63 The Cognitive Neuroscience of Language Acquisition

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ABSTRACT This chapter reviews findings from several research areas-normal language acquisition, learnability theory, developmental and acquired language disorders, and language acquisition after the critical period-indicating that the ability to acquire language is the result of innate brain mechanisms. It is possible that infants' brains are predisposed to perceive categorically such stimuli as phonemes, words, syntactic categories, and phrases, and this predisposition allows children to acquire language rapidly and with few errors.

Because the ability to learn a language is a uniquely human ability, language acquisition is an important topic in cognitive neuroscience. Perhaps the most fundamental question about language and language acquisition is the extent to which the ability to learn language is the result of innate mechanisms or predispositions (henceforth referred to as innate abilities). Innate abilities often share certain characteristics. An innate ability is usually present in all normal individuals. Its acquisition tends to be uniform and automatic, with all normal individuals going through the same stages at the same ages, without specific instruction. There may be a critical period for successful acquisition. The ability is likely to be functionally and anatomically autonomous or modular. Finally, the trait may be heritable.

Although these characteristics are by no means definitive, they can be used to evaluate traits that may be innate. Consider, for example, the ability to walk and the ability to knit. The ability to walk exhibits most of the hallmarks of innate abilities, and is presumably innate; but the ability to knit exhibits few of these hallmarks, and is presumably not innate. If children's brains are innately predisposed to learn language, then given adequate ex to language, all children with normal brains should, without instruction, learn language in a relatively uniform way, just as normal vision develops given adequate exposure to visual stimuli (Hubel and Wiesel, 1970). But if the ability to learn language is not innate, instruction may be necessary, the course of acquisition may vary greatly from person to person (perhaps as a function of the quality of instruction), and there may be no critical period for acquisition.

Even if the ability to learn language is the result of innate mechanisms and. predispositions, another question remains: Are these mechanisms specific to language and language acquisition (e.g., Chomsky, 1981, 1986; Pinker, 1994) or are they also involved in tasks and abilities that are not linguistic (e.g.,. Karmiloff Smith, 1991; Elman et al., 1996)? If the ability to acquire language is the result of innate mechanisms used solely for language and language acquisition, language may be functionally and anatomically autonomous or modular from other abilities, in which case developmental and acquired lesions may specifically impair or spare the ability to learn language. Conversely, if general-purpose mechanisms are involved in language acquisition, we would not expect to find evidence of the functional or anatomical modularity of language or language acquisition.

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Language development

LINGUISTICS AND THE UNIVERSAL FEATURES OF LANGUAGE Superficially, learning to talk differs from learning to walk in that children are capable of learning many different languages, but just one basic walk. If children really are predisposed to learn all human languages, then all languages must be fundamentally the same. In fact, linguists have discovered that, although some languages seem to differ radically from other languages (e.g., Turkish and English), in essential ways all human languages are remarkably similar to one another (Chomsky, 1981,1986; Croft, 1990).

Generative linguists usually assume that language involves rules and operations that have no counterparts in nonlinguistic domains and that the ability to use and acquire language is part of our innate endowment.. For example, within principles-and-parameters (P&P) generative theory (Chomsky, 1981, 1986), all languages are said to share a common set of grammatical principles. Differences among languages result from the different parametric values chosen for those principles. According to P&P theory, at some level, children are born knowing

the principles that are universal to all languages (Universal Grammar); thus, to learn a particular language, all they must do is learn the vocabulary and parametric settings of that language. Similarly, within another generative linguistic theory-optimality theory (OT)-the same universal constraints operate in all languages, and languages differ merely in the ranking of these constraints (Prince and Smolensky, 1993). According to OT, children are born "knowing" the universal constraints; thus, to learn a particular language, all children must do is, learn the vocabulary and ranking of constraints for that language (Tesar and Smolensky, 1996). Linguists working within the functionalist tradition (e.g., Foley and Van Valin, 1984) are more likely to assume that language shares properties with nonlinguistic abilities, and that operations used in language acquisition are used in other, nonlinguistic domains (e.g., Bates and MacWhinney, 1982; Budwig, 1995; Van Valin, 1991).

UNIFORMITY IN LANGUAGE ACQUISITION Within a given language, the course of language acquisition is remarkably uniform (Brown, 1973).' Most children say their first referential words at 9 to 15 months (Morley, 1965; Benedict, 1979; Fenson et al., 1994; Huttenlocher and Smiley, 1987), and for the next 6-8 months, children typically acquire single words fairly slowly until they have acquired approximately 50 words. For most children acquiring English, the majority of their first 50 words are labels for objects (e.g., *cookie, mother, father, bottle)* with a few action verbs *(eat, come, go),* social terms *(good-bye, hello),* and prepositions *(up, down)* rounding out the list (Nelson, 1973; Bates et al., 1994; Benedict, 1979). Once children have acquired 50 words, their vocabularies often increase rapidly (e.g., Reznick and Goldfield, 1992; Benedict, 1979; Mervis and Bertrand, 1995), expanding by 22 to 37 words per month (Benedict, 1979; Goldfield and Reznick, 1990).

At around 18 to 24 months, children learning morphologically impoverished languages such as. English begin combining words to form two-word utterances such as *want cookie, play checkers,* and *big drum* (Brown, 1973). During this two-word stage, the vast majority of children's utterances are legitimate portions of sentences in the language they are learning. Thus, *in* English-a language that has restricted word order-children will *say want cookie* but not *cookie want* (Brown, 1973) and *he big* but not *big he* (Bloom, 1990). Children acquiring such morphologically impoverished languages gradually begin to use sentences longer than two words; but for several months, their speech often lacks phonetically unstressed functional category morphemes such as determiners, auxiliary verbs, and verbal and nominal inflectional endings (Brown, 1973; Mills, 1985; Schieffelin, 1985). Representative utterances during this period include *Sarah want cookie, Where Humpty Dumpty go* and *Adam write pencil.* Children's early speech is often described as "telegraphic" (Brown, 1973) because it resembles the way adults speak when words are at a premium, as in a telegram. Gradually, omissions become rarer until children are between three and four years old, at which point the vast majority of English-speaking children's utterances are completely grammatical (Stromswold, 1990a,b, 1994b). Children who are acquiring languages like Turkish, which have rich, regular, and perceptually salient morphological systems, generally begin to use functional category morphemes at a younger age than children acquiring morphologically poor languages (Aksu-Koc and Slobin, 1985; Berman, 1986; Peters, 1995). For example, in striking contrast to the telegraphic speech of English-speaking children, Turkish-speaking children often begin to produce morphologically complex words before they begin to use multiword utterances (Aksu-Koc and Slobin, 1985) 2

Within a given language, children master the syntax (grammar) of their language in a surprisingly similar manner (Brown, 1973). For example, children acquire the 14 grammatical morphemes of English in essentially the same order (Brown, 1973; deVilliers and deVilliers, 1973). Similarly, all 15 of the children I studied acquired the 20-odd English auxiliary verbs in essentially the same order (Stromswold, 1990a). The order in which these 15 children acquired complex constructions-questions, negative constructions, passives, datives, exceptional-case-marking constructions, embedded sentences, preposition-stranding constructions, causative constructions, small clause constructions, verb-particle constructions, and relative clauses constructions-was. also extremely regular '(Stromswold, 1988, 1989a,b, 1990a,b, 1992, 1994b, 1995; Stromswold and Snyder, 1995; Snyder and Stromswold, 1997). Finally, to a remarkable degree, within and across languages, children make certain *types* of mistakes and not others.

ACQUISITION OF SYNTACTIC CATEGORIES In order to acquire their language, children must not only learn the meanings of words like *cat* and *eat,* they must also learn that words like *cat* are nouns and words like *eat* are verbs. That is, they must learn the categorical membership of words. This is critical because whether a syntactic or morphological ri& applies to a particular word depends on its categorical'nembership; not on its meaning. Consider, for example, the entence *Linus cratomizes Lucy:* Any speaker of English automatically knows that *Linus is* the grammatical subject of the sentence (be-,, cause, within an intonational clause, it is in preverbal position), *Lucy* is the grammatical object (because it is in

postverbal position), and the nonsense word *cratomize is* a lexical verb. Even without knowing what *cratomize* means, an English speaker automatically knows that its progressive form is *cratomizing* and that its past tense form is *cratomized;* that do-support is required to ask a standard matrix question (e.g., *Did Linus cratomize Lucy?* and not **Cratomized Linus Lucy?*[3] or. **Linus cratomized Lucy?)* or negate an utterance (e.g., *Linus didn't cratomize Lucy* and not **Linus cratomized not Lucy, *Linus not crat= omized Lucy);* and that the grammatical subject precedes rather than follows *cratomize* in simple declarative utterances (e.g., *Linus cratomizes Lucy* and not **cratomizes Linus Lucy).* The fact that English speakers know the syntactic and morphological behavior of *cratomize* without having the slightest idea what *cratomize* means demonstrates that categorical membership and not meaning determines syntactic and morphological behavior. A central question in the field of language acquisition is how children learn the categorical membership of words. For adults, the answer is simple. Even from the single sentence *Linus cratomizes Lucy,* adults recognize that cratomize is clearly a verb-it appears after the grammatical subject *Linus* and before the object *Lucy,* has the third-person verbal inflection -s, and exhibits other verb-like properties. The answer is much trickier for children.

How do children learn which words are verbs if they don't know what properties are typical of verbs? And how can they learn the properties of verbs if they don't know which words are verbs? One simple possibility is that every verb in every human language shares some readily accessible property for which children are innately predisposed to look. Unfortunately, no such property seems to exist. Instead, infants probably rely on a combination of cues-prosodic, semantic, and correlational-to learn which words are nouns and which are verbs (Pinker, 1987). Infants may, for example, use prosodic cues such as changes in fundamental frequency and lengthening to help determine where major clausal and phrasal boundaries are. Combined with knowledge of the universal properties of clauses and phrases (e.g., that verbs are contained within verb phrases and sentential clauses contain noun phrases and verb phrases), this could help children learn which words are verbs (Jusczyk et al., 1992; Jusczyk and Kemler Nelson, 1996; Morgan and Demuth, 1996). Infants might also set up an enormous correlation matrix in which they record all of the behaviors associated with words; in that case, categories are the result of children noticing that certain behaviors tend to be correlated. Thus, having noticed that certain words often end in *-ing, -ed,* or *-s,* frequently occur in the middle of sentences, and rarely appear in the beginning of a sentence, children sort out these words as a verb category (see Maratsos and Chalkley, 1981). The problem with the notion of a simple, unconstrained, and unbiased correlational learner is the infinite number of correlations that children must consider, most of which will never appear in any language (Pinker, 1984, 1987). If infants are born "knowing" that, in language, objects are expressed by nouns, physical actions by verbs, and attributes by adjectives, infants could infer that words referring to physical objects are nouns, words referring to actions are verbs, and words referring to attributes are adjectives. They could learn the properties of nouns and verbs from these semantically prototypical cases, a process often referred to as "semantic bootstrapping" (see Pinker, 1984, 1987).

ACQUISITION OF AUXILIARY AND LEXICAL VERBS

The paradox of syntax acquisition is this: Unless children basically know what they have to learn before they begin, they cannot successfully learn the grammar of their language. However, even if it is demonstrated that children do indeed have innate mechanisms for learning the categorical membership of words, it is possible that such mechanisms are not specifically linguistic (for one such proposal, see Elman et al., 1996). To examine this proposition, we can look at the acquisition of auxiliary verbs and lexical verbs.

The acquisition of English auxiliary and lexical verbs is a particularly good test case because the two types of verbs are semantically, syntactically, and lexically similar; that is, a learner who has no knowledge of auxiliary and lexical verbs (i.e., a simple, unbiased correlational learner) is almost certain to confuse the two types of verbs. For many auxiliaries there is a lexical verb counterpart with an extremely similar meaning-e.g., the pairs *can/is able to, wilV is going to,* and *must/have to). Auxi*liary and lexical verbs are syntactically similar in that both types often take verbal endings, follow subject noun phrases, and lack the grammatical properties of nouns, adjectives, and other syntactic categories. Moreover, auxiliary and lexical verbs typically have identical forms (e.g., copula and auxiliary forms of *be,* possessive and auxiliary forms of *have,* lexical verb and auxiliary forms of *do).* The remarkable degree of similarity can be appreciated by comparing pairs of sentences such as *he is sleepy* and *he is sleeping, he has cookies* and *he has eaten cookies,* and *he does windows* and *he does not do windows.*

The syntactic and morphological behavior of auxiliaries is extremely complex, and there are no obvious nonlinguistic correlates for this behavior to aid in learning (Stromswold, 1990a). Without innate, specifically linguistic mechanisms, how could children correctly identify the 99 unique strings of auxiliaries that are acceptable in English from among -23! (2.59×10^{22}) unique strings of English auxiliaries?[4] Descriptively, the basic restrictions on auxiliaries can be summarized as follows:

AUX --->

(Modal) *(have -en)* (progressive *be -ing* (passive *be -en)*

Any or all of the auxiliaries are optional, but if they are present, they must occur in the above order. In addition, each auxiliary requires that the succeeding verb be of a certain form. Modal auxiliaries (e.g., *can, wil4 might)* require that the succeeding verb be an infinitival form (e.g., *eat),* perfect *have* requires that the succeeding verb be a perfect participle (e.g., *eaten),* progressive *be* requires that the succeeding verb be a progressive participle (e.g., *eating),* and passive *be* requires that the main verb be a passive participle (e.g., *eaten).* In addition, the first verbal element must be tensed in a matrix clause. Finally, matrix questions and negative statements are formed by inverting or negating the first auxiliary. If no auxiliary is . present, do-support is required (see Stromswold, 1990a, and Stromswold, 1992, for additional restrictions and complications). Lexical and auxiliary verbs pose a serious learnability question (Baker, 1981; Pinker, 1984; Stromswold, 1989a, 1990a, 1992): How can children distinguish between auxiliary and lexical verbs before they learn the behavior of the two types of verbs, and how do children learn the two types of verbs' behaviors before they can distinguish between them?

If children don't distinguish between auxiliary and lexical verbs, they will generalize what they learn about one type of verb to the other type of verb. This will result in rapid learning. It will also lead children to make errors that can be set right only by negative evidence (information that a particular construction is ungrammatical). Unfortunately, parents don't seem to provide usable negative evidence (Brown and Hanlon, 1970; Marcus, 1993). Thus, if children do not distinguish between auxiliaries and lexical verbs, they are destined to make certain types of inflectional errors (e.g., **I aming go, *I musts eat)* and combination errors involving multiple lexical verbs (e.g., **I hope go Disneyland),* negated lexical verbs (e.g., **I eat not cookies),* lone auxiliaries (e.g., **I must cqfee),* and unacceptable combinations of auxiliaries (e.g., **I may should go).* They will also make word order errors, scrambling the order of lexical verbs and auxiliaries (e.g., **Igo must),* scrambling the order of auxiliaries (e.g., **He have must gone),* and incorrectly inverting lexical verbs (e.g., *eats he meat?).* If, on the other hand, children have innate predispositions that allow them to distinguish between auxiliary and lexical verbs, they will not make these errors.

In order to test whether English-speaking children distinguish between auxiliary and lexical verbs, I searched the transcripts of 14 children's speech, examining by hand more than 66,000 utterances that contained auxiliaries (Stromswold, 1989a, 1990a). I found that the children acquired the auxiliary system with remarkable speed and accuracy. In fact, I found no clear examples of the types of inflectional errors, combination errors, or word order errors they would have made if they confused auxiliary and lexical verbs. Thus, children seem to have innate, specifically linguistic mechanisms that allow them to distinguish between auxiliary and lexical verbs.

ERRORS, INSTRUCTION, AND THE AUTOMATICITY OF LANGUAGE One of the hallmarks of innate abilities is that they can be acquired without explicit instruction. This seems to be true for language. Parents do correct their children when they make errors that affect the meaning of utterances, but they do not reliably correct grammatical errors (Brown and Hanlon,. 1970; Marcus, 1993). And even when parents do try to correct grammatical errors, their efforts are often in vain (McNeill, 1966). Furthermore, correction is not necessary for lexical and syntactic acquisition because some children who are unable to speak (and hence cannot be corrected by their parents) have normal receptive language (Stromswold, 1994a). If teaching and correction are necessary for language development, it should not be possible for children to have impaired production and intact comprehension. I have studied the language acquisition of a young child who is unable to speak. Despite the fact that he had essentially no expressive language (he could say only a handful of phonemes), his receptive language was completely intact. At age 4, he was able to distinguish between reversible active and passive sentences (correctly distinguishing the meanings conveyed by sentences such as *The dog bit the cat, The cat bit the dog, The dog was bitten by the cat,* and *The cat was bitten by the dog)* and to make grammaticality judgments (e.g., correctly recognizing that *What can Cookie Monster eat?is* grammatical whereas **What Cookie Monster can eat?* is not) (see Stromswold, 1994a).

Children learn language quickly, never making certain types of errors that seem very reasonable (e.g., certain types of auxiliary errors). But as Pinker (1989) points out, children are not perfect: They do make certain types of errors. They overregularize inflectional endings, saying *eated* for *ate* and *mouses* for *mice* (Pinker, 1989). They make lexical errors, sometimes passivizing verbs such as *die* that do not passivize (e.g., *He get died,* from Pinker, 1989). They also make certain types of syntactic errors, such as. usAy, do-support when it is not required (e.g., *Does it be aiound it?* and *This doesn't be straight,* Stromswold, 1990b, 196 5) and failing to use *do*-support when it is required (e.g., *What she eats?* Strom swold, 1990a, 1994b). What do these errors tell us? First, they confirm that children use language productively and are not merely repeating what they hear their par-

ents say because parents do not use these unacceptable forms (Pinker, 1989). These errors may also provide an insight into the peculiarities of languages. For example, children's difficulty with do-support suggest that do-support is not part of universal grammar, but rather is a peculiar property of English (Stromswold, 1990a,b, 1994b).

Finally, these errors may provide insight into the types of linguistic categories that children are predisposed to acquire. Consider, for example, the finding that children overregularize lexical *be, do,* and *have,* but they never overregularize auxiliary *be, do,* and *have* (Stromswold, 1989a, 1990a, in press-a). The fact that children say sentences like **She beed happy* but not **She beed smiling* indicates that children not only distinguish between auxiliary verbs and lexical verbs, but they treat the two types of verbs differently. What kind of innate learning mechanism could result in children's overregularizing lexical verbs but not the homophonous auxiliaries? One possibility is that children have innate learning mechanisms that specifically cause them to treat auxiliary and lexical verbs differently. Unfortunately, there are. problems with this explanation. Although many languages contain words that are semantically and syntactically similar to English auxiliaries (Steele, 1981), and all languages are capable of making the semantic and syntactic distinctions that in English are made by auxiliaries, some languages either lack auxiliaries (instead making use of inflectional affixes) or make no distinction between auxiliaries and lexical verbs. Given that not all languages contain easily confused auxiliary verbs and lexical verbs, the existence of a specific innate mechanism for making this distinction seems unlikely. In addition, hypothesizing a specific innate mechanism has little explanatory power-it explains nothing beyond the phenomena that led us to propose its existence.

Alternatively, children's ability to distinguish between auxiliary and lexical verbs might reflect a more general ability to distinguish between functional categories (determiners, auxiliaries, nominal and verbal inflections, pronouns, etc.) and lexical categories (nouns, verbs, adjectives, etc.). Lexical categories are promiscuous: They freely admit new members *(fax, modem, emai4* etc.) and the grammatical behavior of one member of a lexical category can fairly safely be generalized. to another member of the same lexical category. Functional categories are conservative: New members are not welcome and generalizations, even within a functional category, are very dangerous (see Stromswold, 1990a, 1994c). Innate mechanisms that specifically predispose children to distinguish between lexical and functional categories have a number of advantages over a specific mechanism for auxiliary and lexical verbs. Unlike the auxiliary/lexical verb distinction, the lexical/functional category distinction is found in all human languages; thus, mechanisms that predispose children to distinguish between lexical categories and functional categories are better candidates, a priori, *for* being innate. In addition, research on speech errors (e.g., Garrett, 1976), neologisms (Stromswold, 1994c), parsing (e.g., Morgan and Newport, 1981), linguistic typology (e: g., Croft, 1990), aphasia. (e.g., Goodglass, 1976), and developmental language disorders (e.g., Guilfoyle, Allen, and Moss, 1991) as well as findings from event-related potentials (Neville, 1991; Holcomb, Coffey, and Neville, 1992; Neville et al., 1993; Neville, 1995; Neville, Mills, and Lawson, 1992) and functional magnetic resonance imaging (Neville et al., 1994) all point to the importance of the lexical/functional distinction.

. Innate mechanisms that predispose children to distinguish between lexical and functional categories would also help them to distinguish between auxiliary and lexical verbs, as well as pronouns and nouns, determiners and adjectives, verbal stems and verbal inflections, and other pairs of lexical and functional categories. If these innate mechanisms predispose children to distinguish between syntactic categories that allow for free generalization (lexical categories) and those that do not (functional categories), this would explain why children overregularize lexical *be, do,* and *have* but not auxiliary *be, do,* and *have.* It would also help explain why children are able to learn language so rapidly and with so few errors; that is, such a learning mechanism would permit children to generalize only where it is safe to do so (i.e., within a lexical category). Computationally, the difference between lexical and functional categories might be expressed as the difference between rule-based generalizations and lists, or within a connectionist framework, between network architectures that have different degrees and configurations of connectivity (see Stromswold, 1994c).

Role of linguistic input and critical periods in language acquisition

PIDGINS The uniformity of language development under normal conditions could be due to biological or environmental processes. One way to investigate the relative roles of biological and environmental factors is to investigate the linguistic abilities of children whose early language environments are suboptimal. Studies of 'creolization provide compelling evidence that human children are innately endowed with the ability to develop a very specific kind of language even when they receive minimal input. Creolization may occur, for example, when migrant workers who speak a variety of languages must work together and their only common language is a simplified pidgin of another, dominant language. Pidgins typically consist of fixed phrases and pantomimes and

can express only basic needs and ideas. Bickerton (1981, 1984), studying the language of second-generation pidgin speakers (i.e., the children of pidgin speakers), has found that they use a creolized language that is much richer than their_ parents' pidgin. For example, the creolized language of second-generation pidgin speakers includes embedded and relative clauses, aspectual distinctions, and consistent word order, despite the absence of such features in the input (pidgin) language (Bickerton, 1981, 1984). Thus, second-generation pidgin speakers "invent" a language that is more complex than the pidgin language to which they are exposed.

HOMESIGN How minimal can the input be? Although children who hear only pidgin languages have impoverished input, there are even more extreme situations of language deprivation. Consider deaf children born to hearing parents who do not use or expose their infants to sign language but otherwise provide normal care (i.e., their parents neither abuse nor neglect them). Such children are deaf isolates-they receive essentially no linguistic input. Deaf isolates offer us a fascinating picture of the limits of the innate endowment to create language, and hence a glimpse at the early unfolding of language in all infants. As infants and toddlers, deaf isolates seem to achieve the same early-language milestones as hearing children. Right on schedule, at around 6-8 months, deaf isolates begin to "babble"-they make hand motions analogous to the spoken babbling of hearing babies. They invent their first signs at about the same age that hearing children produce their first words. They even begin to form short phrases with these signs, also on a comparable schedule to hearing children (Goldin-Meadow and Mylander, 1984, 1998; Morford, 1996). Thus, these early linguistic milestones are apparently able to unfold even without linguistic input. Preliminary research on older deaf isolates indicates that their gesture communication systems are more sophisticated than those used by young deaf isolates, although even their systems do not exhibit the complexity of natural sign languages (Coppola et al., 1998).

The ability to learn language appears to be the result of innate processes; however, childhood language exposure is necessary for normal language development, just as the ability to see is innate but visual stimulation is necessary for normal visual development (Hubel and Wiesel, 1970). The hypothesis that exposure to language must occur by a certain age in order for language to be acquired normally is called the critical (or sensitive) period hypothesis. The critical period for language acquisition is generally believed to coincide with the period of great neural plasticity and is often thought to end at or sometime before the onset of puberty (see Lenneberg, 1967).

WILD CHILDREN Skuse (1984a,b) reviewed nine well-documented cases of children who had been raised under conditions of extreme social and linguistic deprivation for 2.5 to 12 years. All of these cases involved grossly impoverished environments, frequently accompanied with malnourishment and physical abuse. At the time of discovery, the children ranged in age from 2.5 years to 13.5 years, had essentially no receptive or expressive language, and were globally retarded in nonlinguistic domains. The six children who eventually acquired normal or near-normal language function were all discovered by age 7 and had no signs of brain damage. Of the three children who remained language-impaired, one was discovered at age 5 but had clear evidence of brain damage (Davis, 1940, 1947) and one was discovered at age 3.5 but had organic abnormalities not attributable to extreme deprivation (Skuse, 1984a). Genie, the third child with persistent linguistic impairments, is remarkable both for having the most prolonged period of deprivation (12 years) and, at almost 14 years of age, for being the oldest when discovered (Curtiss, 1977). Neuropsychological testing suggests that Genie does not have the expected left hemisphere lateralization for language. It is tempting to conclude that Genie's failure to acquire normal language and her anomalous lateralization of language function are both the result of her lack of exposure to language prior to the onset of puberty; however, it is possible that cortical anomalies in the left hemisphere are the cause of her anomalous lateralization and her failure to acquire language (Curtiss, 1977).

DEAF ISOLATES As Curtiss (1977, 1989) points out, it is impossible to be certain that the linguistic impairment observed in children such as Genie are the result of linguistic isolation, and not the result of social and physical deprivation and abuse. Curtiss (1989) has described the case of Chelsea, a hearing-impaired woman who had essentially no exposure to language until age 32. Unlike Genie, Chelsea did not experience any social or physical deprivation. Chelsea's ability to use language (particularly syntax) is at least as impaired as Genie's, an observation consistent with the critical period hypothesis (Curtiss 1989). To test whether there is a critical period for first language acquisition, Newport and colleagues (Newport, 1990) have studied the signing abilities of deaf people whose first exposure to American Sign Language (ASL) was at birth.(native signers), before age 6 (early signers), or after age 12 (late signers). Consistent with the critical pelftd hypothesis, even after 30 years of using ASL, on tests of morphology and complex syntax, native signers outperform early signers, who in turn outperform late signers . (Newport, 1990).

SECOND LANGUAGE ACQUISITION To test whether there is a critical period for second language acquisition, Johnson and Newport (1989) studied the English abilities of native speakers of Korean or Chinese who first became immersed in English between the ages of 3 and 39. For subjects who began to learn English before puberty, age of English immersion correlated extremely highly *with* proficiency with English syntax and morphology, whereas no significant correlation was found for subjects who began to learn English after puberty (Johnson and Newport, 1989).

Evidence from studies of children such as Genie, deaf isolates, and people who acquire a second language suggests that the ability to acquire language diminishes with age. Other research has shown that complete language recovery rarely occurs if a left hemisphere lesion occurs after age 5 and substantial recovery rarely occurs if a lesion is acquired after the onset of puberty. Moreover, subtle tests of linguistic abilities reveal that native fluency in a language is rarely attained if one's first exposure to that language occurs after early childhood and competence in a language is rarely attained if first exposure occurs after the onset of puberty. This is consistent with Hubel and Wiesel's (1970) finding that normal visual development requires visual stimuli during a critical period of neural development and suggests that neural fine-tuning is a critical to normal language acquisition-a fine-tuning that can occur only with exposure to language during a certain time period.

Language acquisition and brain development

We have argued that the ability to learn language is the result of innate, language-specific learning mechanisms. And we have investigated the extent to which normal language development depends on receiving appropriate linguistic input during a critical window of cognitive (and presumably neuronal) development. Here we review the neurobiological evidence supporting the idea that language is the result of innate, language-specific learning mechanisms.

DEVELOPMENT OF LANGUAGE REGIONS OF THE BRAIN Lenneberg (1967) notwithstanding, the language areas of the human brain appear to be anatomical and functionally asymmetrical at or before birth. Anatomically, analyses of fetal brains reveal that the temporal plane is larger in the left hemisphere than in the right hemisphere (Wada, Clarke, and Hamm, 1975). [5] Development of the cortical regions that subserve language in the left,hemisphere consistently lags behind the development of the homologous regions in the right hemisphere. The right temporal plane appears during the thirtieth gestational week, while the left temporal plane appears about 7-10 days later (Chi, Dooling, and Gilles, 1977). Even in infancy, dendritic development in the region around Broca's area on the left lags behind that found in the homologous region on the right (Scheibel, 198.4). Event-related potential (ERP) and dichotic listening experiments suggest that the left hemisphere is differentially sensitive for speech from birth (for a review, see Mehler and Christophe, 1995).

Relatively few studies have investigated- the neural bases of lexical or syntactic abilities in neurologically intact children. Among these is the work of Molfese and colleagues (Molfese, 1990; Molfese, Morse, and Peters, 1990), who taught infants as young as 14 months labels for novel objects, then compared the ERPs when the objects were paired with correct and incorrect verbal labels. A late-occurring response was recorded in the left hemisphere electrode sites when the correct label was given but not when an incorrect label was given. Similarly, an early-occurring response was recorded bilaterally in the frontal electrodes when the correct label was given, but not when an incorrect label was given. In recent work, Mills, Coffey-Corina, and Neville (1997) recorded the ERPs when children between 13 to. 20 months of age listened to words meanings they knew, words whose meanings they did not know, and backward words. They found that the ERPs differed as a function of meaning within 200 ms of word onset. Between 13 and 17 months, the ERP differences for known versus unknown words were bilateral and widely distributed over anterior and posterior regions. By 20 months, the differences were limited to left temporal and parietal regions.

In another ERP study, Holcomb, Coffey, and Neville (1992) found no clear evidence prior to age 13 of the normal adult pattern of greater negativity in the left hemisphere for semantically plausible sentences (e.g., *We baked cookies in the oven)* and greater negativity in the right hemisphere for semantically anomalous sentences (e.g., *Mother wears a ring on her school).* In addition, the negative peak associated with semantic anomalies (the N400) was later and longer in duration for younger subjects than older subjects. Holcomb, Coffey, and Neville (1992) also found evidence that the normal adult pattern of a left anterior N280 waveform associated with functional category words and a bilateral posterior N350 waveform associated with lexical category words (Neville, Mills, and Lawson, 1992) does not develop until around puberty. Four-year-old children typically have N350 response to both lexical and functional words. By 11 years of age, the N350 is greatly reduced or absent for functional category words. It isn't until approximately 15 years of age that functional category words result in a clear N280 response with adult-like distribution

(Holcomb, Coffey, and Neville, 1992). In summary, simple linguistic stimuli (e.g., lexical words) appear to evoke similar types of electrical activity in young children's and adult brains; but for more complicated linguistic stimuli involving grammatical aspects of language, children's ERPs may not closely resemble adult ERPs until around puberty. That the critical period for language acquisition (especially syntax) ends at approximately the same age that children develop adult-like ERPs for grammatical aspects of language is intriguing. It is also suggestive, raising the possibility that once adult-like neural pathways and operations are acquired, neural plasticity is so greatly reduced that the ability to acquire all but the most rudimentary aspects of syntax is lost.

MODULARITY OF LANGUAGE ACQUISITION With some notable exceptions, most of what is known about the relationship between brain development and lexical and syntactic development has come from studying language acquisition by children who have developmental syndromes or brain lesions. If, as was argued earlier, language acquisition involves the development of specialized structures and operations having no counterparts in nonlinguistic domains, then it should be possible for a child to be cognitively intact and linguistically impaired or to be linguistically intact and cognitively impaired. But if language acquisition involves the development of the same general symbolic structures and operations used in other cognitive domains, then dissociation of language and general cognitive development should be impossible. Recent studies suggest that language development is selectively impaired in some children with specific language impairment (SLI) and selectively spared in children who suffer from disorders such as Williams syndrome.

Specific language impairment SLI encompasses developmental disorders characterized by severe deficits in the production and/or comprehension of language that cannot be explained by hearing loss, mental retardation, motor deficits, neurological or psychiatric disorders, or lack of exposure to language. Because SLI is a diagnosis of exclusion, SLI children are a very heterogeneous group. This heterogeneity can and does affect the outcome of behavioral and neurological studies, with different studies of SLI children frequently reporting different results depending on how SLI subjects were chosen. The exact nature of the etiology of SLI remains uncertain (for a review, see Leonard, 1998; Stromswold, 1997), with proposals including impoverished or deviant linguistic input (Cramblit and Siegel, 1977; Lasky and Klopp,1982), transient, fluctuating hearing loss (Bishop and Edmundson, 1986; Gordon, 1988; Gravel and Wallace, 1992; Teele et al., 1990), impairment in short-term auditory memory (Graham, 1968, 1974; Rapin and Wilson, 1978), impairment in auditory sequencing (Efron, 1963; Monsee, 1961), impairment in rapid auditory processing (Tallal and Piercy, 1973a,b, 1974), general impairment in sequencing (Poppen et al., 1969), general impairment in rapid sensory processing (Tallal, 1990), general impairment in representational or symbolic reasoning (Johnston and Weismer, 1983; Kahmi, 1981; Morehead and Ingrain, 1973), general impairment in hierarchical planning (Cromer, 1983), impairments in language perception or processing [e.g., the inability to acquire aspects of language that are not phonologically salient (Leonard, 1989, 1994; Leonard, McGregor, and Allen, 1992)], impairments in underlying grammar [e.g., the lack of linguistic features such as tense and number (Crago and Gopnik, 1994; Gopnik, 1990a,b; Gopnik and Crago, 1991), the inability to use government to analyze certain types of syntactic relations (van der Lely, 1994), and the inability to form certain types of agreement relations (Clahsen, 1989, 1991; Rice, 1994)], or some combination thereof. Some researchers have even suggested that SLI is not a distinct clinical entity, and that SLI children just represent the low end of the normal continuum in linguistic ability (Johnston, 1991; Leonard, 1991).

At the neural level, the cause of SLI is also uncertain. Initially, it was theorized that children with SLI had bilateral damage to the perisylvian cortical regions that subserve language in adults (Bishop, 1987). Because SLI is not a fatal disorder and people with SLI have normal life spans, to date, only one brain of a possible SLI child has come to autopsy. Post-mortem examination of this brain revealed atypical symmetry of the temporal planes and a dysplastic microgyrus on the interior surface of the left frontal cortex along the inferior surface of the sylvian fissure (Cohen, Campbell, and Yaghmai, 1989), findings similar to those reported in dyslexic brains by Geschwind and Galaburda (1987). It is tempting to use the results of this autopsy to argue-as Geschwind and Galaburda (1987) have for dyslexia-that SLI is the result of subtle anomalies in the left perisylvian cortex. However, the child whose brain was autopsied had a performance IQ of just 74 (verbal IQ 70); hence the anomalies noted on autopsy may be related to the child's general cognitive impairment rather thaq to her language impairment.

Computed tomograp f (CT) and magnetic resonance imaging (MRI) scans of SLI children have failed to reveal the types of gross perisyl~an lesionitypically found in patients with acquired aphasia (Jernigan et al., 1991; Plante et al., 1991). But CT and MRI scans have revealed vealed that the brains of SLI children often fail to exhibit the normal pattern in which the left temporal plane

is larger than the right (Jernigan et al., 1991; Plante, 1991; Plante, Swishes, and Vance, 1989; Plante et al., 1991). Examinations of MRI scans have revealed 'that dyslexics are more likely to have additional gyri between the postcentral sulcus and the supramarginal gyrus than are normal readers (Leonard et al., 1993). Jackson and Plante (1997) recently performed the same type of gyral morphology analyses on MRI scans of 10 SLI children, their parents, 10 siblings, and 20 adult controls.[6] For the control group, 23% of the hemispheres showed an intermediate gyrus, whereas 41% of the hemispheres for SLI family members (probands, their siblings, and parents combined) showed an intermediate gyrus. However, affected family members. did not appear to be more likely to have an intermediate gyrus than unaffected members. Clark and Plante (1995) compared the morphology of Broca's area in parents of SLI children and adult controls. Overall, parents of SLI children were no more likely to have an extra sulcus in the vicinity of Broca's area. However, parents with docu=mented language impairments were more likely to have an extra sulcus than unaffected parents.

A number of researchers have studied the functional characteristics of SLI children's brains. Data from dichotic listening experiments (e.g., Arnold and Schwartz, 1983; Boliek, Bryden, and Obrzut, 1988; Cohen et al., 1991) and ERP experiments (e.g., Dawson et al., 1989), suggest that at least some SLI children have aberrant functional lateralization for language, with language present either bilaterally or predominantly in the right hemisphere. Single photon emission computed tomography (SPECT) studies of normal and language-impaired children have revealed hypoperfusion in the inferior frontal convolution of the left hemisphere (including Broca's area) in two children with isolated expressive language impairment (Denays et al., 1989), hypoperfusion of the left temporoparietal region and the upper and middle regions of the right frontal lobe in nine of twelve children with expressive and receptive language impairment (Denays et al., 1989), and hypoperfusion in the left temporofrontal region of language-impaired children's brains (Lou, Henriksen, and Bruhn,. 1990).

Courchesne and colleagues (1989) did not find any differences in ERP amplitude or latency between SLI adolescents and adults and age-matched controls. But in a subsequent study of school-age SLI children (Lincoln et al., 1995), they found that normal age-matched controls exhibited the normal pattern of larger amplitude N 100s for more intense auditory stimuli intensity, while SLI subjects did not exhibit that pattern. This finding suggests. the possibility of some abnormality in the auditory cortex of S.LI children. Neville and colleagues (1993) compared the ERPs of SLI children and normal age-matched controls for three tasks. In the first task, subjects pressed a button when they detected 1000-Hz tones among a series of 2000-Hz tones. In the second task, subjects were asked to detect small white rectangles among a series of large red squares. In the third task,, children read sentences one word at a time and judged whether or not the sentences were semantically plausible (half of the sentences ended with a semantically appropriate word and half ended with a semantically inappropriate word). Overall, for the auditory monitoring task, the SLI children's ERPs did not differ from those of the control children. However, when the SLI children were divided into groups according to their performance on Tallal and Piercy's (1973a,b) auditory processing task, children who performed poorly on that task exhibited reduced-amplitude ERP waves over the anterior portion of the right hemisphere together with greater latency for the N140 component. In general, the SLI children had abnormally large N400s on the sentence task. As is typically seen with adults, the normal children's N400s for closed-class words were larger over the anterior left hemisphere than the anterior right hemisphere. However, the SLI children with the greatest morphosyntactic deficits did not exhibit this asymmetry.[7]

Despite decades of intensive and productive research on SLI, a number of fundamental questions about SLI remain unanswered. Researchers disagree about the etiology of SLI at a neural or cognitive level, and offer proposals ranging from a specific impairment in a circumscribed aspect of abstract linguistics to general cognitive/processing impairments due to environmental causes. Even among researchers who believe that SLI specifically affects linguistic competence, there is disagreement about what aspect of the underlying grammar is impaired. Furthermore, numerous studies have revealed that many (if not most) children with SLI exhibit nonlinguistic deficits, although some researchers argue that these nonlinguistic deficits are secondary to their primary linguistic impairments (for a review, see Leonard, 1998). A first step in seeking answers to these questions is to study more homogeneous subgroups of children diagnosed with SLI.[B] In summary, although generally consistent with the hypothesis of a specific module for language and language acquisition, the emerging picture of SLI is not as "clean" as modularists might hope: SLI children are a heterogeneous group, and many (perhaps all) are not perfectly intact but for a damaged language module.

Williams syndrome Although mental retardation generally results in depression of language function (Rondal, 1980), researchers have reported that some mentally

retarded children have remarkably intact language. This condition has been reported in some children with hydrocephalus (Swisher and Pinsker, 1971), Turner's syndrome (Yamada and Curtiss, 1981), infantile hypercalcemia or Williams syndrome (Bellugi et al., 1992), and mental retardation of unknown etiology (Yamada, 1990).

Williams syndrome (WS) is a rare (1 in 25,000) genetic disorder involving deletion of portions of chromosome 7 around and including the elastin gene (Ewart et al., 1993a,b). People with WS often have particularly extreme dissociation of language and cognitive functions (Bellugi et al., 1992). Hallmarks of WS include microcephaly with a "pixie-like" facial appearance, general mental retardation with IQs typically in the 40s and 50s, delayed onset of expressive language, and "an unusual command of language combined with an unexpectedly polite, open and gentle manner" by early adolescence (Von Arman and Engel, 1964). In a recent study, the MacArthur Communicative Development Inventory (a parental report measure) was used to assess the earliest stages of language development for children with WS and children with Down syndrome (DS). This study revealed that WS and DS children were equally delayed in the acquisition of words, with an average delay of 2 years for both groups (Singer Harris et al., 1997). WS and DS children who had begun to combine words (mean age 46 months) did not differ significantly in language age (mean ages 23.7 months and 21 months, respectively). However, compared to the DS children, these older WS children had significantly higher scores on grammatical complexity measures and on mean length of utterance for their longest three sentences (Singer Harris et al., 1997). The gap in linguistic abilities of WS and DS children increases with age (Bellugi, Wang, and Jernigan, 1994). Although adolescents with WS use language that is often deviant for their chronological age and do poorly on many standardized language tests, they have larger vocabularies than do children of equivalent mental ages and speak in sentences that are syntactically and morphologically more complex and well-formed. In addition, WS adolescents and adults demonstrate good metalinguistic skills, such as the ability to recognize an utterance as ungrammatical and to respond in a contextually appropriate manner (Bellugi et al., 1992).

Volumetric analyses of MRI scans indicate that compared to normal brains, cerebral volume and cerebral gray matter of WS brains are significantly reduced in size and the neocerebellarr vermal lobules are increased in size, with paleocerebellar vermal regions of low-normal size (Jernigan and Bellugi, 1994; Jernigan et al., 1993; Wang et al., 1992). To date only one WS brain has come to autopsy (Galaburda et al., 1994). This brain had extensive cytoarchitectural abnormalities, including exaggeration of horizontal abnormalities within layers (most striking in area 17 of the occipital lobe), increased cell density throughout the brain, and abnormally clustered and oriented neurons. In addition, although the frontal lobes and most of the temporal lobes were relatively normal in size, the posterior forebrain was much smaller than normal. Galaburda and colleagues interpreted these findings as evidence of developmental arrest between the end of the second trimester and the second year of life. They further suggested that these findings may be related to hypercalcemia found in WS. Alternatively, elastin may have a direct, but hitherto undiscovered, neurodevelopmental function, in which case the macroscopic and microscopic abnormalities may be associated with the decreased levels of elastin in WS.

Early studies revealed that, although auditory ERPs for WS adolescents are similar in morphology, distribution, sequence, and latency to those of age-matched controls, WS adolescents display large-amplitude responses even at short interstimulus intervals, suggesting hyperexcitability of auditory mechanisms at the cortical level with shorter refractory periods (Neville, Holcomb, and Mills, 1989; Neville, Mills, and Bellugi, 1994). When WS subjects listened to spoken words, their ERPs had grossly abnormal morphology not seen in normal children at any age (Neville, Mills, and Bellugi, 1994). In contrast, the morphology of their ERPs for visually presented words was normal. Compared with normal subjects, WS subjects had larger priming effects for auditorily presented words, but priming effects for visually presented words were normal or smaller than those observed for normal subjects (Neville, Mills, and Bellugi, 1994). These results suggest that WS subjects' relative sparing of language function is related to hypersensitivity to auditorily presented linguistic material. To date no PET or SPECT studies of WS children have been reported. It will be interesting to learn from such studies whether it is the classically defined language areas in general or just primary auditory cortex in WS brains that become hyperperfused in response to auditory linguistic stimuli. In summary, although generally, consistent with the hypothesis of specific module for language and language acquisition, the emerging picture of WS is not as "clean" as modularists Ynight hope: Although WS adolescents and adults have bet~+er linguistic. abilities than others with comparable IQs, tl ' language is far from perfect and the mechanisms they use for language acquisition may not be the same as those used by normal children (see, for example, Karmiloff-Smith et al., 1997, 1998; Stevens and Karmiloff-Smith, 1997).

GENETIC BASIS OF . LANGUAGE If the acquisition of language is the result of specialized structures in the brain and these linguistically specific structures are coded for by information contained in the genetic code, one might expect to find evidence for the heritability of language (see Pinker and Bloom, .1990; Ganger and Stromswold, 1998). But if language acquisition is essentially the result of instruction and involves no specifically linguistic structures, one should find no evidence of genetic transmission of language.

Familial aggregation studies A comprehensive review of family aggregation studies, sex ratio studies, pedigree studies, commingling studies, and segregation studies of spoken language disorders reveals that spoken language disorders have a strong tendency to aggregate in families (Stromswold, 1998). Stromswold (1998) reviewed 18 family aggregation studies of spoken language impairment (see table 63.1). In all seven studies that collected data for both probands and controls, the incidence of positive family history was significantly greater for probands than controls.' In these seven studies, the reported incidence of positive family history for probands ranged from 24% (Bishop and Edmundson, 1986) to 78% (van der Lely and Stollwerck, 1996), with a mean incidence of 46% and a median incidence of 35%. [10] For controls, positive family history rates ranged from 3%. (Bishop and Edmundson, 1986) to 46% (Tallal, Ross, and Curtiss, 1989a), with a mean incidence of 18% and a median incidence of 11%.

Of all the studies of family aggregation Stromswold (1998) reviewed, eleven reported the percentage of probands' relatives who were impaired. For probands, the percentage of family members who were impaired ranged from 20% (Neils and Aram, 1986) to 42% (Tallal, Ross, and Curtiss, 1989a), with a mean impairment rate of 28% and median impairment rate of 26%. For controls, the percentage of family members who were impaired ranged from 3% (Neils and Aram, 1986) to 19% (Tallal, Ross, and Curtiss, 1989a), with a mean impairment rate of 9% and a median impairment rate of 7%. The incidence of impairment was significantly higher among proband relatives than control relatives in seven of the eight studies that made such a comparison.

Although data on familial aggregation suggest that some developmental language disorders have a genetic component, it is possible that children with language-impaired parents or siblings are more likely to be linguistically impaired themselves because they are exposed to deviant language (the deviant linguistic environment hypothesis, DLEH). Some studies have reported that mothers are more likely to use directive speech and less likely to use responsive speech when talking to their language-impaired children than are mothers speaking to normal children (e.g., Conti-Ramsden and Friel-Patti, 1983; Conti-Ramsden and Dykins, 1991). However, children's language impairments may cause mothers to use simplified speech, rather than vice versa. That is, mothers of language-impaired children may use directive speech because they cannot understand their impaired children and their impaired children do not understand them if they use more complicated language. Furthermore, although within a fairly wide range, linguistic environment may have little or no effect on language acquisition by normal children (e.g., Heath, 1983), genetics and environment may exert a synergistic effect in children who are genetically at risk for developing language disorders. Such children may be particularly sensitive to subtly impoverished linguistic environments.

Despite the DLEH prediction that the most severely impaired children should come from families with the highest incidence of language impairments, Byrne, Willerman, and Ashmore (1974) found that children with profound language impairments were less likely to have positive family histories of language impairment than children with moderate language impairments. Similarly, Tallal and colleagues (1991) found no differences in the language abilities of children who did and did not have a positive family history of language disorders. According to the DLEH, the deficits exhibited by language-impaired children result from "copying" the ungrammatical language of their parents. Therefore, the DLEH predicts that language-impaired children should have the same type of impairment as that of their relatives. However, Neils and Aram (1986) found that 38% of parents with a history of a speech and language disorder said that their disorder differed from their children's disorder. According to the DLEH, parents with a history of spoken language impairment who are no longer impaired should be no more likely to have language-impaired children than parents with no such history. But Neils and Aram (1986) found that a third of the probands' parents who had a history of a spoken language disorder did not suffer from the disorder as adults. The DLEH predicts that all children with SLI should have at least one close relative with a language impairment; however, in the studies reviewed, an average of 58% of thee language-impaired children had no first-degree relatives with impairments. If the DLEH is correct, birth order might affect the likelihood that a child will exhibit a language disorder. But birth order apparently affects neither the severity nor the likelihood of developing language disorders (see Tomblin, Hardy, and Hein, 1991). In our society, mothers typically have the primary responsibility for child-rearing; hence the DLEH predicts that the correlation of language status

TABLE 63.1

Family aggregation studies of spoken language disorders

Study	Sample Size	Other Family Diagnoses	Positive Family History	Frequency of Impairment among Relatives (Proband vs. Control)
Ingrain (1959)	75 probands	None	24% parental history 32% sibling history	N/A
Luchsinger (1970)	127 probands	None	36% probands	N/A
Byrne, Willerman, & Ashmore (1974)	18 severely impaired 20 moderately impaired	None	17% "severe" probands 55% "moderate" probands**	N/A
Neils & Aram (1986)	74 probands 36 controls	Dyslexia Stuttering Articulation	46% 1st-degree proband 8% 1st-degree controls****	20% vs. 3% all relatives **'
Bishop & Edmundson (1987)	34 probands 131 controls	None (for strict criteria)	24% 1st-degree proband 3% 1st-degree control****	N/A
Lewis, Ekelman, & Aram (1989)	20 probands 20 controls	Dyslexia Stuttering LD	N/A	Any: 12% vs. 2% all relatives**** 26% vs. 5% 1st-degree relatives' SLI: 9% vs. 1% all relatives**"
Tallal, Ross, & Curtiss (1989a)	62 probands 50 controls	Dyslexia LD School problems	77% 1st-degree proband 46% 1st-degree control**	42% vs. 19% 1st-degree relatives"*
Tomblin (1989)	51 probands 136 controls	Stuttering Articulation	53% 1st-degree probands Controls: N/A	23% vs. 3% 1st-degree relatives****
Haynes & Naido (1991)	156 probands	None	54% all probands 41% 1st-degree probands	28% proband parents 18% proband sibs
Tomblin, Hardy, & Hein (1991)	55 probands 607 controls	None	35% probands 17% controls***	N/A
Whitehurst et al. (1991)	62 probands 55 controls	Speech Late talker School problems	N/A	Any: 24% vs. 16% 1st-degree relatives Speech: 12% vs. 8% 1st-degree relatives Late-talker: 12% vs. 7% 1st-degree relatives School probs.: 7% vs. 5% 1st-degree relatives
Beitchman, Hood, & Inglis (1992)	136 probands 138 controls	Dyslexia LD Articulation	47% vs 28% all relatives *** 34% vs. 11% 1st-degree ****	Multiple affected relatives: 19% vs. 9%*
Lewis (1992)	87 probands 79 controls	Dyslexia LD Stuttering Hearing loss	N/A	LI: 15% vs. 2% all relatives**** 32% vs. 5% 1st-degree relatives***" I) exia: 3% vs. 1% all relatives"** 6^0 vs. 3% 1st-degree relatives LD: 3% v~s 1% all relatives**" 6% vs. ~'l0 1st-degree relatives*

TABLE 63.1 *Continued*

Study	Sample Size	Other. Family . Diagnoses	Positive Family History	Frequency of Impairment among Relatives (Proband vs. Control)
Tomblin & Buckwalter (1994)	26 probands	None	42% 1st-degree	Overall: 21% Mother: 15% Father: 40% Sister: 6%, Brother: 24%
Lahey & Edwards (1995)	53 probands	Learning problems	60% 1st-degree	Overall: 26% Mother: 26% Father: 22% Siblings: 29%
Rice, Rice, & Wexler (1996)	31 probands 67 controls	Reading Spelling Learning	N/A	Any: 18% vs. 9% all relatives`** 26% vs. 13% 1st-degree relatives `! LI: 15% vs. 6% all relatives`** 22% vs. 7% 1st-degree relatives*** Other: 7% vs. 5% all relatives 12% vs. 9% 1st-degree relatives
Tomblin (1996)	534 probands 6684 controls		29% probands 11% controls****	
van der Lely & Stollwerck (1996),	9 probands 49 controls	Reading or writing	78% 1st-degree probands 29% 1st-degree controls **	Overall: 39% *vs.* 9% **** Mothers: 33% vs. 2% *`* Fathers: 38% vs. 8%* Sisters: 40% vs. 8%* Brothers: 44% vs. 19%

* $p < .05$; ** $p < .01$; *** $p < .001$, ****$p < .0001$; Significance tests are for one-tailed tests.
SLI = Specific Language Impairment, LI = Speech or Language Impairment; LD = Learning Disability.
Adapted from Stromswold (1998).

should be greatest between mother and child. Contrary to this prediction, Tomblin (1989) found that among the family relations he studied (i.e., mother-child, father-child, male sibling-child, female sibling-child), the relationship was weakest between mother and child. Other studies that measured the ratio of impaired fathers to impaired mothers also report contra-DLEH results: The father:mother ratio has been reported as 2.7:1 (Tomblin and Buckwalter, 1994), 1.4:1 (Neils and Aram, 1986), and approximately 1:1 (Tallal, Ross, and Curtiss, 1989b; Whitehurst et al., 1991; Lewis, 1992).

Twin studies The influences of environmental and genetic factors on language disorders can be teased apart by comparing the concordance rates for language impairment in. monozygotic (MZ) and dizygotic (DZ) twins. MZ and DZ twins share the same pre- and postnatal environment. Thus, if the concordance rate for a particular trait is greater for MZ than DZ twins, it probably reflects the fact that MZ twins share 100% of their genetic material while DZ twins share, on average, just 50% of their genetic material (for a review, see Eldridge, 1983). Stromswold (1996, in press-b) reviewed five studies that examined the concordance rates for written language disorders and four studies that examined the concordance rates for spoken language disorders (see tables 63.2 and 63.3). In all nine studies, the concordance rates for MZ twin pairs were greater than those for DZ twin pairs, with the differences being significant in all but one study (Stevenson et al., 1987). In these studies concordance rates ranged from 100% (Zerbin-Rudin, 1967) to 33% (Stevenson et al., 1987) for MZ twins, and from 61% (Tomblin and Buckwalter, 1995) to 29% (Stevenson et al., 1987) for DZ twins."

The studies Stromswold reviewed included 212 MZ and 199 DZ twins pairs in which at least one member of the pair had a written language disorder, for concordance rates of 74.9% for MZ twins and 42.7% for DZ twins ($z = 6.53$, $p < .00000005$). The studies included 188 MZ and 94 DZ twin pairs in which at least one member of the twin pair had a spoken language disorder, for concordance rates of 84.3% for MZ twins and 52.0% for DZ twins ($z = 5.14$, $p < .00000025$). Overall, the studies included 400 MZ twin pairs and 293 DZ twin pairs, for concordance rates of 79.5% for MZ twins and 45.8% for DZ twins ($z = 8.77$, $p < .00000005$).

The finding that concordance rates were significantly greater for MZ than for DZ twins indicates that genetic factors play a significant role in the development of language disorders. The overall concordance rates for written and spoken language disorders are reasonably similar, with

TABLE 63.2

Concordance rates for twins with written language disorders

Study	Twin Pairs	Diagnosis	Proband Concordance
Zerbin-Rubin (1967)	17 MZ 33 DZ	Word blindness	100% MZ vs. 50% DZ***
Bakwin (1973)	31 MZ 31 DZ	Dyslexia	Overall: 91% MZ vs. 45% DZ*** Male: 91% MZ vs. 59% DZ* Female: 91% MZ vs. 15% DZ****
Matheny, Dolan, & Wilson (1976)	17 MZ 10 DZ	Dyslexia or academic problems	86% MZ vs. 33% DZ
Stevenson et al. (1987)	18 MZ t 30 DZt	Reading and spelling retardation (Neale & Schonell tests)	Neale reading: 33% MZ vs. 29% DZ Schonell reading: 35% MZ vs. 31% D2 Spelling: 50% MZ vs. 33% DZ
DeFries & Gillis (1993)	133 MZ 98 DZ	Dyslexia (PIAT scores)	66% MZ vs. 43% DZ***
Overalltt	212 MZ 199 DZ		74.9% MZ vs. 42.7% DZ****

Significance tests are one-tailed tests comparing concordance rates for MZ and DZ twins: * $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$.

t Number of pairs of twins varied according to diagnosis.

tt Overall rates include data for Stevenson and colleagues' "Schonell reading retarded" group.

Tests: PIAT (Peabody Individual Achievement Test); Word Recognition Reading (Dunn and Markwardt, 1970); Schonell Reading and Spelling Tests (Schonell and Schonell, 1960); Neale Reading Test (Neale, 1967).

Adapted from Stromswold (in press-b).

TABLE 63.3

Concordance rates for twins with spoken language disorders

Study	Twin Pairs	Diagnosis	Proband Concordance
Lewis & Thompson (1992)	32 MZt 25 DZt	Received speech or language therapy	Any disorder: 86% MZ vs. 48% DZ** Articulation: 98% MZ vs. 36% DZ**** LD: 70% MZ vs. 50% DZ Delayed speech: 83% MZ vs. 0% DZ*
Tomblin & Buckwalter (1994)	56 MZ 26 DZ	SLI (questionnaire to speech pathologists)	89% MZ vs. 55% DZ**
Bishop, North, & Dolan (1995)	63 MZ 27 DZ	SLI (by test scores)	Strict criteria: 70% MZ vs. 46% DZ* Broad criteria: 94% vs. 62% DZ**
Tomblin & Buckwalter (1995)	37 MZ 16 DZ	SLI (composite score >1 SD below mean)	96% MZ vs. 61% DZ**
Overallt	188 MZ 94 DZ		84.3% MZ vs. 52.0% DZ ****

Significance tests are one-tailed tests:* $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$.

LD = Learning Disorder.

t Overall rates include data for Lewis and Thompson's "any diagnosis" group and Bishop and coll ues' strict criterid group.

Adapted from Stromswold (in press-b).

concordance rates for spoken language disorders being approximately 10 percentage points higher than the rates for written language disorders. However, the fact that the difference between MZ and DZ concordance rates *was* very similar for written and spoken language disorders is consistent with the hypothesis that genetic factors play an equal role in both types of impairments.

Modes of transmission In a recent review of behavioral genetic studies of spoken language disorders, Stromswold (1998) concluded that most familial language disorders are the product of complex interactions between genetics and environment. In rare cases, however, language disorders may have a single major locus. For example, researchers have reported a number of kindred with extremely large numbers of severely affected family members (e.g., Arnold, 1961; Gopnik, 1990; Hurst et al., 1990; Lewis, 1990) *in* which transmission seems to be autosomal-dominant with variable rates of expressivity and penetrance. When Samples and Lane (1985) performed a similar analysis on a family in which six of six siblings had a severe developmental language disorder, they concluded that the mode of transmission in that family was a single autosomal recessive gene. If there are multiple modes of transmission for SLI, as the above results seem to indicate, SLI is probably genetically heterogeneous, just as dyslexia appears to be genetically heterogeneous.

The final-and most definitive-method for determining whether there is a genetic basis for familial language disorders is to determine which gene (or genes) is responsible for the language disorders found in these families. Typically, this is done by using linkage analysis techniques to compare the genetic material of language-impaired and normal family members, thereby allowing researchers to determine how the genetic material of affected family members differs from that of unaffected members. Linkage analyses of dyslexic families suggest that written language disorders are genetically heterogeneous (Bisgaard et al., 1987; Smith et al., 1986), with different studies revealing involvement of chromosome 15 (Smith et al., 1983; Pennington and Smith, 1988), the HLA region of chromosome 6 (Rabin et al., 1993), and the Rh region of chromosome 1 (Rabin et al., 1993). Froster and colleagues (1993) have reported a case of familial speech retardation and dyslexia that appears to be caused by a balanced translocation of the short arm of chromosome I and the long arm of chromosome 2. Recently, Fisher and colleagues (1998) conducted the first linkage analyses for spoken language disorders, performing genome-wide analyses of the genetic material of the three-generation family studied by Gopnik (1990a) and Hurst and colleagues (1990). They determined that the impairments exhibited by members of this family are linked to a small region on the long arm of chromosome. *7,* confirming autosomal dominant transmission with near 100% penetrance. However, it is important to note that in addition to the grammatical deficits described by Gopnik (1990a), affected members of this family also suffer from orafacial dyspraxia and associated speech disorders (see Hurst et al., 1990; Fisher et al., 1998). We cannot, therefore, conclude that the identified region of chromosome 7 necessarily contains a gene or genes specific to language. Clearly, linkage studies must be performed on other families whose deficits are more circumscribed.

At least three distinct relationships could obtain between genotypes and behavioral phenotypes. It is possible (albeit unlikely) that there is a one-to-one relationship between genotypes and phenotypes, with each genotype causing a distinct type of language disorder. Alternatively, there might be a one-to-many mapping between genotypes and phenotypes, with a single genetic disorder resulting in many behaviorally distinct types of language disorders. For example, one MZ twin with a genetically encoded articulation disorder might respond by refusing to talk at all, whereas his cotwin with the same genotype might speak and make many articulation errors. Finally, there may be a many-to-one mapping between genotypes and phenotypes, with many distinctive genetic disorders resulting in the same type of linguistic disorder. For example, SLI children who frequently omit grammatical morphemes (see Leonard, 1998; Stromswold, 1997) might do so because they suffer from an articulation disorder such as dyspraxia which causes them to omit grammatical morphemes. that are pronounced rapidly, because they have difficulty processing rapid auditory input such as unstressed, short-duration grammatical morphemes or because they have a syntactic deficit.

Although a single genotype may result in different linguistic profiles and, conversely, different genotypes may result in very similar profiles, researchers should attempt to limit behavioral heterogeneity. Doing so will increase the likelihood of identifying specific genotypes associated with specific types of linguistic disorders. And such focused research would help answer the fundamental question: Is the ability to learn language the result of genetically encoded, linguistically specific operations?

RECOVERY FROM ACQUIRED BRAIN DAMAGE Lesions acquired during infancy typically result in relatively transient, minor linguistic deficits, whereas similar lesions acquired during adulthood typically result in permanent, devastating language impairments (see, for example, Guttman, 1942; Lenneberg, 1967; but see Dennis, 1997, for a critique).[12] The generally more

optimistic prognosis for injuries acquired during early childhood may reflect the fact that less neuronal pruning has occurred in young brains (Cowan et al., 1984), and that the creation of new synapses and the reactivation of latent synapses is more likely in younger brains (Huttenlocher, 1979). Language acquisition after childhood brain injuries typically has been attributed either to recruitment of brain regions adjacent to the damaged perisylvian language regions in the left hemisphere or to recruitment of the topographically homologous regions in the undamaged right hemisphere. According to Lenneberg (1967), prior to puberty, the right hemisphere can completely take over. the language functions of the left hemisphere. The observation that infants and toddlers who undergo complete removal of the left hemisphere acquire or recover near-normal language suggests that the right hemisphere can take over *most* of the language functions of the left hemisphere provided the transfer of function happens early enough (Byrne and Gates, 1987; Dennis, 1980; Dennis and Kohn, 1975; Dennis and Whitaker, 1976; Rankin, Aram, and Horwitz, 1981; but see Bishop, 1983, for a critique). Because few studies have examined the linguistic abilities of children who undergo left hemispherectomy during middle childhood, the upper age limit for hemispheric transfer of language is unclear. Right-handed adults who undergo left hemispherectomy typically become globally aphasic with essentially no recovery of language (e.g., Crockett and Estridge, 1951; Smith, 1966; Zollinger, 1935). The observation that a right-handed 10-year-old (Gardner et al., 1955) and a right-handed 14-year-old (Hillier, 1954) who underwent left hemispherectomy suffered from global aphasia with modest recovery of language function suggests that hemispheric transfer of language function is greatly reduced but perhaps not completely eliminated by puberty.

Studies revealing that left hemisphere lesions are more often associated with (subtle) syntactic deficits than are right hemisphere lesions (Aram et al., 1985; Aram, Ekelman, and Whitaker, 1986; Byrne and Gates, 1987; Dennis, 1980; Dennis and Kohn, 1975; Dennis and Whitaker, 1976; Kiessling, Denckla, and Carlton, 1983; Rankin, Aram, and Horwitz, 1981; Thal et al., 1991; Woods and Carey, 1979) call into question the complete equipotentiality of the right and left hemispheres for language, and suggest that regions in the left hemisphere may be uniquely suited to acquire syntax. It should be noted, however, that some studies have not found greater syntactic deficits with left than right hemisphere lesions (e.g., Basser, 1962; Feldman et al., 1992; Levy, Amir, and Shalev, 1992). These studies may have included children whose lesions were smaller (Feldman et al., 1992) or in different locations than those in studies in which a hemispheric difference for syntax was found. Bates and colleagues (1997) have examined early language acquisition in children who suffered unilateral brain injuries prior to 6 months of age. Parents of 26 children (16 with left hemisphere lesions, 10 with right hemisphere lesions) between the ages 10 and 17 months completed the MacArthur Communicative Development Inventory. According to parental report, overall, children with brain injuries had smaller vocabularies than normal children.[13] Consistent with Mills, Coffey-Corina, and Neville's (1997) ERP findings that the right hemisphere is particularly crucial in the perception of unknown words by children between 13 and 20 months of age, children with right hemisphere lesions had smaller expressive vocabularies and used fewer communicative gestures than children with left hemisphere lesions (Bates et al., 1997). Parental report for 29 children (17 with left hemisphere lesions, 12 with right hemisphere lesions) between 19 and 31 months of age generally revealed that children with left hemisphere lesions had more limited grammatical abilities than children with right hemisphere lesions (Bates et al., 1997). This was particularly true for children with left temporal lesions. Bates and colleagues also compared the mean length of utterance (MLU) in free speech samples for 30 children (24 with left-hemisphere lesions, 6 with right hemisphere lesions) between the ages of 20 and 44 months. Consistent with the parental report results, children with left hemisphere lesions had lower MLUs than children with right hemisphere lesions. MLUs for children with left temporal lesions were especially depressed compared to children without left temporal injuries..

In children who suffer from partial left hemisphere lesions rather than complete left hemispherectomies, language functions could be assumed by adjacent undamaged tissues within the left hemisphere or by homotopic structures in the intact right hemisphere. Results of Wada tests (in which lateralization of language is determined by testing language function when each hemisphere is temporarily anesthetized) indicate that children with partial left hemisphere lesions often have language represented bilaterally or in the right hemisphere (Mateer and Dodrill, 1983; Rasmussen and Milner, 1977). However, one ERP study suggests that children with partial left hemisphere lesions are more likely to have language localized in the left hemisphere than the right hemisphere (Papanicola.ou et al., 1990). There are a number of possible reasons "" t this discrepancy, including differences in the types of linguistic tasks used in the ERP and Wada studies and possibl ifferences in sizes. and sites of left hemisphere lesions in the children studied. In addition, it is possible that. the. discrepancy is due to the fact that most of the children in the ERP study. acquired their lesions after age 4. Furthermore, the extent to which

any of the children in the ERP study ever exhibited signs of language impairment is unclear.

Despite disagreement about the details of language recovery after postnatally acquired left hemisphere lesions, the following generalizations can be made (but see Dennis, 1997). Behaviorally, the prognosis for recovery of language is generally better for lesions acquired at a young age, and syntactic deficits are among the most common persistent deficits. If a lesion is so large that little or no tissue adjacent to the language regions of the left hemisphere remains undamaged, regions of the right hemisphere (presumably homotopic to the left hemisphere language areas) can be recruited for language. The essentially intact linguistic abilities of children with extensive left hemisphere lesions are particularly remarkable when contrasted with the markedly impaired linguistic abilities of SLI children who have minimal evidence of neuropathology on CT or MRI scans. Perhaps the reason for this curious finding is that, although SLI children's brains are not deviant on a macroscopic level, SLI brains may have pervasive, bilateral microscopic anomalies such that no normal tissue can be recruited for language function. One piece of data that supports this hypothesis is found in the results of an autopsy performed on a boy who suffered ,a severe cyanotic episode at 10 days of age. This child subsequently suffered from pronounced deficits in language comprehension and expression until his death (from mumps and congenital heart disease) at age 10. Autopsy revealed that the boy had bilateral loss of cortical substance starting at the inferior and posterior margin of the central sulci and extending backward along the course of the insula and sylvian fissures for 8 cm on the left side and 6 cm on the right side (Landau, Goldstein, and Kleffner, 1960). Perhaps this child did not "outgrow" his language disorder because these extensive bilateral lesions left no appropriate regions that could be recruited for language.

Summary

Evidence from normal. and abnormal language acquisition suggests that innate mechanisms allow children to acquire language. Given adequate early exposure to language, children's language development proceeds rapidly and fairly error-free, despite little or no instruction. The brain regions that permit this. development seem to be functionally and anatomically distinct at birth, and may correspond to what linguists call Universal Grammar. To account for the fact that mastery of a particular language does not occur without exposure to that language during infancy or early childhood, it is possible that the neural fine-tuning associated with learning a language's particular parameters must take place during a period of high neural plasticity. There is some evidence to suggest that the structures and operations involved in language are at least partially anatomically and functionally modular. and apparently have no nonlinguistic counterparts. One possibility is that children have innate mechanisms that, predispose them to perceive categorically linguistic stimuli such as phonemes, words, syntactic categories, and phrases and exposure to these types of linguistic stimuli facilitates the neural fine-tuning necessary for normal language acquisition. For example, some innate mechanisms might predispose children to assume that certain types of meanings and distinctions are likely to be conveyed by morphemes. Also, some innate mechanisms might specifically predispose children to distinguish between syntactic categories that allow for free generalization (lexical categories) and those that do not (functional categories). These innate mechanisms may allow children's brains to solve the otherwise intractable induction problems that permeate language acquisition.

In the future, fine-grained linguistic analyses of the speech of language-impaired children may be used to distinguish between different types of SLI. Linkage studies of SLI may tell us which genes code for the brain structures that are necessary for language acquisition. MRI's exquisite sensitivity to white matter/gray matter distinctions means that MRI could be used to look for more subtle defects associated with developmental language disorders, including subtle disorders arising from neuronal migration or dysmyelinization (Barkovich and Kjos, 1992; Edelman and Warach, 1993). Furthermore, the correlation between myelinization and development of function (Smith, 1981) means that serial MRIs of normal children, SLI children, and WS children could shed light on the relationship between brain maturation and normal and abnormal language development. Finally, functional neuroimaging techniques such as ERP, PET, and fMRI may help to answer questions about the neural processes that underlie language and language acquisition in normal children, SLI children, WS children, children with left hemisphere lesions, and children who are exposed to language after the critical period.

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NOTES

1. Children differ dramatically in the rate of acquisition. For example, Brown (1973) and Cazden (1968) investigated when three children mastered the use of 14 grammatical

morphemes. Although all three children eventually obtained competence in the use of the third-person singular verbal inflection -s (as in *he sings)* and all three reached this point after they achieved adult-like performance on plurals and possessives, one of the children reached competence at 2;3 (2 years and 3 months), one at 3;6, and one at 3;8. Similar findings concerning individual differences have been found in the rate of acquisition of questions (Stromswold, 1988, 1995) and auxiliaries (Stromswold, 1990a,b) as well as datives, verb particles, and related constructions (Snyder and Stromswold, 1997; Stromswold, 1989a,b). A number of studies have also reported that children's vocabulary development can vary greatly in both rate and style (e.g., Nelson, 1973; Goldfield and Reznick, 1990).

2. Although the observation that the pattern of acquisition varies depending on the structure of the language is consistent with functionalist accounts of language acquisition (e.g., MacWhinney, 1987), such observations can be accounted for within generative theories if one makes the assumption that children must receive a certain amount of positive data from the input in order to set parameters (for P&P) or rank constants (for OT).
3. Throughout this chapter, ungrammatical sentences are indicated with an asterisk (•).
4. There are 23! logically possible unique orders of all 23 auxiliaries. The total number of orders including sets with fewer than 23 auxiliaries is considerably bigger. Because the 23! term is the largest term in the summation, it serves as a lower bound for the number of unique orders and suffices as an estimation of the number of orders.
5. The mere existence of cerebral asymmetries does not prove that there is an innate basis for language, as other mammals also exhibit such asymmetries.
6. Fifteen of the 20 parents and 4 of the 10 siblings had language deficits. The controls had no personal or family history of language impairment or delay.
7. These were not, however, the same children who did poorly on Tallal and Piercy's (1973a,b) auditory processing task.
8. Clinicians and researchers generally agree that considerable diversity exists in the behavioral profiles and manifestations of children diagnosed with SLI and that it is important to distinguish between various subtypes of SLI. However, no system for classifying subtypes of SLI is generally accepted (Stromswold 1997).
9. The term "proband" refers to an affected individual through whom a family is brought to the attention of an investigator.
10. The variance is due in large part to what was counted as evidence of language impairment in families. As indicated in table 63.1, some studies considered family members to be affected only if they suffered from a spoken language disorder, whereas other studies counted as affected any family members having a history of dyslexia, nonlanguage learning disabilities, or school problems.
11. In this chapter, all concordance rates are for proband-wise concordance rates. Proband-wise concordance rates are calculated by taking the number of affected individuals in concordant twin pairs (i.e.; twin pairs where both twins are affected) and dividing this number by the total number of affected individuals.
12. In a recent review of research on children whose brain injuries occurred after the onset of language acquisition, Dennis (1997) argues that the prognosis is no better four children than adults once the etiology of the brain injury is taken into account.
13. Bates and colleagues (1997) report large variance in language abilities among their lesioned subjects, with some of the children's language being at the high end of the normal and other children suffering from profound impairments. This probably reflects, at least in part, variations in the size and sites of the lesions among their subjects.

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