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Origins of Language Disorders: A Comparative Approach

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The five empirical articles in this special issue illustrate the value of a comparative approach to the study of normal and abnormal language development. The information presented here does not just add up; it multiplies, yielding insights that would not be available from any of the individual parts examined alone. All five articles focus on the first stages of language learning, from first signs of word comprehension to the emergence of grammar. They underscore the immense variation that can be observed in normal children, and the range of ways that language can break down or go awry in the early stages. These results place important constraints on our understanding of the origins of communication disorders. At the same time, they provide substantial hope to families of children who are not developing on a normal schedule, demonstrating there may be several different paths to the achievement of language abilities within the normal range. The human language faculty is remarkably plastic, at least within the early stages. To be sure, there are some circumstances in which this plasticity appears to be quite limited. Here too, however, there are reasons to believe that our increased knowledge of the origins of communication disorders may lead to treatments that release children from these limitations on plasticity and learning, placing them on the road to recovery.

The summary and discussion that follows is divided into four parts: (a) a brief discussion of the potential for neural and behavioral plasticity revealed by the literature on normal brain development in human and nonhuman primates, (b) a discussion of the article by Mills, Coffey-Corina, and Neville (this issue) on event-related brain potentials associated with such variations, (c) a comparison of these data with the four articles in this issue describing early language in atypical

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populations (Thal, Bates, Goodman, & Jahn-Samilo, on late talkers; Bates et al., on focal brain injury; Singer Harris, Bellugi, Bates, Jones, & Rossen, on Williams and Down syndromes; and Dixon, Thal, Potrykus, Bullock Dickson, & Jacoby, on infants of substance-abusing mothers), and (d) a consideration of the neural factors that may be responsible when children fail to display the kind of plasticity observed in most of our populations.

ON THE NEURAL BASES OF NORMAL LANGUAGE DEVELOPMENT

Twenty years ago, speculation about the neural bases of language development focused on the addition of new neural structures (e.g., Geschwind, 1964, 1970; Lenneberg, 1967; Parmelee & Sigman, 1983), now called *additive events.* These include (a) the birth and migration of cells to their proper sites in the cortex, (b) the growth and establishment of long-distance axonal connections (i.e., basic "wiring up"), (c) the myelination of those lines, (d) the establishment of local synaptic connections, and (e) the strengthening or weakening of those local connections. Major changes in behavior during the first years of life often have been ascribed to *maturation,* a term reserved for additive events in the first four categories; traditionally, the fifth category has been viewed as the neural consequence of experience (also called *Hebbian learning;* Hebb, 1949). In the past 20 years, this additive and unidirectional view of brain development has been replaced by a much more complex and dynamic view, a bidirectional interplay of maturation and experience achieved through a combination of additive and subtractive events (for reviews, see Bates, Thal, & Janowsky, 1992; Deacon, in press; Edelman, 1987; Elman et al., in press, chap. 5; Oyama, 1992; Quartz & Sejnowski, in press; Smith & Thelen, 1993; Thelen & Smith, 1994; Wills, 1993). Here are some reasons why the old maturational view has lost its appeal.

Among other things, we now know that additive events play a less important role in postnatal brain development than previously was believed (or, at least, a less exclusive and less direct role). In humans and other higher primates, the birth and migration of cells is essentially complete well before birth. Axonal connections are set up in the first months of postnatal life, reaching an adult configuration by approximately 9 months of age in humans. This fact may help to explain the wide range of behavioral changes that typically are observed around 8 to 10 months in humans (e.g., Diamond, 1991), but this kind of basic wiring cannot be used to explain gross changes in behavior after that point (e.g., in the period from 10 to 48 months, when most of language development takes place). By contrast, myelination of axonal connections continues for many years, a fact that makes it seem like a good first-pass candidate for a maturational account of behavioral change (Parmelee & Sigman, 1983). However, we also know that myelination is a continuous process that goes on for at least 2 decades, and that information is conducted reasonably well across axonal connections before myelination is complete. For these two reasons, it is not clear how developmental changes in the myelin sheath could be used to explain massive and apparently discontinuous changes in language ability in the first years of life.

What about synaptic growth, the fourth category of additive events? Three points are relevant here. First, it is now clear that a huge burst in synaptic branching takes place some time after birth (Rakic, 1975), reaching its peak between 9 and 24 months in humans (Huttenlocher, 1979, 1990; Huttenlocher & de Courten, 1987; Huttenlocher, de Courten, Garey, & van der Loos, 1982). In fact, synaptic density in human 2-year-olds is approximately 50% greater than the densities observed in human neonates or in human adults—which means (at least in synaptic terms) that children start to work on the language problem with a brain and a half at their disposal.

Second, it is clear that synaptic growth continues throughout life, and that the amount of growth observed in later years is affected by experience. Although synaptogenesis never occurs again on the scale observed in the first months of life, increases up to 20% or more in second- or third-order branching have been observed in some areas of the brain in older animals exposed to a radical increase in environmental stimulation (Greenough, Black, & Wallace, 1993).

Third, the patterns of synaptic connectivity established in each cortical region are affected strongly by the input to that region (e.g., Sur, Pallas, & Roe, 1990; for reviews, see Elman et al., 1996; Killackey, 1990). For example, when plugs of fetal somatosensory cortex are transplanted to visual areas, they establish patterns of connectivity appropriate for visual input; and when plugs of visual cortex are transplanted to a somatosensory zone, they take on representations appropriate to somatosensory inputs (i.e., "When in Rome, do as the Romans do"). Hence, there is remarkable plasticity at the synaptic level in the neocortex.

Putting these lines of evidence together, we may conclude that additive events play an important role in brain and behavioral development, and those additive events that take place up to age 2 are strongly constrained by genetic factors (including the burst of synaptogenesis observed between 9 and 24 months). However, the line between maturation and experience is not at all clear at the local synaptic level, and there are (to the best of our knowledge) no dramatic additive events after age 2 that could be used to explain massive, discontinuous changes that take place later on at a behavioral level.

These findings are complemented by some major discoveries in developmental neurobiology during the past 20 years, revolving around the role of *subtractive* or *regressive events,* including (a) cell death, (b) axon retraction, and (c) synaptic elimination. We now know that subtractive events play a crucial role in postnatal brain development, serving as the instrument by which experience sculpts the brain into its mature form (Chugani, Phelps, & Mazziotta, 1987; Huttenlocher et al., 1982;

Janowsky & Finlay, 1986; Rakic, 1975). The first class of subtractive events includes *programmed cell death,* a large-scale form of neural suicide that takes place in the first weeks before and after birth. It generally is assumed that the onset of programmed cell death is under strict genetic control, although the fate of individual cells has more to do with their relative success or failure (i.e., their overall level of activity and connectivity when the programmed plague begins). In a sense, programmed cell death can be viewed as a neural game of musical chairs: An arbitrary signal determines that the game is over, and those that are able to find a chair can stay. Cell death continues after this point as well, although later versions of cell death have more to do with success or failure at a local (cell by cell) level. Axon retraction is a natural consequence of cell death (i.e., dead cells lose their connections), but it is also a potential cause of cell death (i.e., cells that fail to make appropriate long-range connections are candidates for elimination).

For the most part, major changes in axonal connectivity (both additive and subtractive events) are complete within the first months of life. Under normal conditions, these axonal processes lead to species-specific, "default" forms of large-scale connectivity. However, numerous experiments in the last few years have demonstrated that alternative forms of large-scale axonal connectivity are possible if the default situation does not hold (e.g., animals that are deprived of normal visual or auditory input; Frost, 1990; Hubel & Wiesel, 1970; Killackey, 1990; Killackey, Chiaia, Bennett-Clarke, Eck, & Rhoades, 1994; Miller, Keller, & Stryker, 1989; Sur, Garraghty, & Roe, 1988; Sur et al., 1990). In particular, exuberant axons that normally would wither away can be retained into adulthood. This kind of alternative routing at the axonal level may play an important role in the forms of neural and behavioral plasticity observed in some of the clinical populations under study here, although such developments are probably restricted to the 1st year of life.

In contrast with the dramatic but time-limited events that take place at the cellular and axonal levels, a single neuron can make hundreds or even thousands of synaptic connections. Furthermore, changes at this level take place over and over again across the lifetime of a successful cell. For this reason, additive and subtractive events at the synaptic level are the most likely candidates for the neural substrates of language and cognitive development after 1 year of age. Crucial for our purposes here, these events are the direct result of neural activity and neural competition, which means that it is impossible to distinguish between maturation and learning at this level of development.

Figure 1 (from Bates et al., 1992) illustrates the developmental course of synaptic growth and synaptic elimination from birth to adulthood in the human brain. According to Huttenlocher's (1979) cross-sectional analysis of human brains, synaptic density reaches a peak in the posterior cortex (especially the visual areas) around 9 to 12 months of age, whereas the corresponding peak in the frontal cortex is not observed until approximately 24 months. Rakic, Bourgeois, Eckenhoff,

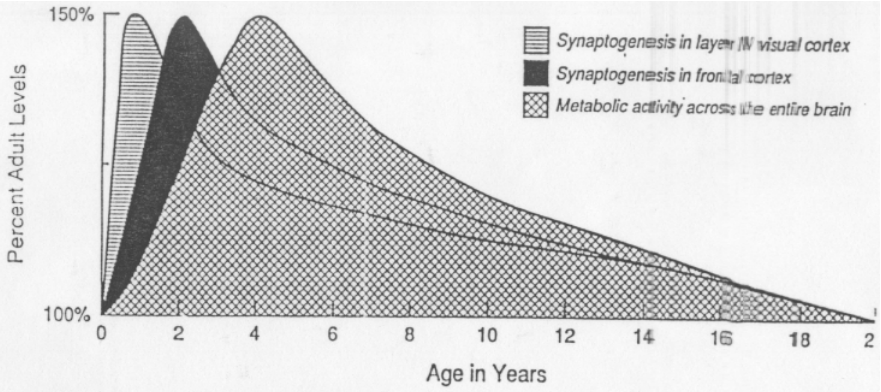

FIGURE 1 Successive "peaks" in brain development for synaptogenesis and metabolic activity from birth to adolescence. (Reprinted from Bates et al., *Early Language Development and its Neural Correlates,* 1992, pp. 69–110, with kind permission from Elsevier Science - NL, Sara Burgerhartstraat 25, 1055 KV Amsterdam, The Netherlands.)

Zecevic, & Goldman-Rakic (1986) disputed this point, arguing instead that synaptogenesis reaches its peak simultaneously in all areas of the cortex. Because Rakic et al.'s results are based on monkey data, it is difficult to be precise about the temporal location of the corresponding peak in human brains; but if they are correct, the most likely point would be somewhere around 24 months. Figure 1 also includes metabolic results from Chugani et al.'s (1987) positron emission tomography studies of human infants and children. Although the peak in brain metabolism observed in Chugani's studies appears approximately 1 to 2 years downstream from Huttenlocher's data for synaptogenesis, the shape of all three curves in Figure 1 is quite similar: a huge burst between 9 and 48 months of age, followed by a long, slow decline that reaches asymptote somewhere in the 2nd decade of life.

The functional consequences of Figure 1 are quite dramatic for our understanding of the relation between brain and language development. Language typically starts around 8 to 9 months (with first signs of word comprehension) and stabilizes around 4 years of age (when most normal children have acquired the fundamental structures of their native grammar). This means that normal language development takes place during a neural firestorm, when synapses are coming and going at an astronomical rate. This is the period in which the child has an enormous number of potential connections (i.e., maximum capacity), which in turn means the child has an enormous number of possible outcomes (i.e., maximum plasticity).

We should note there are two opposing ways to interpret the data in Figure 1. We propose these events form the basis for a highly interactive, bidirectional theory of brain and behavioral development. By contrast, Piatelli-Pal-

marini (1989) argued for a radical nativist interpretation of the same facts. In particular, he suggested the infant brain contains a large set of innately specific, domain-specific patterns of connectivity, available prior to any form of learning or experience. Brain development after birth constitutes nothing more than a process of selection from this stock of innate possibilities. Hence, the gradual process of synaptic elimination illustrated in Figure 1 could be viewed as the elimination of innate options that will not be used (referred to in the linguistic literature as *parameter setting*). Indeed, Piatelli-Palmarini went on to suggest that learning itself is an illusion; everything of any importance is already there at the beginning: "I, for one, see no advantage in the preservation of the term 'learning.' I agree with those who maintain that we would gain in clarity if the scientific use of the term were simply discontinued" (p. 2).

Although this radical nativist scenario is logically possible, it is not borne out by modern research in developmental neurobiology. As noted earlier, a growing body of evidence on plasticity and development at the cortical level shows that fine-grained patterns of cortical connectivity are not laid out in advance, at least not at the synaptic level (see Elman et al., 1996, chap. 5, for a detailed review). Neurons are not born knowing what kinds of patterns they have to establish with their near neighbors. Instead, brain development in mammals appears to be based on a process of synaptic exuberance (i.e., more connections than the animal will need), followed by a process of elimination through competition—what Edelman (1987) called "neural Darwinism" (p. 19). The huge burst in connectivity illustrated in Figure 1 thus can be viewed (metaphorically) as a large block of marble delivered to the artist's studio. The sculpting process that follows is a bidirectional event, reflecting the joint constraints of structure and experience, maturation and learning. It is probably no accident that language development takes place in this period of maximum capacity and maximum plasticity.

VARIATION IN NORMAL LANGUAGE DEVELOPMENT

As Thal and Reilly note in the introduction to this issue, recent large-sample studies of early language development using the MacArthur Communicative Development Inventories (CDI) (Fenson et al., 1993, 1994) provide evidence for massive individual differences in onset time and rate of development for all major milestones—differences large enough to challenge the very concept of "normality." However, this variation is quite orderly, and systematic relations have been observed between the pace of language development (independent of age) and brain activity associated with linguistic stimuli. Studies using variants of the MacArthur CDI in other languages confirmed that similar means and similar ranges of variability can be observed in languages as diverse as English, Spanish, Italian, Japanese, Swedish, Icelandic, and American Sign Language (ASL).

Among other things, these findings raise a red flag for cross-linguistic studies based on small longitudinal samples—a common methodology in the child language literature (cf. Slobin, 1985, 1992). If we were to select at random one child from each of the languages we are interested in, we might find huge differences that have little or nothing to do with language input per se. If we were unlikely enough to choose children from opposite extremes (e.g., a slow Italian child and a precocious ASL child), we might be tempted to conclude that these variations are caused by differences in the structure, the processing, or both the structure and the processing of Italian and ASL. It is much more likely, however, that these differences are based on chance variation (i.e., the putative cross-linguistic difference is a false positive). Within a given language, case studies can be a rich source of information about patterns that are *possible* (e.g., interesting error types, cross-language variations in the sequence of development), but they should not be used to draw inferences about those patterns that are *probable* or *typical* within that language environment.

At the same time, cross-linguistic studies of normal children also demonstrate that a certain amount of variation is *inevitable,* regardless of the language to which children are exposed. So, from where does all this variation come? Some of this variation undoubtedly is due to differences in the amount and quality of parental input (although Fenson et al., 1994, noted that correlations with crude environmental measures such as birth order and social class are relatively low in the original norming sample). Some of it may come from natural variations in the rate of maturation (e.g., in the additive and subtractive events illustrated in Figure 1); if this is the case, it would help to explain why equivalent variability is observed in dramatically different language types. Some of it also may come from characteristics of the child that are related only indirectly to language itself. These might include temperamental differences (e.g., shyness, risk taking, and sociability), cognitive differences in rate and style of learning that cut across linguistic and nonlinguistic domains, differences in perceptual acuity, auditory short-term memory, and so forth (for a discussion, see Bates, Dale, & Thal, 1995). For our purposes, the point is that large individual differences are present across the normal range in every language community studied to date, from first signs of word comprehension to the mastery of grammar. Hence, the word *normal* applies to a very broad range of performance at any given point in development—a fact that must be kept in mind when we undertake studies of the origins of communication disorders in clinical populations.

Fortunately, evidence suggests that this variation is neither random nor chaotic. In particular, the article by Mills et al. (this issue) demonstrates that individual differences in the rate of language development during the 2nd year of life are linked to observable changes in brain activity. Several findings from Mills et al. are worthy of note.

First, Mills et al. (this issue) uncover distinct patterns of electrical activity over the scalp in response to (a) familiar words (as established for each child by parental report and laboratory testing), (b) real English words that are unfamiliar to the child, and (c) backwards speech (auditory stimuli that are unrecognizable as English words but are equivalent in complexity). This methodological contribution has great theoretical and empirical consequences. Comprehension is notoriously difficult to measure in this age range. Parent report measures of comprehension have proven to be valid and reliable up to, but not beyond, 16 to 18 months of age (at least for children who are developing at an average or greater-than-average rate). Laboratory measures are quite unreliable before this point because they require a degree of cooperation that is difficult to obtain in children under 18 months of age. Mills, Coffey, and Neville (1991) demonstrated that electrophysiological techniques can be used to measure word recognition across this age range, starting as early as 10 months and continuing through the preschool years and beyond. To be sure, some cooperation is required (e.g., the child must be willing to wear the electrode cap and sit still long enough to register single word stimuli without movement artifacts), but the degree of cooperation is far less than usually is required for picture-pointing or acting-out tasks. The authors also show that these event-related potentials (ERP) measures correlate with parent report and behavioral indexes of word comprehension, concurrently and longitudinally (over periods as long as 12 to 36 months in some cases; see also Molfese, 1989, 1990). This finding constitutes yet another cross-validation of ERP, laboratory, and parent report measures (for reviews of validity studies, see Bates, Bretherton, & Snyder, 1988; Fenson et al., 1993). More important still, it provides us with another way to uncover risk for language impairment before 2 years of age (Thal et al., this issue; see discussion later in this article).

Second, Mills et al. (this issue) demonstrate a consistent developmental pattern in the neural activity associated with familiar words. In the first stages (when a difference between known and unknown words first appears), this activity is distributed broadly over the two hemispheres and is (if anything) slightly larger on the right. Later on in the 2nd year, brain response to familiar words is larger on the left and more focally distributed over the frontotemporal cortex. As is discussed in more detail later, this kind of dynamic change in brain activity is compatible with studies of language ability in children with focal brain injury (Bates et al., this issue).

Finally, the Mills et al. (this issue) article clearly shows that language ability is a better predictor of these developmental changes than is chronological age. This finding is compatible with theories of brain development that emphasize the bidirectional relation between maturation and experience. In fact, the left-anterior shift in brain activity observed by Mills et al. may be the product of linguistic experience rather than its cause. This finding also suggests that the wide range of individual differences seen across the many languages now sampled using variants of the MacArthur CDI (e.g., Caselli & Casadio, 1995; Jackson-Maldonado, Marchman, Thal, Bates, & Gutierrez-Clellen, 1993; Ogura, Tamashita, Murase, & Dale, 1993; Reilly, Provine, & Anderson, 1996) have clear and measurable neural correlates, opening up promising avenues for future research on normal language development.

DEVELOPMENT, DEVIANCE, AND DELAY IN ATYPICAL POPULATIONS

The four articles in this issue on early language development in atypical populations provide a wealth of detailed information that is difficult to summarize. I concentrate here on highlights that are particularly relevant for the issues of variation and plasticity.

Late and Early Talkers

Thal and her colleagues have spent many years seeking early indicators of risk for language delay. The article by Thal et al. in this issue focuses on short-term predictors at both the group and the individual levels. Three studies are presented using the MacArthur CDI (Fenson et al., 1993), identifying children in the top and bottom 10th percentiles for expressive vocabulary at various points in time (i.e., early talkers and late talkers). The first two studies present new information from a 6-month longitudinal follow-up of children who participated in the original MacArthur norming study (Fenson et al., 1994). The third study represents a first round of results from an intensive, month-by-month longitudinal study of 30 children from 8 to 30 months of age (Goodman & Bauman, 1995).

Demographic factors such as sex, birth order, ethnicity, and social class did not have any reliable predictive value in any of their analyses, but measures of language and communication did show continuity over time at the group level. In the two short-term longitudinal studies, late and early talkers both had a higher-than-chance probability of retaining their extreme-group status across a 6-month period. In the period between approximately 13 and 20 months, Time 1 word comprehension, gesture, and word production (including the percentage of comprehended words that also are produced) all made significant and independent contributions to later status. It is interesting to note, however, that the pattern of predictors was different for early versus later talkers. For late talkers at 20 months, the best 13-month predictors were gesture and the percentage of comprehended words that also are produced (both significantly lower in children who later were destined to end up in the bottom 10th percentile for expressive vocabulary). For early talkers at 20 months, the only predictor that made a significant independent contribution was total vocabulary size when the children were just 1 year of age. Results from their

third study, with more finely grained longitudinal data, confirm this picture: Late talkers lagged consistently throughout the study in comprehension and gesture as well as in expressive vocabulary, but early talkers differed from average children only on the expressive language measures by which they were defined. The differential predictive value of these 13-month measures is interesting when the Thal et al. (this issue) results for late and early talkers are compared with the Bates et al. (this issue) results for differential breakdown of comprehension, gesture, and production in children with focal brain injury. In the period between approximately 20 and 26 months, the single best predictor of either late- or early-talker status at Time 2 was total expressive vocabulary at Time 1. There was no compelling evidence that grammar and vocabulary dissociate or make differential predictions within this age range. That is, 20-month vocabulary is a solid predictor of both precocity and delay at 26 months in both vocabulary and grammar. As is discussed in more detail later, this apparent inseparability of lexical and grammatical development also is compatible with the Bates et al. results for children with unilateral brain damage.

These findings at the group level are intriguing, but Thal et al. (this issue) conclude that it still is difficult to predict the outcome for individual children within these groups. Some late talkers stay delayed, others burst upward to the median and beyond. Unfortunately, none of the variables examined by these authors can distinguish those who "catch up" from those who stay behind. These findings can be viewed from three different points of view.

At a clinical level, these results are discouraging, that is, we do not have a litmus test that can be used before 2 years of age to identify children who need services. The litmus test approach is often used in educational circles to identify children who require special services for learning disabilities (broadly defined). In recent writings, Thal and others (Thal & Katich, in press; Whitehurst & Fischel, 1994) have suggested that we abandon this approach to the early identification of risk for language impairment and turn instead to a risk factor model of the sort that commonly is used in medical research and practice. For example, we know that a number of different risk factors contribute to the likelihood that an individual will develop breast or colon cancer (e.g., positive family history and dietary patterns). However, responsible physicians use this information with great caution when dealing with individual patients. Risk factors are assessed, advice is given where warranted, and patients with a high risk index are carefully monitored; but probabilistic information of this sort never is used to say to an individual patient "You are in trouble." Instead, patients are given all the available information, including a list of options that they may want to follow while their progress is monitored. This is the approach that Thal and her colleagues (this issue) recommend for the assessment of language delay before 3 years of age (see also Paul, 1991; Rescorla et al., 1995; Whitehurst, Fischel, Arnold, & Lonigan, 1992).

At a more personal level, the Thal et al. (this issue) results provide a message of hope for worried families. Parents are right to be concerned and vigilant if their children are delayed in the attainment of language milestones. Under the risk factor model recommended by Thal et al., parents should be encouraged to bring their concerns to the attention of physicians and other practitioners as soon as they think there might be a problem, and to insist (if necessary) that someone take a closer look. Indeed, a host of robust and positive results using the MacArthur CDI proves that parent report is a valid index of current language abilities, with considerable predictive validity as well. Parents know a lot about their children's progress, and their concerns should be taken seriously. At the same time, the dramatically different growth trajectories displayed by individual children in the Thal et al. article can be a source of comfort for parents who have undertaken this course of action. A slow start does not preclude a brilliant finish.

At a scientific level, these results for late and early talkers provide a substantial challenge. There clearly is evidence for continuity in the rate and style of development (see also Bates et al., 1988), but there also is substantial evidence for plasticity. Children who start out at the same level can reach very different endpoints, and children can move toward the same endpoint from very different positions. The mechanisms responsible for continuity versus plasticity are virtually unknown, and they constitute an important direction for future research. In this respect, the electrophysiological methods described by Mills and her colleagues (this issue) for normally developing children may prove very useful in assessing alternative pathways from first words to grammar. Our efforts to understand the mechanisms responsible for behavioral plasticity also will profit from investigations of both behavioral and neural plasticity in other clinical populations—which brings us to results for children with focal brain injury.

Children With Focal Brain Injury

Bates et al. (this issue) summarize more than 8 years of research in three large cities that looked at the course of early language development for 53 children with unilateral brain injuries incurred some time before 6 months of age (i.e., before the point at which language acquisition normally begins). These results were compared with findings in another article (Reilly, Bates, & Marchman, in press) that looked at language and discourse abilities between 3.5 and 12 years of age for a partially overlapping group of children with the same etiology. In total, the results discussed by Bates et al. are based on a sample of 72 children. Although results still are tentative (and must be viewed as working hypotheses for future research), it is worth noting that this is the largest and most homogeneous sample of children with early focal brain injury ever studied across this age range. A brief summary of these complex results includes the following highlights:

1. Although children with early brain lesions are (as a group) behind their normal age mates, individual differences within the focal-lesion population span the full range of variability, from under the 5th percentile to above the 90th percentile at every point in development and on every measure of language and communication.

2. Deficits in comprehension and gesture in the earliest stages of language development are more common in children with right-hemisphere damage. This directly contradicts findings for brain-injured adults (for whom deficits in comprehension, communicative gesture, and pantomime are more common following left-hemisphere damage), but it is compatible with dissociations observed in normal infants.

3. There is no evidence whatsoever for a dissociation between vocabulary and grammar during the first stages of language development (i.e., the passage from first words to grammar, from 8 to 30 months). This result contrasts sharply with claims made in the literature on adult aphasia, but it is compatible with the close association between lexical and grammatical development observed in cross-sectional and longitudinal studies of normal infants.

4. Deficits in expressive vocabulary and grammar are more likely in children with left-temporal injuries throughout the period from 10 to 60 months. None of the infants with left-temporal damage in our sample showed delays in comprehension, and none of them displayed the kind of fluent but empty speech associated with damage to the left-temporal cortex in an adult (i.e., no developmental cases of Wernicke's aphasia). In other words, injury to the region that includes Wernicke's area leads to a "Broca-like" pattern of deficits in very young children.

5. Frontal damage increases the risk for expressive language delay, but only within a narrow time window from 19 to 30 months, and it occurs with both left- and right-frontal damage; there is no evidence that left-frontal tissue is "special" at any point from first words through the acquisition of grammar. One possible interpretation of this finding is that the localization of key language functions in and around Broca's area is the product, rather than the cause, of normal language development.

6. None of these site-specific effects are observed in the Reilly et al. (in press) study of older focal-lesion children with the same early-onset etiology. After some point between 5 and 7 years of age, focal-lesion children, as a group, perform within the normal range but significantly below a matched sample of normal controls on most language measures—which means that some kind of price has been paid for reorganization in response to early injury. However, there are no longer any significant effects of size or site of lesion, suggesting that reorganization for language in the focal-lesion population takes place within the period from 0 to 5 years of age.

These findings underscore the important role of neural plasticity during the infant and preschool years. In contrast with old claims about equipotentiality (e.g., Lenneberg, 1967), lesion site does matter, even in children whose lesions are acquired at or shortly after birth. However, the lesion-symptom mappings observed in young children are qualitatively and quantitatively different from those observed in adults with homologous injuries. Among other things, this suggests that "the regions responsible for language learning are not necessarily the same as the regions responsible for use and maintenance of language in the adult" (Thal et al., 1991, p. 499). At the same time, these findings prove that alternative forms of brain organization for language are possible, and that these alternatives probably emerge at some point between 0 and 5 years of age.

Although these results are quite surprising from the point of view of lesion-symptom mapping in adults, they are compatible with other studies of children with focal brain injury (see Bates et al., this issue, for a list), and they are compatible with results by Mills et al. (this issue; see also Mills, Coffey-Corina, & Neville, 1993, 1994, in press) on the normal development of language-related brain activity. As noted, Mills et al. showed that the electrophysiological response to familiar words is broadly distributed and (if anything) somewhat larger on the right during the first stages of language learning. This finding complements the Bates et al. finding for word comprehension from 10 to 17 months, that is, a small but reliable disadvantage for children with right-hemisphere damage, suggesting that the right hemisphere may play a more important role (or, at least, an equally important role) during the period in which children break the code connecting sound and meaning. Later in the 2nd year for normal children (sooner for children who are developing at a rapid rate), Mills et al. observe a progressive left-anterior shift in the ERP to familiar words, suggesting that left-temporal areas are taking on a more important role in the mediation of meaningful speech. This developmental pattern may reflect the emergence of default organization in children without brain injury. It remains to be seen what kind of pattern would emerge at a comparable point in the development of children with left-temporal injuries. Mills is now conducting collaborative studies with the San Diego focal-lesion team, using ERP technology to observe the emergence of alternative forms of brain organization for language in the focal-lesion population.

But why is the left-temporal cortex so important? On the one hand, the Mills et al. (this issue) finding for normal children is compatible with 100 years of research on normal and brain-injured adults suggesting that the perisylvian areas of the left hemisphere play a critical role in language processing. On the other hand, research on infants with focal brain injury contradicts the adult findings in two important respects: (a) In the short run, left temporal lesions have a greater effect on production

than on comprehension; and (b) in the long run, left-temporal lesions do not result in a permanent aphasia. Bates et al. (this issue) propose that this contradiction can be resolved if we assume the default pattern of brain organization for language emerges out of initial biases in style of computation that are related only indirectly to the final state. In the infant brain there are no "innate language centers." Instead, there are regional differences in information-processing capacity (i.e., what Elman et al., 1996, called "architectural contraints" rather than "representational constraints," p. 27).

Comparing their results for language to those of Stiles and colleagues (Stiles & Thal, 1993) for visuospatial cognition in children with focal brain injury, Bates et al. (this issue) suggest the left-temporal cortex is particularly well suited for the extraction of detailed perceptual information (both spatial and temporal), a capacity that is especially important during language learning. Ironically, the ability to perceive fine-grained details may be more important for production than it is for comprehension because children have to conduct a detailed perceptual analysis to create their first motor templates for familiar words. Under default conditions, the left-temporal cortex is recruited for this function via a competitive process (not unlike the process by which tall and agile children end up on the basketball team). However, if the left-temporal cortex is injured in some way, other areas are able to take over the task. They may not perform quite as well, but they are perfectly adequate for the job.

Williams Syndrome and Down Syndrome

The article by Singer Harris et al. (this issue) compares the first stages of language development in populations of children with two forms of mental retardation: Williams syndrome and Down syndrome. Both are genetically based disorders, and in both cases the cognitive disorder is accompanied by a host of other physical and behavioral markers. Hence, we would not expect (and we did not find) any simple mapping from genes to language (for a more detailed discussion of this point, see Elman et al., 1996). The crucial point for our purposes is that adults and adolescents with Williams syndrome display a remarkable sparing of language, compared with the severe delays and disabilities that they display in many other cognitive domains (Bellugi, Wang, & Jernigan, 1994; Giannotti & Vicari, 1994; Wang & Bellugi, 1994). By contrast, adults and adolescents with Down syndrome often display language abilities below their mental age, including a marked deficit in the production of grammatical morphemes (Chapman, 1995). To be sure, this apparent double dissociation between grammar and cognition must be interpreted with caution. First, there is substantial variability in both groups, and many children who do not display the "signature profile." Second, both groups display uneven patterns of sparing and impairment within language and within nonverbal cognition (including a remarkable sparing of face perception in children with Williams syndrome, who are otherwise quite impaired in visuospatial cognition). Nevertheless, the degree of dissociation observed in older children and adults provides a substantial challenge to theories that presuppose a tight link between linguistic and cognitive abilities.

How serious is this challenge? Suppose we can show that a retarded 16-year-old with an IQ of 50 has mastered English grammar? Is this is a surprise? Not necessarily. Roughly speaking, a 16-year-old with an IQ of 50 has a mental age equivalent to that of a normal 6- to 8-year-old. Because most normal 6-year-olds have mastered their grammar, we should not be surprised to find that a retarded adolescent with a 6-year-old mind has managed the same feat (particularly when we take into account the fact that this adolescent has been 6 years old for a very long time). For this reason, a better test of the link between language and cognition would come from the study of much younger children. When do children with Williams and Down syndromes diverge? Are their contrasting profiles evident from the very beginning, or do they emerge at a later point in development? If language is an independent cognitive system, or *module,* as some authors have proposed (Curtiss, 1988; Pinker, 1991, 1994; Roeper, 1988), it should be possible for language to develop well before the point at which putative cognitive prerequisites are in place. However, if language development depends on the prior attainment of certain key cognitive abilities, it should not be possible for children to acquire grammar until those abilities are in place (Bates & Thal, 1991; Bates, Thal, & Marchman, 1991; A. Gopnik & Meltzoff, 1987).

The article by Singer Harris et al. (this issue) resolves a number of important points in this regard. First, their results clearly indicate that both groups of children are delayed massively in the attainment of early language milestones, approximately 2 years behind normal controls on virtually every aspect of language and communication. Second, the two groups are delayed equally in expressive vocabulary across the period covered by this cross-sectional study (i.e., from 1 to 6 years of age). Third, there appear to be qualitative differences between the two groups in the relations observed among word comprehension, word production, gesture, and grammar. In the first stages (equivalent to normal development from 8 to 16 months), children with Down syndrome have a marked advantage in the use of gesture compared with children with Williams syndrome and with younger normal controls at the same levels of word comprehension or production). In the later stages (equivalent to normal development from 16 to 30 months), children with Down syndrome also have a significant disadvantage in the production of grammar (compared with children with Williams syndrome and with younger normal controls at the same level of expressive vocabulary). In contrast with the very uneven profile displayed by children with Down syndrome, the Williams syndrome sample shows a relatively normal profile of cross-domain relations—except, of course, that this "normal" profile is shifted downstream by approximately 2 years.

What can we conclude from these findings? The global delays in language development displayed by both groups are compatible with the claim that language is dependent on the attainment of certain minimum cognitive/conceptual abilities (i.e., "cognitive infrastructures"; Bates et al., 1995, p. 146). On the other hand, the two groups display markedly different patterns of development across domains. In particular, children with Down syndrome manifest a selective deficit in grammatical production from the very beginning, a deficit that cannot be explained by their cognitive levels or by the lack of progress in expressive or receptive vocabulary. This finding suggests that cognitive, lexical, or both cognitive and lexical abilities are necessary, but not sufficient, for grammar to emerge, but it does not tell us anything about the "missing ingredient." There are at least two possibilities: (a) Grammar is selectively delayed in children with Down syndrome because these children lack a language-specific ability revolving around the analysis and acquisition of grammatical morphemes, or (b) grammar is selectively delayed in children with Down syndrome because grammatical morphemes make a greater demand on perceptual abilities that are selectively impaired in this group.

Support for the second hypothesis comes from several quarters. First, it has been established independently that children with Williams syndrome tend to have remarkably acute hearing (i.e., *hyperacusis;* Klein, Armstrong, Greer, & Brown, 1990; Martin, Snodgrass, & Cohen, 1984; Morris, Demsey, Leonard, Dilts, & Blackburn, 1988), and there is also some evidence to suggest that their visual processing is disturbed (Morris et al., 1988). These two facts could lead indirectly to a relatively greater reliance on linguistic communication. Second, the selective advantage in gesture and the selective disadvantage in grammar displayed by children with Down syndrome are both compatible with a profile in which vision outstrips audition. This idea received support from a study by Wang and Bellugi (1994), who reported a double dissociation between visual and auditory short-term memory in adults and adolescents with Williams and Down syndromes (i.e., visual > auditory in Down syndrome; auditory > visual in Williams syndrome). Finally, studies of word and sentence processing in normal adults have demonstrated that the perception and use of grammatical morphemes can be selectively impaired when normal adults are tested under adverse processing conditions. If children with Williams and Down syndromes differ in their ability to perceive, encode, and remember phonologically weak morphemes, we might expect different profiles of grammatical development even though both groups are impaired equally at a semantic/conceptual level.

Do we know anything about differences between individuals with Williams and Down syndromes at the neural level that could be used to interpret these behavioral contrasts? Detailed neuroanatomical and neurophysiological comparisons have been conducted (Galaburda, Wang, Bellugi, & Rossen, 1994; Jernigan & Bellugi, 1990, 1994; Jernigan, Bellugi, & Hesselink, 1989; Jernigan, Bellugi, Sowell, Doherty, & Hesselink, 1993), but they raise more questions than they answer. Based on the behavioral contrasts observed in adults with Williams and Down syndromes, one might predict a difference along the left–right axis (e.g., left-hemisphere abnormalities in Down syndrome, right-hemisphere abnormalities in Williams). However, magnetic resonance imaging studies yield no evidence whatsoever for a left–right difference between these two populations. Instead, a range of significant differences is observed along the rostral–caudal axis (e.g., hyperfrontal presentation in Williams syndrome, hypofrontal in Down syndrome, controlling for overall cerebral volume, which is smaller than normal in both groups). In addition, the two groups show surprising differences in the shape and size of the cerebellum. This structure is about the right size in individuals with Down syndrome (controlling for overall brain size), but it is abnormally large in individuals with Williams syndrome. Furthermore, an area within the cerebellum called the vermis is also disproportionally large in individuals with Williams syndrome. This last finding is particularly interesting in view of reports by Courchesne and colleagues (Courchesne, 1991; Courchesne, Hesselink, Jernigan, & Yeung-Courchesne, 1987; Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988) suggesting that the neocerebellum may be disproportionately small in individuals with autism. Finally, electrophysiological studies suggest that the relative sparing of language displayed by older individuals with Williams syndrome may be mediated by an unusual form of brain organization for language, including unexpected evidence for greater activity over right-anterior regions during sentence processing (Neville, Mills, & Bellugi, 1994). In other words, the spared language of individuals with Williams syndrome may not be based on the neural mechanisms that normally are used to mediate language processing.

Infants of Substance-Abusing Mothers

As Dixon et al. (this issue) note in their article on early language development in children of substance-abusing mothers (CSAMs), the use of stimulant drugs has reached record levels among pregnant women, now estimated to occur in 5% to 12% of all pregnancies nationwide. Because many of these drugs can cross the placental barrier to alter the course of prenatal brain development, it is imperative that we learn more about the effect of prenatal drug exposure on language, cognition, and social development in this growing population of children at risk. This is no easy task because of the many environmental and biomedical confounds that plague any study of development in CSAMs.

Dixon et al. (this issue) focus on the early stages of language development in infants and toddlers with prenatal exposure to cocaine, methamphetamine, or both. Sixty children were studied, including a sample who had been removed from their birth homes and placed in foster care. Using a combination of laboratory measures and parental report (the MacArthur CDI), Dixon et al. report that CSAMs, as a group, performed below normal controls on several different measures of language

and communication. In fact, 5 times as many children as would be expected by chance fell into the 5th percentile on one or more language measures in the period between 8 and 30 months. Although this finding is discouraging, it is not surprising because it is consistent with a growing literature on early development in CSAMs. However, some of their other findings were quite unexpected.

First, children raised in foster care appear to be at greater risk than children who are in their home environment. This is surprising if we assume that foster parents provide better care than do biological mothers with a history of substance abuse. However, as Dixon et al. (this issue) note, this finding is subject to a number of confounds (e.g., mothers who lose or abandon their children to foster care may have had more serious problems, including, but not restricted to, a more serious drug habit).

Second, it looks like things may be getting worse instead of better for a substantial number of children. In fact, the lowest levels of performance in this study are observed in children between 25 and 30 months. Grammatical abilities, which emerge in this phase of development, were particularly at risk. Among the possibilities they entertain to explain this downward shift, Dixon et al. (this issue) underscore the growth of the frontal cortex that normally takes place around 24 months of age (e.g., the synaptic peak in the frontal cortex, illustrated in Figure 1). Recall that Bates et al. (this issue) found evidence that children with injuries to the left-frontal or right-frontal cortex perform especially poorly in the period from 19 to 31 months. As Dixon et al. note, there is some reason to believe that prenatal exposure to cocaine and methamphetamine has particularly deleterious consequences for dopaminergic areas on the frontal cortex. In fact, at least one study uncovered visible evidence of bilateral structural damage in the frontal cortex for a subset of the CSAMs population (Dixon & Bejar, 1989). Hence, the linguistic drop from 25 to 30 months may be a "sleeper effect," reflecting specific damage to frontal areas that takes on special importance at this point in development (see Goldman-Rakic, Isseroff, Schwartz, & Bugbee, 1983, for an animal model of this delayed frontal effect).

A third result in the Dixon et al. (this issue) article brings us back to the themes of variation and plasticity. Although some of the CSAMs in this study are at serious risk for language impairment (falling below the 5th percentile on several measures), others are doing surprisingly well. In fact, the mean percentile scores in Dixon et al.'s Study 1 range from 33% to 49% for expressive language, with standard deviations of more than 20 percentile points. Why are some children protected from the effect of prenatal drug exposure? Some of this apparent sparing may have to do with the biomedical and demographic confounds that plague this research area. Other factors may include timing and the amount of drug exposure, with consequences for the presence or absence of bilateral cortical damage. Another factor that we must keep in mind is the immense variation that also is observed in normal children growing up in healthy environments. Prenatal drug exposure occurs in children who might fall anywhere within the broad range of normal development. Hence, some of the sparing or impairment that we see in drug-exposed children may reflect individual differences that are independent of drug use (Bates & Appelbaum, 1994).

WHEN PLASTICITY FAILS: A FINAL COMMENT

The articles in this special issue underscore a vast range of variation in the early stages of language development, in normal children and in several different clinical populations. When the brain is damaged in some fashion during the early stages of development (due to accidents of nature or nurture), this damage is superimposed on a dynamic landscape of individual differences. Drawing on a growing literature in developmental neurobiology, I argue here that human brain development involves intricate, bidirectional interactions between maturation and experience. Mechanisms of plasticity and learning that are crucial to normal development come to play an even more important role when areas of the brain are injured early on. In a complex, nonlinear system of this kind, brain injury may serve to augment some forms of variation and to dampen others. It also may result in qualitatively different forms of brain organization for language and other higher cognitive processes.

Evidence for alternative forms of organization comes from the focal-lesion findings described by Bates et al. (this issue), and from the behavioral and neural findings for individuals with Williams and Down syndromes described by Singer Harris et al. (this issue). It is less clear how recovery from delay or resistance to damage are achieved by those late talkers and drug-exposed children who catch up with their age mates. However, there are good reasons to believe that the same additive and subtractive events that underlie normal brain development also are responsible for all these alternative patterns, for example, exuberant growth, competition, elimination of unsuccessful connections, and retention of those that work.

Although these "alternative brain plans" all are sufficient to support the acquisition of language, some of them are more successful than others. For example, some of the late talkers studied by Thal and her colleagues (this issue) went on to qualify for a diagnosis of specific language impairment (SLI). Like the children with Down syndrome described by Singer Harris et al. (this issue), children with SLI eventually acquire their grammar (see also Chapman, 1995), but they often display (among other things) sporadic, residual deficits in the production of grammatical morphemes (Johnston & Kamhi, 1984; Marchman, Wulfeck, & Weismer, 1995; Rice, 1996). Does this mean some children suffer from selective and irreparable damage to an autonomous "language organ" (Chomsky, 1980; M. Gopnik & Crago, 1991; Pinker, 1994)? And if this is the case, where is the language organ located in the brain? Why is plasticity for language blocked or limited in some clinical groups when children with extensive left-hemisphere damage are able to acquire language within the normal range?

SLI is particularly relevant to this argument. SLI has been under study for more than 30 years (West, 1962). It usually is defined as a syndrome in which expressive and (perhaps) receptive language abilities are 1 *SD* or more below the mean, and below the same child's performance IQ, in the absence of evidence for mental retardation, frank neurological impairment, abnormal hearing, socioemotional disorders, or sociodemographic conditions that could explain the disparity between language and other cognitive functions (Bishop, 1992; Bishop & Rosenbloom, 1987). Hence, by definition, SLI is supposed to represent an impairment that affects language and nothing else. Furthermore, it has been demonstrated repeatedly that grammatical morphology is particularly vulnerable in children with SLI (Johnston & Kamhi, 1984; Leonard, 1992), albeit with little evidence for the claim that regular morphemes are more vulnerable than are irregular morphemes (see Marchman et al., 1995, for a discussion of this point). Perhaps most important for our purposes, a number of studies have shown that SLI and associated disorders (especially dyslexia) tend to run in families (Bishop, 1992; Bishop & Rosenbloom, 1987; Pennington, 1991; Tallal, Ross, & Curtiss, 1989; Tallal, Townsend, Curtiss, & Wulfeck, 1991).

At face value, this looks like evidence for a specific and genetically based language organ, one that cannot be replaced if it is damaged early on. However, detailed studies of children with SLI have shown that the syndrome is not restricted to language, and it certainly is not restricted to grammar. For instance, Tallal and her colleagues (Tallal, 1988; Tallal, Stark, & Mellits, 1985) amassed a large and compelling body of evidence suggesting that children with SLI suffer from a deficit in the processing of rapid temporal sequences of auditory and (perhaps) visual stimuli. Other specific nonlinguistic deficits implicated in SLI include symbolic play in younger children (Rescorla & Goossens, 1992; Thal et al., 1991), aspects of spatial imagery in older children (Johnston, 1994), specific aspects of nonverbal attention (Townsend, Wulfeck, Nichols, & Koch, 1995), and a wide range of neurological soft signs (e.g., Trauner, Wulfeck, Tallal, & Hesselink, 1995). Particularly informative in this regard is a study by Tallal et al. (1991) of a very large sample of children with SLI comparing children with and without a family history of language disorders. There were no differences whatsoever between the two groups in the nature or extent of their language impairment (i.e., they were equally impaired, with specific pockets of vulnerability in the same areas); however, the subgroup with a family history of language disorders appeared to have a less specific disorder, with greater impairments on nonlanguage tasks, including a test of auditory temporal processing. At this writing, the nonlinguistic correlates of SLI have accumulated to the point where the original definition of the syndrome is at risk. For example, Bishop (1992) and Locke (1995) argued that children with SLI suffer from subtle but diffuse neurological impairments that have particularly cruel implications for language but which are in no way specific to it.

Although SLI is not as specific as we once believed, it is still the case that grammatical morphology is a highly vulnerable domain in SLI and in other disorders with an attested or suspected genetic base. As we just noted, a variant of the SLI pattern is frequently reported for children with Down syndrome. Although Singer Harris et al. (this issue) do not find a selective deficit in grammar in young children with Williams syndrome, other investigators have reported specific problems with grammatical morphology in older children with Williams syndrome. For example, Karmiloff-Smith and Grant (1993) reported that French-speaking adolescents with Williams syndrome have difficulty extracting and generalizing information about grammatical gender; and Volterra, Sabbadini, Capirci, Pezzini, and Osella (1995) reported that Italian-speaking children with Williams syndrome make a broad range of substitution errors in grammatical morphology, including many errors that are never observed in Italian children with Down syndrome, Italian children with SLI, or in normally developing Italian children at any stage of grammatical development. So we find grammatical impairments in several different genetically based syndromes. Could there be a gene for grammar that sits in close proximity to other genetically based functions, so close that these functions are felled by the same developmental axe?

Perhaps such a gene exists, but it has been demonstrated that grammatical morphology is selectively vulnerable in populations for whom genetic arguments are not viable. For example, problems with expressive grammar have been observed in both fluent and nonfluent aphasia (Bates, 1991; Pick, 1913/1973). They also have been observed in spoken and written language among neurologically intact individuals who are congenitally deaf (Caselli, Maragna, Pagliarini-Rampelli, & Volterra, 1994; Volterra & Bates, 1989). Problems with the receptive processing of grammatical morphemes have been observed in an even wider range of populations, including anomic aphasics who displayed no grammatical deficits in their spoken language (Bates et al., 1994; Devescovi et al., 1997) and a subset of elderly patients hospitalized for nonneurological disorders (Bates, Friederici, & Wulfeck, 1987). In fact, receptive agrammatism was induced in college students who were forced to process sentences under stress (e.g., perceptual degradation, cognitive overload, or both; Bates et al., 1994; Blackwell & Bates, 1995; Kilborn, 1991; Miyake, Carpenter, & Just, 1994). It appears that grammatical morphology is selectively vulnerable under a wide range of conditions, genetic and environmental. A parsimonious account of all these findings would be that grammatical morphology is a weak link in the processing chain, one that is highly likely to fall apart when things go awry.

That leaves us with a problem: Some children continue to suffer from communication disorders into adolescence and adulthood, whereas others recover and move into the normal range. Whether or not the grammatical deficits displayed in SLI and other syndromes are specific to language, their persistence cannot be denied. Why are children with unilateral left-hemisphere injuries able to overcome

their initial deficits and find a workable solution, whereas populations of children with no apparent lesions to the classical brain region have persistent problems (e.g., SLI, Down syndrome, autism, and a number of other groups)? What factors might account for the failure of plasticity in these populations? At least three possibilities come to mind.

One possibility might be that some children with persistent deficits do have damage to the classical language zones, but this damage is so subtle that other regions of the brain do not come to the rescue (Galaburda, Menard, & Rosen, 1994). In other words, they keep trying to solve the language problem with functional but inefficient parts. In this regard, consider the data in Figure 2, taken from Irle's (1990) review of more than 200 primate lesion studies. The dotted line in Figure 2 represents findings from Lashley's (1950) classic article, In Search of the Engram, which showed the effect of lesion size on subsequent learning ability in adult rats. These data indicate that learning ability is a linear function of lesion size, a finding that is virtually independent of lesion location. These are, of course, the findings that led Lashley to propose his famous principles of mass action and equipotentiality. By contrast, the full line in Figure 2 represents findings from Irle's meta-analysis of lesion size and learning ability in primates. This function is noteworthy in two key respects: (a) Primates are a lot smarter than rats (performing very well with less than half a brain), and (b) lesion size has a nonlinear, U-shaped effect on learning ability. That is, the worst performance is observed in primates with midsized lesions; animals with very small or very large lesions perform much better. Obviously, this nonlinear function cannot be extrapolated very far ("Gee, if I can do so well with half a brain, imagine how well I could do if they took the whole thing out!"). Nevertheless, Irle's findings for lesions in the middle range demand an explanation.

Irle's (1990) explanation for this U-shaped function is based on what I would call the "fresh start hypothesis." That is, primates with very large lesions are forced to adopt a completely new learning strategy, based on the remaining healthy tissue (i.e., a fresh start). By contrast, animals with midsized lesions persist in their efforts to learn with familiar strategies, based on damaged and inefficient neural mechanisms. Similar accounts have been offered to explain why some epileptic children improve markedly on linguistic tasks following a left hemispherectomy (e.g., the right hemisphere is finally released for service without interference from the left hemisphere (F. Vargha-Khadem, personal communication, July 1995). In the same vein, it is possible that some populations of children with communication disorders are working with functional but inefficient neural tools, preventing them from taking a fresh start with one of the alternative brain plans adopted by children with frank lesions to the left-perisylvian cortex.

A second possibility may be that children with persistent language impairments suffer from bilaterally distributed cortical microlesions, too small to be detected in most magnetic resonance imaging studies, but serious enough to deter and delay

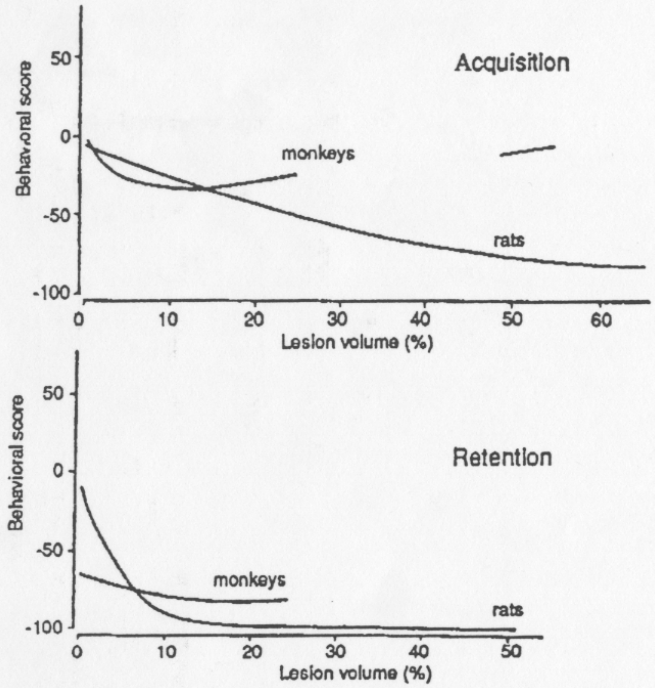

FIGURE 2 Effects of lesion volume on acquisition (top) and retention (bottom) of a new skill. There is a linear relation between lesion volume and loss of new learning in rats, whereas monkeys maintain relatively high levels of learning even after 40% to 50% of the cortex is lesioned (Reprinted from *Brain Research Review, 15,* by E. Irle, "An Analysis of the Correlation of Lesion Size, Location, and Behavioral Effects in 283 Published Studies of Cortical and Subcortical Lesions in Old-World Monkeys," pp. 181–213, 1990, with kind permission from Elsevier Science - NL, Sara Burgerhartstraat 25, 1055 KV Amsterdam, The Netherlands.)

language learning. In other words, these children do not arrive at an alternative organization for language because there simply is not enough healthy cortical tissue available to support a new brain plan. Some evidence for this view comes from autopsy studies of dyslexic individuals by Galaburda and his colleagues (Galaburda, 1994; Galaburda & Livingstone, 1993; Galaburda et al., 1994), who reported a high incidence of ectopias (literally "brain warts") and other abnormalities in both

cerebral hemispheres. Galaburda insisted that these abnormalities are more common in the left hemisphere, but their diffuse and unpredictable distribution suggests to me that a more pervasive and diffuse defect may be present across the cerebral cortex (for related results in magnetic resonance imaging studies of children with SLI, see Trauner et al., 1995).

Finally, we should consider the possibility that the neural deficits underlying persistent language impairment are caused not by cortical abnormalities but by some kind of abnormality at a subcortical level that affects the learning process. For example, Eisele and Aram (1995) argued that linguistic deficits are more severe and more persistent in children with focal injuries involving the basal ganglia and related subcortical structures. The cerebellum is another region that has been implicated in language and cognitive disorders, including the contrasting patterns in neo- and paleocerebellar areas described previously for Williams syndrome. Another candidate might be the thalamus, a critical input station that is known to play a major role in the establishment of cortical maps. The major point for our purposes is that language learning in the child and maintenance and the use of language in the adult are not the same thing. Lesions that affect early learning may have no discernible effect on adult performance, and vice versa. We may have to look outside the cortex to find the neural culprit when developmental plasticity fails.

We do not yet know the answer to these and related questions about the origins of communication disorders. However, there are many reasons to be optimistic that answers soon will be obtained. Twenty years of research in developmental neurobiology have underscored the plastic and activity-dependent nature of cortical specialization in vertebrate animals. These lessons apply equally to brain and behavioral development in human beings. Our understanding of the nature and etiology of communication disorders has been enriched greatly by detailed behavioral studies such as the ones reported here, which identify these deficits at their point of origin and lay out trajectories of growth and change from infancy to adolescence. New technologies have made it possible to complement these behavioral studies with structural and functional studies of the developing brain. The message that has emerged from these studies to date supports a dynamic, bidirectional theory of brain and behavioral development, a perspective that gives great cause for hope to parents and scientists alike.

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Origins of Language Disorders

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This special issue represents the initial products of the first 5 years of a multiproject center titled Origins of Communicative Disorders. The center is part of the Project in Cognitive and Neural Development at the University of California, San Diego. As the title implies, the common goal of investigators involved in this project is to describe the development of communicative skills from their earliest measurable points so that factors that characterize the earliest stages of communicative disorders can be teased apart from those that lead to the development of typical language ability in later childhood.

To meet this goal our studies were designed to obtain information about change over time, to compare individuals, and to compare groups of subjects. Many of our studies are prospective in nature, beginning when the children are at prelinguistic stages and following them through the period during which most basic language skills are acquired. This labor-intensive longitudinal design is combined with cross-sectional studies to increase the numbers of subjects at each datapoint. Together, these research designs provide a powerful means of identifying critical factors related to change over time.

The research in this center also focuses on cross-population comparisons of populations known to be at risk for communicative disorders. The populations studied in the first 5 years of the center included children with late onset of language ("late talkers"), children with pre- or perinatal focal brain injury, children with syndromes that create cognitive impairments (Williams syndrome and Down

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syndrome), and children with prenatal exposure to the stimulant drugs cocaine or methamphetamine.

The articles in this issue illustrate three themes that characterize our research with these populations. First, some of the studies are designed to search for patterns of selective impairment or selective sparing; that is, patterns of association and dissociation within populations and across populations in aspects of language together with those aspects of cognition that may be related to language. Second, these studies emphasize the way communicative ability changes over time in these at-risk and typically developing populations. Third, we are able to pursue questions about brain–behavior relations using new technologies (i.e., event-related brain potentials associated with linguistic and nonlinguistic stimuli), specific populations (e.g., children with well-defined focal brain injury), or both.

In addition to these themes, the five studies that follow share a common methodology: a parent report instrument called the MacArthur Communicative Development Inventory (CDI). The instrument, which has been described in detail in several publications (see especially Fenson et al., 1993; Fenson et al., 1994), was developed over a 20-year period by several of the investigators in our San Diego group, working closely with colleagues in Seattle, New Haven, and Rome. One of the most striking results reported in those studies was the wide range of individual variability present in all aspects of language measured by the CDIs. In fact, the authors concluded that variation is the rule in early language development, and there is no such thing as a "modal child." This makes an endeavor to identify the origins of language disorders not only more challenging but also more urgent, given federal mandates to identify and treat infants and toddlers with communicative disorders (Education of Handicapped Act Amendment, 1986).

Each of the articles in this issue includes a description of those aspects of the CDI that are particularly important for that study. However, two general points are important enough to warrant mention here. First, the reliability and validity of the CDI are both well documented. As reported by Fenson et al. (1994), all the scales on the CDI (which include vocabulary comprehension and production, gesture production, and grammatical complexity) showed strong internal consistency, and longitudinal samples across a 6-month period demonstrated good test–retest reliability as well. Studies of concurrent validity included comparisons of the CDI to standardized and experimental measures of language, including the language tasks on the Bayley Scales of Infant Development, standardized tests of language such as the Expressive One-Word Picture Vocabulary Test (EOWPVT) and the Preschool Language Scale, and various measures from spontaneous language samples (Bates, Bretherton, & Snyder, 1988; Beeghly, Jernberg, & Burrows, 1989; Dale, 1991; Dale, Bates, Reznick, & Morisset, 1989; O'Hanlon & Thal, 1991). Because a number of measures were used in these studies of concurrent validity, it was possible to get some estimates of the range of items sampled through each methodology. Dale, for example, carried out a multiple correlation between children's scores on the CDI and the EOWPVT and a type-token ratio derived from language samples from the same children. The multiple correlation between the children's scores on the vocabulary measure from the CDI and the two vocabulary measures obtained in the laboratory was higher than either of the simple correlations of the single tests to the CDI. In addition, a multiple regression analysis showed that each was related to a distinct proportion of the variance in the CDI vocabulary. The author concluded that the toddler CDI vocabulary checklist assesses a broader vocabulary range than does either the direct observational measure obtained from language samples or the standardized test. Similarly, the sentence complexity subscale and the mean length of the three longest utterances are correlated strongly with the mean length of utterance obtained from a spontaneous language sample. Thus, all the measures obtained from the MacArthur CDI are strongly predictive of the actual behaviors measured in spontaneous communicative situations, and they may provide a more representative sample of children's early language than do other means of sampling early language.

A second important point to make about the CDI is that it provides a method that is comparable across different populations and different laboratories. In the past, research on language acquisition was hampered by the need for different methodologies for children with different handicapping conditions, and by the fact that methodologies were not easy to replicate across laboratories. Within the age range for which the CDIs are normed we finally have a methodology that allows us to make more valid and reliable cross-population and cross-laboratory comparisons.

What follows are four articles, each focused on one or two populations known to be at risk for communicative disorders, and one article concerning changes in brain activity associated with changes in language ability in typically developing children. In the first article, Thal, Bates, Goodman, and Jahn-Samilo examine the continuity of development in children at the extremes of the normal continuum and attempt to determine whether children who start out late or early continue to be late or early 6 months later. They show that there is significant continuity at the group level, and that we do not yet possess the means to predict trajectories of development for individual children.

Bates et al. focus on children with pre- and perinatal focal brain injury, in three cross-sectional studies of communication and language across the period in which typical children progress from first words to grammar. The authors explore the effect of lesion location on language and find effects different from those reported for adults with similar lesions. In the age range between 10 and 17 months, children with right-hemisphere damage are at greater risk for delay in comprehension and use of symbolic gestures than are children with left-hemisphere damage. In addition, throughout the period from 10 to 44 months, children with left-temporal-lobe damage are at risk for greater delay in expressive vocabulary and grammar than are children with damage in other areas.

Singer Harris, Bellugi, Bates, Jones, and Rossen explore the different trajectories and associated patterns of gesture use, vocabulary, and grammar in children with Williams syndrome and Down syndrome. They report that both groups of children are equally and seriously delayed in the early stages of language development. However, the patterns of development differ. In the earliest stages children with Down syndrome use significantly more gestures than do children with Williams syndrome. At a later point, when the vocabulary levels of both groups were high enough to support the development of grammar, children with Williams syndrome began to develop grammatical structure in a manner similar to normally developing children. By contrast, children with Down syndrome demonstrate an atypical dissociation between vocabulary size and the development of grammatical structure.

Dixon, Thal, Potrykus, Bullock Dickson, and Jacoby describe an exploratory study of two groups of children with prenatal exposure to stimulant drugs. One group comprises a large clinical sample of children in a wide range of home settings. The second is a smaller experimental group living in stable, middle-class adoptive homes. Dixon et al. report significant delay in all aspects of language for a large proportion of these children. However, there is also a wide range of variability in this population, with some children falling well within the normal range. In both groups, older children performed more poorly than did younger children. Dixon et al. suggest that this "delayed impact" could reflect damage to frontal lobe structures that develop relatively late, compared with other parts of the cerebral cortex.

Finally, Mills, Coffey-Corina, and Neville describe developmental changes in the brain activity of typically developing children that are linked to comprehension of single words at 13 and 20 months of age. They show that brain activity changes from bilateral activity that is broadly distributed over anterior and posterior portions of the brain at 13 to 17 months, to more specifically localized activity in the temporal and parietal regions of the left hemisphere at 20 months of age. However, this is not a simple age-related phenomenon: Children with higher levels of comprehension are reported to have more localized brain activity in both groups than do those with lower levels of comprehension.

Taken together, the articles in this issue provide a comprehensive picture of early language development and its neural correlates, across a range of typical and atypical populations. By looking at language abilities from their point of origin, that is, from the very first signs of word comprehension to the emergence of grammar, the authors hope to lay the foundation for future research on the nature and etiology of communication disorders.

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