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Commentary

Explaining and interpreting deficits in language development across clinical groups: Where do we go from here?

Elizabeth A. Bates*

Department of Cognitive Sciences, Dept. 0526, 9500 Gilman Drive, University of California, San Diego, La Jolla, CA 92093-0526, USA

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The papers in this issue present a series of comparative studies of language processing and language development across clinical populations, including studies in the earliest stages of language development, as well as aspects of grammar, narrative, and verbal memory across the elementary school years. The populations covered include "late talkers," children with congenital injuries to either the left or right hemisphere, children with Williams Syndrome, children with Down Syndrome, children with a diagnosis of specific language impairment (behaviorally defined), and a range of typically developing controls for each of these groups.

As Holland points out in her commentary, two of the most surprising findings across these studies include the following.

(1) Despite differences in rate of development, the sequences and error types observed are (with a few interesting exceptions) remarkably similar across these very different clinical groups.

It appears that sequences and error types are determined primarily by the "problem space" posed by a particular language (in this case English), so that virtually all children who enter into this problem space end up behaving in much the same way, albeit at variable rates. Reilly et al. (this issue) suggest a metaphor for results like these: all of these children have undertaken a journey along the same highway, but some of them are in the slow lane, with occasional stops along the road.

(2) Children with well-defined brain injuries of the kind that often lead to aphasia in adults perform within the normal range on most measures once they move into the elementary school years, with no trace of a selective disadvantage for children with left-hemisphere damage (LHD) compared to children with right-hemisphere damage (RHD). Indeed, regardless of lesion size, side or site, children with early focal brain injury often perform better than children with behaviorally defined language impairments of unknown origin.

This "second surprise" is clearly illustrated in all of the studies within the elementary school years, including the study by Wulfeck, Bates, Krupa-Kwiatkowski, and Saltzman on detection of grammatical violations, the Weckerly, Wulfeck, and Reilly study on generation of tag questions, and the study by Reilly, Losh, Bellugi, and Wulfeck of grammar and discourse using the Frog Story narratives. However, the paper by Thal, Reilly, Seibert, Jeffries, and Fenson on earlier stages of development shows that things did not always look so optimistic for children in the focal lesion (FL) population. In contrast with all the above-cited studies of children in the elementary school years, Thal et al. found no difference in mean level of language (assessed by free speech) for the late talkers and the focal lesion group at 36 months of age, when both groups were seriously and equally delayed relative to controls. However, the variance around the mean was significantly greater at 36 months for children with FL, suggesting that some have already started to "spurt" forward while others are still lagging markedly behind. This interpretation is bolstered by a small longitudinal study of 12 children with focal brain injury, who were far below controls in Mean Length of Utterance at 36 months, but well within the normal range (indeed, slightly above the mean) on the same measure at 5 years of age. Something happens between 3 and 5 years in the FL population, a window of recovery, compensation, reorganization and remarkable growth that is not observed in our other clinical groups.

In addition to these two points, I would add a third surprise to Holland's list.

* Fax: 1-858-822-5097.
E-mail address: bates@crl.ucsd.edu.

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(3) Despite claims to the contrary (especially in the popular press), children with Williams Syndrome are not language savants. Their performance on the language tasks represented in this special issue is usually solidly linked to mental age, and often resembles performance by children with language impairments.

The latter finding is especially surprising, in view of claims by some investigators (e.g., Piattelli-Palmarini, 2001; Pinker, 1994; Smith & Tsimpli, 1995) that Williams Syndrome and SLI represent a double dissociation or "mirror image" relationship between language and non-verbal cognition (i.e., Language > Cognition in Williams; Language < Cognition in SLI). For example, Piattelli-Palmarini (2001) claims that "Children with Williams Syndrome have barely measurable intelligence," but "an exquisite mastery of syntax and vocabulary," even though they supposedly are "unable to understand even the most immediate implications of their admirably constructed sentences" (p. 887). Based on the putative double dissociation between Williams Syndrome and SLI, Smith and Tsimpli (1995) conclude that "It is no longer plausible to talk of "cognitive prerequisites" for language. This has been apparent on the basis of many studies, especially of Williams Syndrome" (p. 190). Results presented in this special issue are at odds with these rather strong conclusions.

To be sure, some qualitative differences between groups do emerge here and there within this special issue. For example, in the Marchman, Saccuman and Wulfeck paper on generation of past tense markings, overall performance on this grammar task was strongly tied to receptive vocabulary age (on the Peabody Picture Vocabulary Test), regardless of age or clinical group. However, children with SLI did produce a significantly larger number of zero-marking errors, compared with controls or children with FL. Interestingly, Marchman has observed a similar propensity toward zero-marking errors in earlier studies of children who are now in the normal range even though they started out as "late talkers" (Marchman, 1997, 1999). Conversely, children who take off quickly in the first stages of vocabulary development tend to produce a higher proportion of substitutions and overgeneralizations on the past tense task, even though they have moved back toward the mean in their overall rate of development. So it appears that a child's position on a continuum of omission ↔ substitution errors may be determined by overall rate and style of language development. This raises the possibility that children with SLI produce more zero errors for reasons that are linked to rate of development, a by-product of their clinically significant delays but one that should not be viewed as a special clinical marker of SLI.

In their paper on verbal learning and memory, Nichols and colleagues also observe some subtle qualitative differences among children with SLI, children with focal brain injury, and children with Williams Syndrome or Down Syndrome. For example, children with Williams Syndrome performed poorly on the long-term memory component of the verbal learning task, but the same children performed well in the short-term memory components of the same task. This finding is compatible with studies by other investigators suggesting strengths in auditory short-term memory within this group (Vicari, Brizzolara, Carlesimo, Pezzini, & Volterra, 1996; Wang & Bellugi, 1994). Such results raise the possibility that group differences in language may be secondary to differences in the processing dimensions that are important (perhaps crucial) for language, but not specific to language.

Of the "three surprises" laid out in this volume, the first result (qualitative parallels across clinical groups) underscores the importance of interpreting clinical symptoms with reference to the communicative and cognitive problems that all these children are trying to solve in the course of language learning. The second result (developmental progress in all clinical groups, but especially marked progress and recovery in the FL group) testifies to the role of behavioral and neural plasticity in language learning. Learning "sculpts" the brain, and in the case of children with unilateral injuries to what is (we may presume) an otherwise normal brain, the mechanisms that support learning in healthy children are apparently sufficient to support the development of normal language abilities in the face of early injury. So why do we not see the same degree of recovery in children with SLI, Down Syndrome, or Williams Syndrome? Presumably because (as Holland also states) children in the latter groups are delayed in language because of neurological impairments that are either so broadly distributed (bilateral and pervasive) that there is not enough healthy tissue to go around, or else located in "gate-keeper" systems that modulate activity all over the brain (e.g., cerebellar or subcortical structures that are crucial for the acquisition of language).

The relative sparing of auditory short-term memory in Williams Syndrome suggests a broader conclusion: specific profiles of sparing or impairment within language may reflect one or more deficits in information processing that have specific (and sometimes contrasting) consequences for language—even though they are not restricted entirely to the language domain (e.g., a relative sparing of auditory perception and auditory short-term memory in Williams Syndrome, despite mental retardation, and associated deficits in long-term memory).

All of these results have implications for future research in communicative disorders, in three directions.

First, to increase our understanding of those linguistic symptoms that are common across clinical groups, it may be useful to conduct studies in which typically developing children are asked to process words and sentences under "stressed" processing conditions.

An analogous strategy has been adopted in several recent studies of linguistic symptoms in adults with aphasia (Blackwell & Bates, 1995; Dick et al., 2001; Miyake, Carpenter, & Just, 1994). In all these studies, common findings regarding points of vulnerability in word and sentence processing by aphasic patients have been induced in normal adults who are forced to process words and sentences under adverse processing conditions. Of particular interest here, specific-looking deficits in grammatical morphology and/or complex syntax can be induced in normal adults under a range of conditions, including temporal compression of visual or auditory stimuli (to simulate limits in speed of information processing), filtering and/or superimposed noise (to simulate limits in the "bottom up" integrity of perceptual input), or cognitive overload (to simulate limits in attention and/or working memory). I am not suggesting that *all* linguistic symptoms can be simulated simply by placing the normal system under stress. However, it will be useful and informative to determine which symptoms do reflect breakdowns in the normal system, and which symptoms represent qualitative variations or deviations that are never observed in healthy speaker/listeners, under any circumstances. The "simulation of aphasia" strategy that has worked so well in research on adults could be fruitfully extended to studies of word and sentence processing in children.

Second, to increase our understanding of both plasticity and limits on plasticity, we need more longitudinal studies of development and change across the period in which language is typically acquired. This will give us information about the *processes* or *mechanisms* that are employed in language learning, as opposed to a static characterization of *linguistic products*. Even more important, the study of language-as-a-moving-target may also lead to improved programs for intervention and rehabilitation in those children who are falling behind when they are left on their own.

Third and finally, I believe we may have to face up to the possibility that the causes of language impairment are not located inside the language system. In the last few decades, the popular press and much of the academic world has been engaged in a hot pursuit of genes for grammar, instincts for language, and special and separate neural mechanisms or "organs" for phonology, semantics and morphosyntax. It has been suggested that the brain is like a Swiss Army knife, filled with specialized tools, each intricately designed for a specific job (Duenwald, 2002; Horgan, 1995; Pinker, 1994). But the Swiss Army knife may not be the right metaphor for the way that our brains have evolved to acquire language, culture and technology. As my colleagues and I have suggested elsewhere (Bates, 1999; Bates, Benigni, Bretherton, Camaioni, & Volterra, 1979; Elman et al., 1996), language may be a new machine that Nature has constructed out of old parts.

To replace the Swiss Army knife, I would like to suggest that we consider the giraffe's neck. The giraffe has a specialized organ that permits it to eat leaves from the high branches of a tree. This is possible because of quantitative changes in the general-purpose vertebrate neck plan. And yet the giraffe still uses its neck for the older purposes that necks serve in other species: turning the head with its associated sense organs, sending food down, sending air and blood in both directions. In other words, the giraffe's neck has kept all of its "day jobs" while adding on a new function, insured by quantitative changes that make the new function possible without placing older functions in peril. The uniquely human capacity for language, culture and technology may have been acquired across the course of evolution by a similar process—quantitative changes in primate abilities that bring about and insure a qualitative leap in cognition and communication.

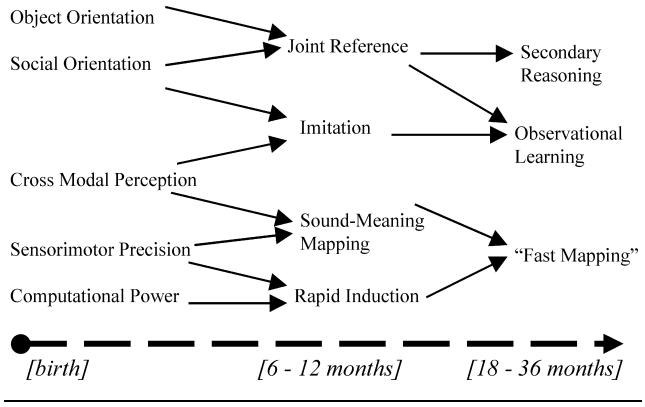
As an opening bid (or perhaps a down payment) on this "giraffe's neck" approach to language, Table 1 summarizes some proposed functional infrastructures for language, starting with abilities that are present at birth and extending these (in increasingly complex combinations) across the first 3 years of human life.

The "starter set" in the left-hand column comprises abilities that are present in other primates, but are known to be exquisitely well-tuned in the human infant at birth or shortly thereafter.

1. A peculiar fascination with small objects, evident in visual tracking and in a propensity to manipulate those objects with the hands and mouth.
2. A strong tendency to orient toward social objects (especially human faces and voices).
3. A remarkable capacity for cross-modal perception, i.e., the ability to detect and contemplate spatial and temporal invariants in sound, vision, and tactile stimulation. To offer just one example, when neonates are given two pictures to contemplate (a smooth figure and a jagged figure), and simultaneously given one of two different pacifiers (a smooth pacifier, or a textured, "bumpy" pacifier), they tend to look more often at the visual figure that most closely matches the object that has been placed in their mouths (Meltzoff & Borton, 1979).
4. A brain that has been colonized in utero by the body, organized into sensory and motor maps that already work together with surprising precision, supporting increasingly fine-grained analyses of sensory and motor experience.
5. An impressive amount of computing power, supporting rapid learning.

This is not intended to be an exhaustive list—far more may be required in order to build a human being (Mandler, 1988, 1992). However, if one follows these starter skills in the left-hand column across the next 3 years of life, it is possible to see how these initial skills

Table 1
Functional infrastructure for language



might converge onto later developmental milestones that correlate with the emergence of language, and may indeed be both necessary and sufficient for language development to occur.

Let us consider some of the most important changes that are known to take place during the first 18 months of life, viewed as the emergent products of the "starter skills" listed in the left-hand column.

1. The competition between orientation toward small objects and orientation toward human beings appears to lead human babies into a solution called *joint attention*. This is a process that is highly developed in our species (Tomasello & Call, 1997), permitting infants and their partners to contemplate the same set of objects and events in the world. This skill first manifests itself in gaze following (between 3 and 9 months), and culminates in both the production and comprehension of pointing and other deictic gestures (between 9 and 18 months) (Butterworth & Jarrett, 1991). Although joint attention is not, strictly speaking, a linguistic skill, it places human children right where they need to be in order to acquire names for things.
2. The infant's fascination with human beings also intersects with cross-modal perception to permit the emergence of increasingly sophisticated forms of imitation, i.e., reproduction of adult sounds and action, including those actions, gestures and sounds that adults produce with just those objects of interest that the couple has come to share. Again, imitation is not, in and of itself, a linguistic skill. But it is a skill in which human infants outperform all other species on the planet (Visalberghi & Fragaszy, 2002), permitting them to pick up the actions, gestures and sounds that comprise not only language, but also culture and technology.
3. By intersecting cross-modal perception with sensorimotor precision, human infants develop an ability to associate sounds with their meanings. At one year of age, this ability appears to be quite plastic—infants will happily pick up any sound, gesture, word or sign that is reliably associated with objects and events of interest (Namy & Waxman, 1998; Volterra & Erting, 1990). However, by 18 months of age, hearing children have developed a marked preference for word-like sounds (especially if those words follow the phonotactic constraints of the language environment in which they have been bathed—Jusczyk, 1997). In the same vein, deaf children exposed to a signed language show a preference for gestures that "look like" the signs of the language in which they have been raised. Thus, a general-purpose ability to match sounds, gesture and their meanings is sculpted across the first 18 months of life into a device that works very well for language learning.
4. When the above skills are crossed with the huge computational capacities of the human brain, the result appears to be a capacity for rapid statistical induction within and across domains (Gomez & Gerken, 2000; Haith & Benson, 1998; Saffran, Aslin, & Newport, 1996). This capacity has no parallel in other species (although some primate cousins come very close—Ramus, Hauser, Miller, & Morris (2000)).

With high-quality input and ample opportunities for learning, this entwining of non-linguistic skills leads human children into a capacity for language, culture and technology that is truly unique to our species. But the story is not over. Between 18 and 36 months of age, the same skills continue to play into each other, supporting an ever-richer complex of human-specific skills. For example, the combination of joint reference and imitation appears to lead our children into two closely related abilities that are, according to Tomasello and Call (1997), the hallmark of our species: secondary reasoning (i.e., the ability to reason about the thought processes of others, also called "theory of mind"—Gopnik & Meltzoff, 1997; Gopnik, Meltzoff, & Kuhl, 1999) and observational learning (the ability to learn new skills through silent observation of others). In the same vein, the human toddler's capacity for sound-meaning mapping and rapid statistical induction leads children (albeit gradually, a few steps at a time—Hoff & Naigles, 1999) into a capacity called "fast mapping"—the ability to learn a new word (or non-linguistic marker—Markson & Bloom, 1997) in only one or two trials. Although we are not the only species capable of one-trial learning, we appear to have elevated this kind of "declarative memory" to a level that is quantitatively if not qualitatively unique. This ability has been demonstrated most clearly within the lexical domain (i.e., experimental studies of word learning), but recent studies of grammatical development in the same age range suggest that grammar and the lexicon develop hand-in-hand, manifesting a tight interdependence that is exceptional in its size and robustness (Bates & Goodman, 1997; Marchman, Martínez-Sussmann, & Dale,

Table 2
Developmental language disorders: Hypothesized deficits in the functional infrastructure for language

Syndrome	Hypothesized functional deficit
Specific language impairment	Impairments in one or more aspects of information processing (spectral, temporal, attention, and memory overload)
Autism	Impaired social motivation and/or joint attention, with or without overall reductions in computing power
Williams Syndrome	Impaired cognitive capacity (computing power) with spared auditory processing and spared social motivation
Down Syndrome	Impaired cognitive capacity (computing power) with impaired auditory processing and spared social motivation
Early focal brain injury	Mild residual motor deficits, hemisphere-specific visual–spatial deficits, with small (subclinical) reductions in IQ (~5–7 points)

under review). Hence "fast mapping" may be just as important for grammar as it is for vocabulary.

Although I could be wrong, I am willing to go out on a limb here, proposing that the "functional infrastructures" listed in Table 1 may be all that is required for the construction of a "language acquisition device." None of these skills is unique to language, and indeed they seem to have evolved to permit the acquisition of culture and technology as well as language. But a defect in one or more of these infrastructures could have serious consequences for language learning. In fact, I would like to suggest that deficits in language and cognition might be traced directly back to these functional infrastructures, and the neural substrates that mediate them. If the giraffe's neck view of our capacity for language is correct, then we may never observe a true case of specific language impairment. If language is impaired in some fashion, then we should always be able to detect at least some subset of non-linguistic skills that are also impaired.

Table 2 presents some speculations about possible non-linguistic causes of the language and cognitive deficits that characterize some of the populations represented in this special issue, as well as another population that we have just begun to study in research group: children with autism spectrum disorders, including autism, pervasive developmental disorders (PDD) and Asperger's Syndrome. The suggestions in Table 2 are admittedly speculative and they are not original to me or to our group. For example, we are not alone in suggesting that autism spectrum disorders may result from deficits in social orientation (Baron-Cohen, Leslie, & Firth, 1985; Happé & Frith, 1996). In the same vein, so-called SLI may be associated with deficits in one or more domains of information processing that are not specific to language, even though their consequences for language are especially serious (e.g., deficits in perceptual timing, deficits in perceptual grain, deficits in attention and working memory). The contrasts that have been observed to date between children with Williams Syndrome and children with Down Syndrome who are matched for mental age may reflect an important difference between these two groups in the integrity of auditory vs. visual perception and memory (Bates, Thal, Finlay, & Clancy, 2002; Wang & Bellugi, 1994). A similar story may apply to the often transient deficits that we and others have observed in children with early focal brain injury. Non-linguistic deficits can have serious consequences for language, temporarily or on a more protracted basis.

If this approach is correct, then we may need to rethink completely the approach to diagnosis and intervention in communicative disorders that has characterized much of the research in our field up to this point. In my view, this may be the most important lesson to be learned from the papers in this special issue.

References

Baron-Cohen, S., Leslie, A. M., & Firth, U. (1985). Does the autistic child have a "theory of mind"? *Cognition, 21*, 37–46.

Bates, E. (1999). Plasticity, localization, and language development. In S. H. Broman, & J. M. Fletcher (Eds.), *The changing nervous system: Neurobehavioral consequences of early brain disorders* (pp. 214–253). New York: Oxford University Press.

Bates, E., Benigni, L., Bretherton, I., Camaioni, L., & Volterra, V. (1979). *The emergence of symbols: Cognition and communication in infancy*. New York: Academic Press.

Bates, E., & Goodman, J. (1997). On the inseparability of grammar and the lexicon: Evidence from acquisition, aphasia ,and real-time processing. *Language and Cognitive Processes, 12*(5/6), 507–586 [redacted version reprinted in M. Tomasello & E. Bates (Eds.), *Essential readings in language development*. Oxford: Basil Blackwell, 2000].

Bates, E., Thal, D., Finlay, B. L., Clancy, B. (2002). Early language development and its neural correlates. In: F. Boller & J. Grafman (Series Eds.) & S.J. Segalowitz & I. Rapin (Vol. Eds.), *Handbook of neuropsychology, Vol. 8: Child neurology* (2nd ed., pp. 109–176). Amsterdam: Elsevier Science.

Blackwell, A., & Bates, E. (1995). Inducing agrammatic profiles in normals: Evidence for the selective vulnerability of morphology under cognitive resource limitation. *Journal of Cognitive Neuroscience, 7*(2), 228–257.

Butterworth, G., & Jarrett, N. (1991). What minds have in common is space: Spatial mechanisms serving joint visual attention in infancy. *British Journal of Developmental Psychology, 9*, 55–72.

Dick, F., Bates, E., Wulfeck, B., Utman, J., Dronkers, N., & Gernsbacher, M. (2001). Language deficits, localization and grammar: Evidence for a distributive model of language breakdown in aphasics and normals. *Psychological Review, 108*(4), 759–788.

Duenwald, M. (2002, October 1) Seeking deeper meaning in the babbling of babies. *The New York Times*, p. D5.

Elman, J., Bates, E., Johnson, M., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness: A connectionist*

perspective on development. Cambridge, MA: MIT Press/Bradford Books [Paperback edition issued 1998; Japanese translation 1998].

Gomez, R. L., & Gerken, L. (2000). Infant artificial language learning and language acquisition. *Trends in Cognitive Sciences, 4*, 178–186.

Gopnik, A., & Meltzoff, A. N. (1997). *Words, thoughts, and theories*. Cambridge, MA: MIT Press.

Gopnik, A., Meltzoff, A. N., & Kuhl, P. K. (1999). *The scientist in the crib: Minds, brains, and how children learn*. New York: William Morrow.

Haith, M., Benson, J. (1998). Infant cognition. In W. Damon (Series Ed.) & D. Kuhn & R. Siegler (Vol. Eds.), *Handbook of child psychology: Vol. 2. Cognitive, language, and perceptual development*. New York: Wiley.

Happé, F., & Frith, U. (1996). The neuropsychology of autism. *Brain, 119*, 1377–1400.

Hoff, E., Naigles, L., (1999, July). *Fast mapping is only the beginning: Complete word learning requires multiple exposures*. Paper presented at the VIIIth International Congress for the Study of Child Language, San Sebastian, Spain.

Horgan, J. (1995). The new Social Darwinists. *Scientific American, 273*(4), 174–181.

Jusczyk, P. W. (1997). Finding and remembering words: Some beginnings by English-learning infants. *Current Directions in Psychological Science, 6*, 170–174.

Mandler, J. (1988). How to build a baby: On the development of an accessible representational system. *Cognitive Development, 3*, 113–136.

Mandler, J. (1992). How to build a baby II: Conceptual primitives. *Psychological Review, 99*(4), 587–604.

Marchman, V.A. (1997). Models of language development: An emergentist perspective. In T. Dolan (Ed.), *Special issue on Communication Processes and Developmental Disabilities, Mental Retardation and Developmental Disabilities Research Reviews*, 3, 293–299.

Marchman, V.A., (1999, April). *The impact of vocabulary growth on morphological productivity*. Paper presented at the Symposium on "Early Vocabulary Growth: Mechanisms of Change" at the Biennial Meeting of the Society for Research in Child Development, Albuquerque, N.M.

Marchman, V.A., Martínez-Sussmann, C., Dale, P.S. (under review). The language-specific nature of grammatical development: Evidence from bilingual language learners.

Markson, L., & Bloom, P. (1997). Evidence against a dedicated system for word learning in children. *Nature, 385*, 813–815.

Meltzoff, A. N., & Borton, R. W. (1979). Intermodal matching by human neonates. *Nature, 282*, 403–404.

Miyake, A., Carpenter, P., & Just, M. (1994). A capacity approach to syntactic comprehension disorders: Making normal adults perform like aphasic patients. *Cognitive Neuropsychology, 11*(6), 671–717.

Namy, L. L., & Waxman, S. R. (1998). Words and gestures: Infants' interpretations of different forms of symbolic reference. *Child Development, 69*(2), 295–308.

Piattelli-Palmarini, M. (2001). Grammar—The barest essentials. *Nature, 411*, 887–888.

Pinker, S. (1994). *The language instinct: How the mind creates language*. New York: Williams Morrow.

Ramus, F., Hauser, M. D., Miller, C., & Morris, D. (2000). Language discrimination by human newborns and by cotton-top tamarin monkeys. *Science, 288*(5464), 349–351.

Saffran, E. M., Aslin, R. N., & Newport, E. L. (1996). Statistical learning by 8-month-old infants. *Science, 274*, 1926–1928.

Smith, N., & Tsimpli, I.-M. (1995). *The mind of a savant: Language learning and modularity*. Oxford: Basil Blackwell.

Tomasello, M., & Call, J. (1997). *Primate cognition*. New York: Oxford University Press.

Vicari, S., Brizzolara, D., Carlesimo, G. A., Pezzini, G., & Volterra, V. (1996). Memory abilities in children with Williams syndrome. *Cortex, 32*(3), 503–514.

Visalberghi, E., & Fragaszy, D. (2002). Do monkeys ape? Ten years after. In K. Dautenhahn, & C. L. Nehaniv (Eds.), *Imitation in animals and artifacts* (pp. 471–499). Cambridge, MA: MIT Press.

Volterra, V., & Erting, C. (Eds.). (1990). *From gesture to language in hearing and deaf children*. New York: Springer.

Wang, P., & Bellugi, U. (1994). Evidence from two genetic syndromes for a dissociation between verbal and visual short-term memory. *Journal of Clinical & Experimental Neuropsychology, 16*(2), 317–322.