of its central 
place in the grammar, there has been great interest in the ways children acquire 
competency with finiteness across many languages (cf. Guasti, 2002; Schütze, 
2004).

The exact ways young children show their understanding of finiteness marking 
depend on the morphosyntactic properties of their native language. In English, 
finiteness is marked by the following morphemes:

- Third-person singular present tense -s: "Patsy runs home every day."
- Past tense -ed or irregular past tense: "Patsy walked/ran home yesterday."
- Copular or auxiliary be: "Patsy is happy" or "Patsy is running."
- Auxiliary do: "Does Patsy like to run?"

Early on, English-speaking children produce uninflected verbal forms, such as 
"Patsy go home," or omitted finiteness marking Be or Do, as in "Patsy happy. Note 
that the set of morphemes is not limited to verbal affixes but includes irregu-
lar stem-internal morphophonological variants and free-standing morphemes 
/forms of Be and Do/.

Wexler (1994, 1996) initially labeled this an optional infinitive stage. Later this 
was amended to an agreement/tense omission model (ATOM) (Schütze & Wexler, 
1996; Wexler, Schütze, & Rice, 1998) and then to a unique checking constraint
**model** (Wexler, 1998). 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. In English-speaking children this is evident in their tendency to omit finite forms early on. As children develop, the likelihood of finiteness marking in obligatory clausal contexts grows to a level that ultimately reaches the adult levels.

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

Growth-curve data are available for the same children studied for MLU and vocabulary development (cf. Figure 5.1 and Figure 5.2). These data make it clear that, as predicted, affected children do show a delay within a delay in this part of the grammar, leading to a disruption in the expected synchrony across the different dimensions of grammar as growth plays out. Figure 5.3 reports the outcome for affected and unaffected children over the period of 3 to 8 years of age. For unaffected children, the percentage correct in obligatory contexts for a composite estimate of finiteness (collapsing across the different morphemes) starts out around 55% for 3-year-olds at the first time of measurement and quickly moves toward adult usage by age 4. In contrast, the affected children are at much lower levels of use at the first time of measurement at 5 years, and this gap persists for years.

![Figure 5.3 Growth in finiteness marking of children with SLI and unaffected control children.](image)
Growth-curve modeling, however, shows that the two groups follow the same models, indicating linear and quadratic components for both groups. The predictor relationships were also the same across groups: Growth was not predicted by a child’s nonverbal intelligence, mother’s education, or PPVT-R vocabulary scores at the outset, although a child’s initial MLU did predict rate of acquisition. The findings replicated for irregular past tense, when irregular accuracy was calculated as finiteness marking by regarding overregularizations as finiteness markers (Rice, Wexler, Marquis, & Hershberger, 2000). Further, the findings also replicate for judgment tasks, indicating that the effects are not restricted to production demands but also are evident in children’s likelihood to accept utterances as well formed with the same kirds of omissions that they produce (Rice, Wexler, & Redmond, 1999).

Cross-sectional studies from other labs also find that children with SLI, as a group, are likely to perform less accurately than younger controls on morphemes associated with the finiteness marker (cf. Bedore & Leonard, 1998; Conti-Ramsden, Botting, & Faragher, 2001; Grela & Leonard, 2000; Joseph, Serratrice, & Conti-Ramsden, 2002; Leonard, Eyer, Bedore, & Grela, 1997; Marchman, Wulfeck, & Ellis Weismer, 1999; Oetting & Horohov, 1997). Thus, the empirical phenomenon is well established.

To recapitulate, for MLU and vocabulary acquisition, children with SLI follow a delayed growth trajectory that shows strong similarity to younger unaffected children. For morphosyntax, and more particularly for the property of finiteness, affected children also follow the same growth trajectory as younger unaffected children, but in this dimension the onset is further delayed and the actual levels of performance fall below that of younger children. So whatever is driving growth shows strong similarities across all children, and at the same time growth is not uniform across all dimensions of language, such that semantics and morphosyntax are unlikely to be sharing the same broad mechanisms to drive change.

STRENGTHS IN THE GRAMMAR: AVOIDING OVERT MISTAKES EVEN WITH DELAYS AND DISRUPTIONS

The nature of children’s grammatical errors has long been appreciated as a source of information about their acquisition mechanisms. In the domain of morphosyntax, current theories posit conditional relationships between morphology and syntax such that certain errors are predicted not to appear if other elements of clause structure are in place. In particular, the theory of the optional infinitive stage, and subsequent modifications to the theory, predict that if finiteness markers appear in obligatory contexts, then certain kinds of other grammatical errors would be very unlikely. This is because the theory carries the related assumption that young children know much of the adult syntactic
system, including rules that apply to overt inflected verb forms and the basic configuration of clausal structure that provides the location in a clause where finite forms appear.

Clause-by-clause inspection of children’s utterances and responses to elicitation tasks are required to determine if there are errors of morpheme use that would indicate underlying syntactic deficiencies. This is laborious and often not done, especially in studies of children with SLI. More recently, however, data systems have included close examination of possible errors, which include the following:

1. Violation of the requirement that there is only one site for finiteness marking in a clause and no more than one finiteness marker can appear, as shown in the following examples:
   • Runs Patsy home every day?*
   • Does Patsy likes to run?*
   • Patsy is runs home very day.*
   • Does Patsy is happy?*

2. Violation of the requirement that agreement marking on the verb must match the person and number features on the subject, as shown in the following examples:
   • He am happy.*
   • They is mine.*
   • I likes to paint.*
   • She are tired.*

3. Violation of the requirement that nominative-case marking of the subject is required in finite clauses, as shown in the following examples:
   • Him wants a cookie.*
   • Her runs home.*
   • Me am going.*

A general finding is that such errors are rare or very unlikely even during the period when children are likely to use finiteness markers inconsistently or are likely to have nominative-case errors (when accusative case appears it is much more likely to be when finiteness markers are omitted; cf. Wexler et al., 1998). This does not mean that such errors never appear but that when benchmarked to obligatory contexts (opportunities for error) the rate is at best very small. Leonard, Camarata, Brown, and Camarata (2004) report a similar outcome for the observed error patterns in an intensive training study. The empirical generalization is that when finiteness errors are defined as violations of the adult grammar, children’s errors (for children with SLI as well as unaffected children) are overwhelmingly omissions of finite forms in obligatory contexts.
The conclusion is that although children with SLI show a laboriously long period of acquisition of finiteness, they nevertheless follow the same growth trajectories as unaffected children and the same relationships with potential predictors of growth and also avoid possible errors in much the same way as unaffected children.

ISSUES FOR WORKING OUT INFANT PATHWAYS TO LANGUAGE

Studies of children with SLI draw our attention to some fundamental issues that don't always show up in the normative literature but that nevertheless are vital components of the general explanation of how babies progress to become such proficient users of language. Beginning with the LLE phenomena described earlier, it raises the question of potential gender effects early on. In contrast to the strong disadvantage for males in the low performance range, there is only a modest advantage for females across the full range of performance (Fenson et al., 1994; Huttenlocher, Haigh, Bryk, Seltzer, & Lyons, 1991; Wells, 1985). This suggests that the influences on language abilities in the low performance range may exaggerate influences within the normal range. Furthermore, by kindergarten, in epidemiologically ascertained samples the risk of SLI for boys and girls is nearly the same: 8% for boys and 6% for girls (Tomblin et al., 1997). This is a puzzling picture if indeed boys are likely to start later but are able to overcome the early delay. The roots of these differences appear to be in infancy.

In addition, the idea that babies share with their family members a likelihood of LLE draws our attention to the ways genetic influences might operate. Granted that there are challenges in the measurement systems available to estimate childhood delays from retrospective reports of adults, there are at the same time growing indications of likely genetic contributions to SLI (see Rice & Smolik, 2007, for a review). The recent discovery of genetic causes of risk for prematurity (Wang et al., 2006) alerts us to the need to more systematically investigate biological bases of individual differences in infants.

Related to this point is the fact that, in the evidence reviewed here, the role of environmental influences related to parenting and home resources points away from these variables as causal agents for LLE and SLI. At least in the way such influences have conventionally been indexed, the evidence is meager at best for the intuitive notion that such delays and differences in language are attributable to a lack of appropriate parenting or environmental resources. This is not to say that neglect in extreme cases cannot play a causal role; obviously, it does, and it probably works in multiple ways to increase risk for language acquisition. The point here is that within the broad range of acceptable parenting practices, with presumably a basic level of language input, some children are LLE, and some are not; some children have SLI, and some do not. It also must be said that this conclusion does not rule out environmental intervention as an important element in
treating LLE and SLI. It does indicate that additional enrichment is needed and
cautions that accelerated growth will be a challenge to achieve. Just as infants'
constitutional factors are considered in models of infant attention, cognition,
and perception, so they need to be considered in language acquisition.

The picture of growth similarities and differences between SLI and unaffected
children poses some very challenging interpretive issues. As noted earlier, there
is a parsimony advantage in assuming that children with SLI share the same
underlying mechanisms as unaffected children, in tandem with a corollary that
language is a specific cognitive domain with internal dimensionality. Contrary
to the position of advocates for a global model of language and intelligence, a \( g \)
factor (cf. Plomin, 1999), evidence from language impairments across clinical
conditions strongly indicate that language has specific properties; it can be dis-
sociated from nonverbal intelligence and the growth of certain dimensions, such
as morphosyntax, is independent of nonverbal intelligence. Further, growth does
not play out the same across language dimensions. To ignore these findings is to
invite heterogeneity in empirical assessments that introduce unknown sources of
error.

Given this, further examination of the interpretive challenges is warranted.
A focus on the differences (i.e., the language impairments relative to unaffected
children benchmarked to age levels) invites the hypothesis that children with
SLI have fundamental, perhaps language-specific, differences in underlying
perceptual or cognitive mechanisms. There have been many versions of this gen-
eral category of hypotheses, but for this discussion it is not necessary to critique
particular ones. The general challenge for such perspectives is to account for
the many ways affected and unaffected children are similar. If they have faulty
perception, memory, or cognitive mechanisms, what constrains their errors and
matches their growth trajectories to those of unaffected children? The similarity
in growth trajectories suggests that the gains over time, and possible points
of change in the acceleration of growth, are similar for affected and unaffected
children. How can this be achieved with systems fundamentally different from
unaffected children? If affected youngsters accomplish what they do with defec-
tive mechanisms, how do they overcome the putative consequences of such limi-
tations? These questions pose strong challenges for interpretive models.

On the other hand, a focus on the similarities by assuming that the underly-
ing language acquisition mechanisms are fundamentally similar for affected and
unaffected children brings other questions: What accounts for the delayed onset?
What accounts for selective weaknesses in particular dimensions of language? The
extended optional infinitive (EOI) hypothesis (Rice et al., 1995) provides an explicit
model for a well-defined element of morphosyntax that accounts for selective
weakness and parallels to younger children and captures well many empirical
predictions pertaining to clause structure and long-term risk for morphosyntax.
This model brings many advantages, including an explicit link to models of adult grammar as the end-state grammar, and explicating of some of the constraints that may be operative in avoiding errors. It has inspired many follow-up studies that push the boundaries of morphosyntactic phenomena accounted for, within English and across other languages, and it challenges the technical points of the interpretation.

These advantages notwithstanding, an important phenomenon outside the scope of the EOI model is ILE. Maturational mechanisms are invoked as causal factors that unify the growth patterns of affected and unaffected children (cf. Rice, 2007; Wexler, 2003), but ultimately this is an intermediate abstraction to describe unknown mechanisms for driving time-referenced change during the early period of language acquisition and perhaps as a sustaining time-locked mechanism that plays out in a broader developmental trajectory. Fisher (2005) describes advances in neurogenetics that carry promise for clarifying time-locked phases of cortical development that have relevance for language acquisition and other areas of cognitive development. Although "maturational mechanisms" are not well specified at the moment, a path into this black box may not be too far in the future.

CONCLUSIONS

To reprise the title of this chapter, "How Different Is Disordered Language?" the answer may be, "Not as different as assumed earlier, and surprisingly similar in many important ways." The full picture requires recognition of the dimensions of language, of the difference between language delay and language disruption, of separate contributions of a delayed start-up versus the slope and trajectories of growth once the acquisition system is under way, and of a healthy skepticism toward intuitive models of poor parenting as a causal agent. The least explored parts of the picture are the nature of the language-acquisition path from infancy through the toddler period and beyond, the nature of individual differences that infants bring to the journey, and the ways the two elements interact to create risk for language impairment or to ensure that a child will follow the expected trajectory. The experimental and empirical challenges are considerable, but the potential benefits of more information are substantive, for adding not only to our scientific knowledge about basic human abilities but also to our ability to ensure that each child is maximally prepared to benefit from language ability.

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ENDNOTE

1. Asterisk denotes ungrammatical sentence.

REFERENCES


