Discussions in
NEUROSCIENCE

Volume X, Nos. 1+2 1994

ISSN 0254-8852

Published by Elsevier for the Foundation for the Study of the Nervous System (FESN)

Correspondence should be addressed to: *Discussions in Neuroscience*, FESN, 14 rue Étienne-Dumont, CH-1204 Geneva, Switzerland. Tel: (41) 22-312-2025. Fax: (41) 22-312-2732.

Printed in The Netherlands This Journal is printed on acid-free paper

Modularity, Domain Specificity and the Development of Language

Elizabeth Bates

Debates about the nature and evolution of language often shed more heat than light, because they confuse three logically separable issues: innateness, localization and domain specificity. Proponents of innateness argue that our ability to acquire a language is determined by genetic factors, and mediated by a form of neural organization that is unique to our species. Proponents of localization argue that our ability to process language is localized to specific regions of the brain. Proponents of domain specificity build on both these points, but add the further specification that our localized language abilities are discontinuous from the rest of mind, separate and 'special', constituting what Chomsky (1988) has termed a 'mental organ'.

The first claim has to be true at some level of analysis, because we are indeed the only species that can acquire a language in its full-fledged form (cf. Greenfield and Savage-Rumbaugh, 1991; Savage-Rumbaugh et al., (1993). The second claim is also well attested. Indeed, one of the oldest findings in cognitive neuroscience is the finding that lesions to specific regions of the left cerebral hemisphere in adults usually lead to irreversible forms of language breakdown, or aphasia – although, as we shall see, there is still considerable controversy about the nature of those symptoms (Bates and Wulfeck, 1989a,b). The real debate revolves around the mental-organ claim. Are the mental structures that support language 'modular', discontinuous and dissociable from all other perceptual and cognitive systems? Does the brain of the newborn child contain neural structures that are destined to mediate language, and language alone? The domain specificity view can be contrasted with an approach in which language is viewed as an innate system, but one that involves a reconfiguration of mental and neural systems that exist in other species (Deacon, 1990a,b; Sereno, 1990), and which continue to serve at least some nonlinguistic functions in our own (Bates et al., 1991, 1992).

In this paper, I will provide arguments for innateness and localization but against domain specificity, in research on adult aphasia (the adult

endpoint that is the source of most hypotheses about early specialization for language), and in research on normal and abnormal language development. I will begin with a brief explication of the modular approach to language, and then describe some general arguments and specific findings that support a different view, i.e., that 'Language is a new machine built out of old parts' (Bates et al., 1988).

Modularity and domain specificity: What are they?

The word 'module' is used in markedly different ways by neuroscientists and behavioral scientists, a fact that has led to considerable confusion and misunderstanding in interdisciplinary discussions of brain and language. When a neuroscientist uses the word 'module', s/he is usually trying to underscore the conclusion that brains are structured, with cells, columns, layers and/or regions that divide up the labor of information pro-cessing in a variety of ways. In all fairness, there are few neuroscientists or behavioral scientists who would quibble with this claim. Indeed, Karl Lashley himself probably had something similar in mind, despite his notorious claims about equipotentiality and mass action (Lashley, 1950). In cognitive science and linguistics, the term 'module' refers to a stronger and more controversial claim, one that deserves some clarification before we proceed.

The strongest and clearest definition of modularity in cognitive science comes from Jerry Fodor's influential book *Modularity of mind* (Fodor 1983; see also Fodor, 1985). Fodor begins his book with an acknowledgment to psycholinguist Merrill Garrett, thanking him for the inspiring line 'Parsing is a reflex.' This is, in fact, the central theme in Fodor's book, and the version of modularity that most behavioral scientists have in mind when they use this contentious word. A module is a specialized, encapsulated mental organ that has evolved to handle specific information types of enormous relevance to the species. Following the MIT linguist Noam Chomsky (Chomsky, 1957, 1965, 1988), Fodor argues that human language fits this definition of a module. Elaborating on this argument, Fodor defines modules as cognitive systems (especially perceptual systems) that meet nine specific criteria. Five of these criteria describe the way that modules process information. These include encapsulation (it is impossible to interfere with the inner workings of a module), unconsciousness (it is difficult or impossible to think about or reflect upon the operations of a module), speed (modules are very fast), shallow outputs (modules provide limited output, without information about the intervening steps that led to that output), and obligatory firing (modules operate reflexively, providing pre-determined outputs for pre-determined inputs regardless of the context). As Fodor himself acknowledges (Fodor, 1985), these five characteristics can also be found in acquired skills that have been learned and practiced to the point of automaticity (Schneider and Shiffrin, 1977; Norman and Shallice, 1980). Another three criteria pertain to the biological status of modules, to distinguish these behavioral systems from learned habits. These include ontoge-

neticuniversals (i.e., modules develop in a characteristic sequence), localization (i.e., modules are mediated by dedicated neural systems), and pathological universals (i.e., modules break down in a characteristic fashion following some insult to the system). It is assumed (although this assumption may not be correct – see below) that learned systems do not display these particular regularities. The ninth and most important criterion is domain specificity, i.e., the requirement that modules deal exclusively with a single information type, albeit one of enormous relevance to the species. Aside from language, other examples might include face recognition in humans and other primates, echo location in bats, or fly detection in the frog. Of course learned systems can also be domain-specific (e.g., typing, driving or baseball), but they lack the instinctual base that characterizes a 'true' module. In the same vein, innate systems may exist that operate across domains (see below for examples). However, in Fodor's judgment such domain-general or 'horizontal' modules are of much less interest and may prove intractable to study, compared with domain-specific or 'vertical' modules like language and facè recognition.

Fodor's version of modularity unifies the three claims that language is innate, localized, and domain-specific. This is a thoroughly reasonable proposal, but other forms of mental and neural organization are possible. In fact, all logical combinations of innateness, domain specificity and localization may be found in the minds and brains of higher organisms. Here are a few possible examples.

1) Well-defined regions of the brain may become specialized for a particular function as a result of experience. In other words, learning itself may serve to set up neural systems that are localized and domain-specific, but not innate. A good example comes from positron emission tomography studies of brain activity showing a region of visual cortex that is specialized for words that follow the spelling rules of English (Petersen et al., 1992). Surely we would all agree that English spelling is not part of our biological heritage (and if it is, it should be clear to every teacher that such a module is not well fixed in the genome of American students). The ultimate location of a 'spelling module' must be based on general facts about the organization of visual cortex, and its connections to the auditory system (in particular, the areas with primary responsibility for language – see below).
2) There may be a strong innate predisposition to set up domain-specific functions in a form that is broadly distributed across many different cortical regions, in patterns that vary widely from one individual brain to another. In other words, these systems may be innate and domain-specific, but not strongly localized. An example comes from cortical stimulation showing that many different regions of the left hemisphere can interrupt naming, although some sites are more vulnerable than others (Ojemann, 1991; Burnstine et al., 1990; Lüders et al., 1991, 1986, 1991).
3) There may be systems that are innate and highly localized, but not domain-specific. Instead, they are used to process many different kinds of information. Posner's three different attentional systems might be good candidates for this category (Posner and Driver, 1992).

In short, although evidence for localization is extremely interesting, it is simply not germane to the problems of domain specificity or innateness. Many studies of localization in adult animals (e.g., Goldman-Rakic, 1987) provide compelling evidence for regional specialization of a very intricate sort under 'default' developmental conditions. On the other hand, there has been a veritable explosion of evidence for cortical plasticity in vertebrates, showing how many alternative forms of organization are possible when the default conditions do not hold (e.g., the 'rewiring' results of Frost, Sur, Killackey, O'Leary, Merzenich and others – see Johnson, 1993, for a review). Indeed, some neuroscientists have argued that experience literally sculpts the brain into its final form (Merzenich et al., 1984; Rakic, 1975; Huttenlocher, 1990). Hence localization and domain specificity may be the endpoints of learning and development, but they are not necessarily the starting points (Karmiloff-Smith, 1993).

My arguments here will focus on domain specificity, but first I should clarify that domain specificity itself can apply at several different levels. A system may have unique properties at one level, while it follows general laws at another. Table 7 lists five levels at which a claim of domain specificity can be made: (1) the task or problem to be solved, (2) the behaviors or skills that evolve (or emerge) to solve the problem, (3) the knowledge or representations that must be present somewhere in the mind/brain of an individual who can solve the problem and produce the requisite behaviors, (4) the neural mechanisms or processors that are required to sustain those representations, and (5) the genetic substrate that makes 1 – 4 possible (in interaction with some environment). What level do we have in mind when we argue that language is 'special'? Surely we can agree that language represents a special response to a special problem, i.e., the problem of mapping thoughts and concepts that are inherently non-linear (or atemporal) onto a channel with heavy linear (temporal) constraints. That is, symbols must be produced one at a time (one word or one sign), fast enough to fall within memory constraints but clearly and efficiently enough for successful production and comprehension. Human languages represent a very broad set of possible solutions to this special problem, but taken together (for all their similarities and differences), languages do not really look very much like anything else that we do (i.e., Turkish and tennis both take place in real time, but they do not look alike). Finally, we can all agree that the detailed and unique set of behaviors that comprise language must be supported by a detailed and unique set of mental/neural representations, i.e., knowledge of Turkish cannot look very much like knowledge of tennis.

In other words, there is no controversy surrounding the claim that language is 'special' at the first three levels in Table 7. The problem of language is unique, it is solved in a special way, and the knowledge required to solve that problem does not look like anything else we know. The real controversy revolves around the next two levels in the chart. To solve a spe-

TABLE 7

Proposed levels of analysis for the domain specificity, localization and innateness of language

	Domain specificity (unique to language)	Localization (restricted to specific sites)
Tasks/Problems to be solved	Yes	No
Behaviors /Solutions	Yes	No
Representations/Knowledge	Yes	No
Processing mechanisms	No	Yes
Genetic substrate	No	Yes

cial problem, do we really have to have a special information processor? Have we evolved new neural tissue, a new region or a special form of computation that deals with language, and language alone? And is that new mechanism guaranteed by its own special stretch of DNA? These are the levels at which I part company with the Fodor/Chomsky view. In the words of Eric Kandel

> The functions localized to discrete regions in the brain are not complex faculties of mind, but elementary operations. More elaborate faculties are constructed from the serial and parallel (distributed) interconnections of several brain regions.
>
> (Kandel et al., 1991, p.15)

Our challenge is to figure out how these older, simpler neural systems have been reconfigured to solve the language problem. I will argue that language is domain-specific at Levels 1 – 3 (the problem, its behavioral solution, the representations that support behavior), but these levels are not innate or localized. On the other hand, linguistic knowledge is acquired and supported by processors that are innate and are localized, but not domain-specific (that is, they can also process information from other domains).

General arguments against the domain specificity of language

My own long-standing skepticism about the mental-organ claim is based on four kinds of evidence: (1) phylogenetic recency, (2) behavioral plasticity, (3) neural plasticity, (4) the arbitrariness of mappings from form to meaning. None of these arguments constitute a disproof of domain specificity, but together they weaken its plausibility.

Phylogenetic recency

Bates et al. (1991) note that the species on this earth have had a great deal of time to evolve ways of dealing with light, gravity, motion, spatial organization, cause and effect, and the boundaries of common objects and events. By contrast, language is a newcomer – about 30,000 years old by current best estimates. It is hard to imagine how we could have developed elaborate, innate and domain-specific mechanisms for language in a relatively short period of time (although 'poverty of the imagination' is an admittedly weak argument for any case, including my own).

Behavioral plasticity

Although one sometimes reads in textbooks that languages are based on a host of universal principles, the same everywhere, cross-linguistic research testifies to a surprising variability in structure and function across natural languages (MacWhinney and Bates, 1989, a volume based on studies of sentence processing in 15 different languages, as drastically different as Hungarian, Warlpiri and Chinese; see also Wurm, this volume). To be sure, there are some similarities (e.g., all languages have a semantics and a grammar). But the variability that has been recorded so far greatly exceeds reports for other putatively innate and domain-specific systems (including the oft-invoked example of birdsong). Oral languages present a daunting range of possibilities, from Chinese (a language with absolutely no inflections of any kind on nouns or verbs) to Greenlandic Eskimo (a language in which a sentence can consist of a single word with 8 – 12 prefixes, suffixes, and infixes). But an even more important lesson comes from the fact that deaf communities have developed full-blown linguistic systems in the visual-manual modality (e.g., Klima and Bellugi, 1988). If bats were suddenly deprived of echo location, would they develop an equally complex and efficient system in some other modality, within two generations? Probably not. To me, the very existence of languages like ASL argues strongly against domain specificity – although it does argue that our species has a robust and passionate urge of some kind to communicate our most complex thoughts, and a powerful set of information processing mechanisms that permit us to solve this problem.

Neural plasticity

In contrast with the best-known examples of innate and domain-specific brain systems, the systems that support languages also show an extraordinary and perhaps unprecedented degree of neural plasticity. Research on the long-term effects of early focal brain injury suggests that children with large lesions to the classic language zones go on, more often than not, to attain levels of language ability that are indistinguishable from normal (Bates et al., 1992; Thal et al., 1991; Marchman et al., 1991; Stiles and Thal, 1993; Vargha-Khadem et al., 1991; Aram, 1988). As Milner and her colleagues have shown (Rasmussen and Milner, 1977; Milner, this volume), this steady state can be achieved in a variety of ways. In roughly 40% of the adult survivors of early focal brain injury who received a sodium amytal test to determine the hemispheric specialization for speech, language production was

interrupted by paralysis of the right hemisphere. Another 40% of this sample displayed left-hemisphere dominance for speech, suggesting that some kind of reorganization has taken place within the left hemisphere. The remaining 20% displayed some form of bilateral organization for speech, with some language functions controlled by the left and others by the right.

This does not mean that the two hemispheres are initially equipotential for language. For the last ten years, we have carried out prospective studies of language development in children with focal injuries to the left or right hemisphere. That is, we locate children with early focal brain injury in the prelinguistic period (before six months of age), and follow them through their first encounters with cognitive domains that are lateralized in normal adults (e.g., language, spatial cognition, facial affect). Our findings for language are largely compatible with retrospective studies of the same populations, i.e., most children go on to achieve linguistic abilities within the normal or low-normal range. However, it is also clear that this reorganization takes place after an initial phase where regional biases for language are evident (whether or not those biases map onto the adult picture). Regardless of side, size or site of lesion, most children with focal brain injury are delayed in the first stages of language production. Receptive delays are not uniquely associated with left-hemisphere injury at any point in the stages that we have studied so far, suggesting that the acquisition of receptive control over language may be a bilateral phenomenon (indeed, receptive deficits tend to be slightly greater with right-hemisphere injury) On the other hand, recovery from initial delays in expressive language does take longer (on average) in children with left-hemisphere injury. We may conclude with some confidence that the recovery of language observed in children with focal brain injury represents a true reorganization, an alternative to the default model that is discovered after an initial delay.

The same degree of plasticity is not observed in other, phylogenetically older cognitive domains (Stiles and Thal, 1993). Working with the same population of children, Stiles and colleagues (Stiles-Davis, 1988; Stiles-Davis et al., 1988; Stiles and Nass, 1991) have observed patterns of behavioral deficit along the lines that we would expect from work on spatial cognitive deficits in adults (although the childhood variants are more subtle). Reilly and colleagues have reported similar parallels to the adult model in their research on facial affect in these children (Reilly et al., submitted). Although it is difficult to compare apples and oranges, it looks as though there may be more plasticity for language than we observe in other perceptual and cognitive systems.

Arbitrariness of form-meaning mapping

This final point is a bit more difficult to summarize, but I think it is at least as important as the first three. A defining characteristic of language (indeed, one of its few universals) is the arbitrariness of the relationship between sound and meaning (and, to a surprising degree, between signs and their meanings in ASL). The words 'dog', 'chien', 'perro', 'cane', 'Hund', etc. do not in any way resemble the fuzzy four-legged creatures that they signify. The same is true for the relationship between grammatical forms and the communicative work that those forms carry out. For example, depending on the language that one speaks, basic information about 'who did what to whom' can be signalled through word order (as it is in English), case inflections on nouns (e.g., Latin, Russian, Hungarian), agreement marking between subject and verb (a major source of information in Italian, but only a minor source in English), and a range of other cues. What does this have to do with modularity? If one examines all the known examples of innate and domain-specific knowledge, there is always some kind of a physical constant, a partial isomorphism between the source of information in the world to which the animal must respond, and the internal state that the animal must take (at some level in the nervous system) in order to respond correctly. Consider, for example, the 'bug detector' in the retina of the frog (Lettvin et al., 1959), or the line angle detectors located in the visual cortex of kittens (Hubel and Wiesel, 1963). To evolve an innate perceptual and/or motor system, it seems that nature needs something to work with, something that holds still, something physically solid, constant, reliable. Language lacks this property, and for that reason, I find it hard to understand in concrete, material terms what an 'innate language-specific acquisition device' might look like.

As I have said, none of these are knock-down arguments by themselves. They simply serve to put us on our guard, to raise an appropriate level of skepticism in the face of claims about a grammar gene or a language neuron. Let us turn now to some more specific claims about innateness, localization and domain specificity, starting with the adult aphasia (the first test case for localization and domain specificity in the history of cognitive neuroscience).

Arguments based on adult aphasia

Let us assume, for the moment, that there is good evidence for localization of language in our species, in high-probability default patterns that must (I agree) mean that some kind of genetic bias is at work. Exactly what is localized?

In the early stages of research on aphasia, it was generally argued that Broca's aphasia (non-fluent with spared comprehension) results from a breakdown in the motor aspects of language, while Wernicke's aphasia (comprehension deficits in the presence of fluent speech) results from injury to sensory areas. This characterization made reasonably good neuroanatomical sense, in view of the fact that Broca's aphasia correlates with frontal injury while Wernicke's aphasia is associated with posterior lesions, but its fit to the behavioral data was always fairly loose. As Freud (1891/1953) pointed out a hundred years ago, a sensory deficit cannot explain the severe word-finding deficits and substitution errors that characterize the fluent output observed in Wernicke's aphasia. In the 1970s, analogous problems arose for the motor account of Broca's aphasia (Zurif and Caramazza, 1976; Heilman and Scholes, 1976). In particular, careful

experimental studies showed that these patients also suffer from comprehension problems when they are forced to rely on grammatical markers to interpret complex sentences (e.g., patients could interpret 'The apple was eaten by the boy', but not 'The boy was chased by the girl'). At this point, several investigators offered an alternative view based on modular theories of linguistic organization (e.g., Caramazza and Berndt, 1985). In particular, it was argued that Broca's aphasics have lost the ability to comprehend or produce grammar (resulting in telegraphic output, and subtle comprehension deficits that are most evident when semantic information is too ambiguous to support sentence interpretation). Conversely, the comprehension deficits and word-finding problems observed in Wernicke's aphasia could be jointly and parsimoniously explained if these patients have lost the ability to process content words. This apparent double dissociation provided support for the idea that the brain is organized into innate, domain-specific and localized modules for grammar and semantics, respectively (see Gazzaniga, this volume, for arguments along the same lines).

But this unifying view has also fallen on hard times. More recent studies of language breakdown in aphasia have forced investigators to abandon the idea of a 'grammar box', i.e., neural tissue that is devoted exclusively to grammar, and contains the representations that are necessary for grammatical processing. To offer just a few examples, there are (1) numerous studies showing that so-called agrammatic aphasics can make remarkably fine-grained judgments of grammaticality (Linebarger et al., 1983; Wulfeck, 1987; Shankweiler et al., 1989; Wulfeck and Bates, 1991), and (2) a host of cross-linguistic studies showing differences in the symptoms displayed by agrammatic patients in different language communities – differences that can only be explained if we acknowledge that the patient still retains detailed knowledge of his/her grammar (Bates et al., 1991; Menn and Obler, 1990). It begins to look as though linguistic knowledge is broadly represented in the adult brain – a conclusion that is also supported by studies of brain activity during normal language use (Petersen et al., 1992; Kutas and Kluender, 1991). Some areas do play a more important role than others in getting a particular process underway in real time, but the knowledge itself is not strictly localized.

So what is localized? The classic sensorimotor view of Broca's and Wernicke's aphasia has fallen by the wayside, and now the grammar/semantics view has fallen as well. But their successor is still unnamed. Some investigators have argued that left frontal regions are specialized for the rapid processes required for fluent use of grammar, while posterior regions play a more important role in controlled, strategic choice of words and sentence frames (e.g., Frazier and Friederici, 1991; Zurif et al., 1990; Milberg and Albert, 1991). These ideas are still distressingly vague, but they point us in a new direction.

From a developmental perspective, the default pattern of brain organization for language observed in adults can be viewed as the end product of regional differences in neural computation and processing that 'attract' or 'recruit' language processes under default conditions. The perisylvian areas of the left hemisphere are not 'innate language tissue', any more than a tall child constitutes an 'innate basketball player'. However, all other things being equal, the left perisylvian areas will take over the language problem, and the tall child has a very good chance of ending up on the basketball team. This brings me to the problem of how (and where) language is acquired.

Arguments based on normal and abnormal language development

In line with Fodor's criterion for ontogenetic universals, it is well known that children go through a series of universal stages in language learning: from babbling in vowel sounds (around 3 months) to babbling in consonants (between 6 – 9 months); from first signs of word comprehension (from 8 – 10 months) to the onset of word production (averaging 12 months, with a substantial range of individual variability); from the single-word stage (from 12 – 20 months, on average) to the onset of word combinations; from simple two-word strings (so-called telegraphic speech) to complex grammar (evident in most normal children by three years of age). But can we conclude that these milestones reflect the unfolding of a domain-specific module? Probably not, at least not on the basis of the evidence that is currently available (see Bates et al., 1992, for details). First of all, there is enormous variability from one child to another in the onset and duration of these stages. Second, there are important variations in this basic pattern from one language to another (e.g., children who are exposed to a richly inflected language like Turkish often display signs of productive grammar in the one-word stage). Third, each of these milestones in early language is correlated with specific changes outside the boundaries of language (e.g., the use of familiar gestures like drinking, combing or putting a telephone receiver to the ear as a way of 'labelling' common objects – gestures that appear in the hearing child right around the time that naming takes off in the vocal modality). In other words, one cannot conclude that the universal maturational timetable for language is really universal, or that it is specific to language.

These problems of interpretation are compounded in research on abnormal language development. Two recent examples illustrate the confusion between innateness and domain specificity that has plagued this field, much like the confusion between domain specificity and localization that has characterized research on adult aphasia.

Petitto and Marentette (1991) published an influential paper demonstrating that deaf infants exposed to sign language 'babble' with their hands, producing meaningless but systematic actions that are not observed in hearing children. Furthermore, this form of manual babbling occurs around 8 – 10 months of age, the point at which vocal babbling appears in the hearing child. The authors conclude that language learning involves innate abilities that are independent of modality (i.e., vocal or manual); they also claim that these abilities are specific to language, pro-

viding support for Chomsky's mental-organ claim. Their first conclusion is clearly supported by the evidence, but the second is not. We have known for more than 100 years that children begin to imitate novel actions (i.e., actions that are not already in their repertoire) around 8 – 10 months. The more systematic the adult input, the more systematic the child's imitation is likely to be. Petitto and Marentette's demonstration of babbling in the visual modality constitutes a particularly beautiful example of this interesting but well-established fact. The kind of imitation that underlies babbling is undoubtedly based upon abilities that are innate, and particularly well developed in our species (human children imitate far better and more often than any other primate (Greenfield and Savage-Rumbaugh, 1991; Chevalier-Skolnikoff, 1991), but proof of its existence does not, in itself, constitute evidence in favor of the notion that language is 'special'.

A somewhat different example appeared in a letter to *Nature* by Gopnik (1990; for further details see Gopnik and Crago, 1991), describing preliminary results from a study of grammatical abilities in a family of individuals suffering from some kind of genetically based disorder (see also Tallal et al., 1991). Members of this family have difficulty with particular aspects of grammar, including regular verb inflections (e.g., the '-ed' in verbs like 'walked' and 'kissed'). By contrast, they are reported to have less trouble with irregular forms like 'came' or 'gave'. This pattern is offered as an example of an innate and domain-specific disorder, termed 'feature-blind dysphasia', and has been cited as evidence in favor of Pinker's claim that regular and irregular forms are handled by separate mental and perhaps neural mechanisms (Pinker, 1991). Shortly after Gopnik's letter appeared, *Nature* published a rebuttal by Vargha-Khadem and Passingham (1990; see also Fletcher, 1990), who have studied the same family for a number of years. These authors point out that the members of this family suffer from a much broader range of linguistic and non-linguistic deficits than one might conclude from Gopnik's description. Their peculiar grammatical symptoms are only the tip of an iceberg, one by-product of a disorder with repercussions in many different areas of language and cognition, providing further evidence for innateness but none for domain specificity (Marchman, 1993).

The above examples are part of a long tradition in neurolinguistics, where unusual profiles of language ability and disability are cited as events for the eccentricity and modularity of language. Some other 'parade cases' include Specific Language Impairment or SLI, and children with Williams Syndrome.

By definition, specific language impairment (SLI) refers to delays in receptive and/or expressive language development in children with no other known form of neurological or cognitive impairment. However, recent studies of SLI suggest that this definition may not be accurate (Cohen, 1991; Tallal et al., 1985). Although these children do not suffer from global forms of mental retardation, they do show subtle impairments in aspects of cognition and/or perception that are not specific to language. For example, many children with SLI experience difficulty in

processing rapid transitions in acoustic information (including non-linguistic stimuli). This may help to explain new studies comparing SLI in English, Italian and Hebrew (Rom and Leonard, 1990; Leonard et al., in press) showing that the specific areas of grammar that are most delayed vary from one language to another, and the most vulnerable elements within each language appear to be those that are low in 'phonological substance' (i.e., salience). The subtle deficits associated with SLI may also transcend the acoustic modality, affecting certain kinds of manual gesture (Thal et al., 1991). Taken together, these studies suggest that SLI may not be a purely linguistic (or acoustic) phenomenon.

The strongest evidence to date in favor of domain specificity comes from rare cases in which language appears to be remarkably spared despite severe limitations in other cognitive domains. Etiologies associated with this unusual profile include spina bifida and hydrocephalus, and a rare form of mental retardation called Williams Syndrome, or WMS (Bellugi et al., 1991; Jernigan and Bellugi, 1990). The dissociations observed in WMS prove that language can 'decouple' from mental age at some point in development. Nevertheless, recent studies of WMS place constraints on the conclusion that language is a separate mental system from the beginning. First, it is clear that language development is seriously delayed in infants and preschool children with WMS, suggesting that certain 'cognitive infrastructures' must be in place before language can be acquired (Thal et al., 1989). Second, studies of older children with WMS demonstrate peculiar islands of sparing in some non-linguistic domains (e.g., face recognition, and recognition of common objects from an unfamiliar perspective), and unusual patterns of deficit in other non-linguistic domains that are not at all comparable to the patterns displayed by Down Syndrome children matched for mental age. Third, the language of older children and adults with WMS includes some deviant characteristics that are not observed in normal children. For example, in a word fluency test in which WMS children and Down Syndrome controls were asked to generate names for animals, Down Syndrome and normal controls tend to generate high-frequency words like 'dog' and 'cat'; WMS individuals tend instead to generate unusual, low-frequency items like 'ibex' and 'brontosaurus'. In view of such findings, it seems that WMS may not represent sparing of normal language, but a completely different solution to the language problem, achieved with a deviant form of information processing.

In short, the dissociations between language and cognition observed in SLI (where language < cognition) and in Williams Syndrome (where language > cognition) cannot be used to support a mental-organ view. Things are just not that simple. Instead, these unusual profiles offer further evidence for the behavioral and neural plasticity of language. There are many ways to solve the problem of language learning. Some are more efficient than others, to be sure, but the problem can be solved with several different configurations of learning, memory, perception and cognition. This brings us to my final point: How is it that language is learnable at all?

There is a branch of language acquisition research called 'learnability theory' (e.g., Lightfoot, 1991), which uses formal analysis to determine the range of conditions under which different kinds of grammars can (in principle) be learned. Until recently, most of this research has been based upon the assumption that language learning in humans is similar to language learning in serial digital computers, where *a priori* hypotheses about grammatical rules are tested against strings of input symbols, based on some combination of positive evidence ('here is a sentence in the target language') and negative evidence ('here is a sentence that is not permitted by the target language'). A famous proof by Gold (1967) showed that a broad class of grammars (including generative grammars of the sort described by Chomsky) could not be learned by a system of this kind unless negative evidence was available in abundance, or strong innate constraints were placed upon the kinds of hypotheses that the system would consider. Since we know that human children are rarely given explicit negative evidence, the learnability theory seems to require the conclusion that children have an extensive store of innate and domain-specific grammatical knowledge.

In the last two years, this conclusion has been challenged by major breakthroughs in the application of a different kind of computer architecture (called neural networks, connectionism, and/or parallel distributed processing) to classic problems in language learnability. Because connectionism makes a very different set of assumptions about the way that knowledge is represented and acquired, Gold's pessimistic conclusions about language learnability do not necessarily apply. This new era began in 1986 with a simulation by Rumelhart and McClelland (1986) on the acquisition of the English past tense, showing that connectionist networks go through stages that are very similar to the ones displayed by children who are acquiring English (producing and then recovering from rule-like overgeneralizations like 'comed' and 'wented', in the absence of negative evidence). This simulation has been severely criticized (see especially Pinker and Prince, 1988; Kim et al., 1991). However, a number of new works have appeared that get around these criticisms, replicating and extending the Rumelhart-McClelland findings in several new directions (Elman, 1990, 1991; MacWhinney, 1991; Plunkett and Marchman, 1991; Marchman, 1993). The most recent example comes from Marchman (1993), who has 'lesioned' neural networks at various points during learning of the past tense (randomly eliminating between 2 and 44% of the connections in the network). These simulations capture some classic 'critical period' effects in language learning (e.g., smaller, earlier lesions lead to better outcomes; later, larger lesions lead to persistent problems in grammar), showing that such effects can occur in the absence of 'special' maturational constraints (compare with Newport, 1990, and Elman, 1991). In addition, Marchman's damaged systems found it more difficult to acquire regular verbs (e.g., 'walked') than irregulars (e.g., 'came'), proving that the specific pattern of deficits described by Gopnik and by Pinker can result from non-specific forms of brain damage in a general-purpose learning device. Such research on language learning in neural networks is still in its infancy, and we do not know how far it can go. But it promises to be an important tool, helping us to determine just how much innate knowledge has to be in place for certain kinds of learning to occur.

In short, a great deal has been learned in the last few years about the biological foundations for language development. Evidence for innateness is good, but evidence for a domain-specific 'mental organ' is difficult to find. Instead, language learning appears to be based on a relatively plastic mix of neural systems that also serve other functions. I believe that this conclusion renders the mysteries of language evolution at issue in this volume somewhat more tractable. That is, the continuities that we have observed between language and other cognitive systems make it easier to see how this capacity came about in the first place.

Acknowledgements

This research was supported by the Center for the Study of the Neurological Basis of Language (NIH-NINDS NS 22343), the Crosslinguistic Studies in Aphasia Project (NIH-NIDCD DC00216) and the Center for the Study of the Neurological Basis of Disorders of Language, Learning and Behavior (NINCDS 1 P50 NS22343), with partial support form the Grammatical Abilities in Altzheimer's Disease Project (NIH No. 5 RO1 DC00348). Selected passages in this essay have appeared in E. Bates, 'Language Development', in E. Kandel and L. Squire (Eds.), Special Issue on Cognitive Neuroscience, Curr. Opinion Neurobiol. 2, 180–185.

References

Aram, D.M. (1988). Language sequelae of unilateral brain lesions in children. In F. Plum, (Ed.), *Language, communication and the brain.* New York: Raven Press.

Bates, E., Bretherton, I., & Snyder, L. (1988). *From first words to grammar: Individual differences and dissociable mechanisms.* New York: Cambridge University Press.

Bates, E., Thal, D., & Janowsky, J. (1992). Early language development and its neural correlates. In I. Rapin & S. Segalowitz (Eds.), *Handbook of neuropsychology, Vol. 7: Child neuropsychology.* Amsterdam: Elsevier.

Bates, E., Thal, D. and Marchman, V. (1991). Symbols and syntax: A Darwinian approach to language development. In N. Krasnegor, D. Rumbaugh, E. Schiefelbusch and M. Studdert-Kennedy (Eds.), *Biological and behavioral determinants of language development.* Hillsdale, NJ: Erlbaum, 29 - 65.

Bates, E. & Wulfeck, B. (1989a). Crosslinguistic studies of aphasia. In B. MacWhinney & E. Bates (Eds.), *The crosslinguistic study of sentence processing.* New York: Cambridge University Press.

Bates, E. & Wulfeck, B. (1989b). Comparative aphasiology: A cross-linguistic approach to language breakdown. *Aphasiology*, **3**, 111-142 and 161-168.

Bates, E., Wulfeck, B. & MacWhinney, B. (1991). Crosslinguistic research in aphasia: An overview. *Brain and Language,* **41**, 123-148.

Burnstine, T.H., Lesser, R.P., Hart, J. Jr., Uematsu, S., Zinreich,S.J., Krauss, G.L., Fisher, R.S., Vining, E.P., and Gordon, B. (1990). Characterization of the basal temporal language area in patients with left temporal lobe epilepsy. *Neurology*, **40(6)**, 966-970.

Bellugi, U., Bihrle, A., Neville, H., Jernigan, T. and Doherty, S. (1991). Language, cognition and brain organization in a neurodevelopmental disorder. In W. Gunnar and C. Nelson (Eds.), *Developmental Behavioral Neuroscience.* Hillsdale, NJ: Erlbaum.

Caramazza, A. & Berndt, R. (1985). A multicomponent view of agrammatic Broca's aphasia. In M.-L. Kean (Ed.), *Agrammatism* (pp. 27-63). New York: Academic Press.

Chevalier-Skolnikoff, S. (1991) Spontaneous tool use and sensorimotor intelligence in Cebus compared with other monkeys and apes. *Behavioral and Brain Sciences,* **14:2**.

Chomsky, N. (1957). *Syntactic structures.* The Hague: Mouton.

Chomsky, N. (1965). *Aspects of the theory of syntax.* MIT Press.

Chomsky, N. (1988) *Language and problems of knowledge.* MIT Press.

Cohen, H., Gelinas, C., Lassonde, M. and Geoffrey, G. (1991). Auditory lateralization for speech in LI children. *Brain and Language,* **41,** 395 - 401.

Deacon, T. (1990a). Brain-language coevolution. In J.A. Hawkins and M. Gell-Mann (Eds.), *The evolution of human languages: Proceedings of the Santa Fe Institute Studies in the Sciences of Complexity.* Addison-Wesley.

Deacon, T. (1990b). Rethinking mammalian brain evolution. *American Zoologist,* **30,** 629 - 705.

Elman, J. (1990). Finding structure in time. *Cognitive Science,* **14,** 179 - 211.

Elman, J. (1991). Incremental learning, or the importance of starting small. *Proceedings of the Thirteenth Annual Conference of the Cognitive Science Society.* Hillsdale, NJ: Erlbaum, 443 - 448.

Fletcher, P. (1990). Speech and language defects. *Nature,* **346,** 226.

Frazier, L. & Friederici, A. (1991). On deriving the properties of agrammatic comprehension. *Brain and Language,* **40**, 51-66.

Fodor, J. (1983). *The modularity of mind.* Cambridge, Mass.: MIT Press.

Fodor, J.A. (1985). Multiple book review of 'The modularity of mind'. *Behavioral and Brain Sciences,* **8,** 1-42.

Freud, A. (1953). *On aphasia: A critical study.* New York: International Universities Press. (Original work published in 1891).

Gazzaniga, M. (1993, April). *Language and the cerebral hemispheres.* Paper presented at the FESN Study Group on Evolution and Neurology of Language, Geneva.

Gold, E. (1967). Language identification in the limit. *Information and Control,* **16,** 447 - 474.

Goldman-Rakic, P.S. (1987). Development of cortical circuitry and cognitive function. *Child Development,* **58**, 601-622.

Gopnik, M. (1990). Feature-blind grammar and dysphasia. *Nature,* **344**, 715.

Gopnik, M. and Crago, M. (1991). Familial aggregation of a developmental language disorder. *Cognition,* **39:1,** 1 - 50.

Greenfield, P. and Savage-Rumbaugh, E. (1991). Imitation, grammatical development and the invention of protogrammar by an ape. In N. Krasnegor, D. Rumbaugh, R. Schiefelbusch and M. Studdert-Kennedy (Eds.), *Biological and behavioral determinants of language development.* Hillsdale, NJ: Erlbaum, 235 - 262.

Heilman, K.M. & Scholes, R.J. (1976). The nature of comprehension errors in Broca's, conduction and Wernicke's aphasics. *Cortex*, **12**, 258-265.

Hubel, D.H., & Wiesel, T.N. (1963). Receptive fields of cells in striate cortex of very young, visually

inexperienced kittens. *Journal of Neurophysiology*, **26**, 944-1002.

Huttenlocher, P. R. (1990). Morphometric study of human cerebral cortex development. *Neuropsychologia* **28:6**, 517 - 527.

Jernigan, T. & Bellugi, U. (1990). Anomalous brain morphology on magnetic resonance images in Williams Syndrome and Down Syndrome. *Archives of Neurology*, **47**, 429-533.

Johnson, M. (Ed.). (1993). *Brain development and cognition: A reader.* Oxford: Blackwell Publishers.

Kandel, E.R., Schwartz, J.H., & Jessell, T.H. (1991). *Principles of neural science* (3rd ed.). New York: Elsevier.

Karmiloff-Smith, A. (1993). *Beyond modularity: A developmental perspective on cognitive science.* Cambridge, MA: MIT Press.

Kim, J., Pinker, S., Prince, A. and Sandup, P. (1991). Why no mere mortal has ever flown out to center field. *Cognitive Science,* **15:2,** 173 - 218.

Klima, E. & Bellugi, U. (1988). *The signs of language.* Harvard University Press.

Kutas, M. & Kluender, R. (1991). What is who violating? A reconsideration of linguistic violations in light of event-related potentials. *Center for Research in Language Newsletter, 6:1.* La Jolla: University of California, San Diego, Center for Research in Language.

Lashley, K.S. (1950). In search of the engram. In *Symposia of the Society for Experimental Biology, No. 4. Physiological mechanisms and animal behaviour.* New York: Academic Press,

Leonard, L., Bortolini, U., Caselli, M., McGregor, K. and Sabbadini, L. (in press). Two accounts of morphological deficits in children with Specific Language Impairment. *Language Acquisition.*

Lettvin, J.Y., Maturana, H.R., McCulloch, W.S., & Pitts, W.H. (1959). What the frog's eye tells the frog's brain. *Proceedings of the Institute of Radio Engineering of New York*, **47**, 1940-1951.

Lightfoot, D. (1991). The child's trigger experience — Degree-0 learnability. *Behavioral Brain Sciences,* **14:2,** 364.

Linebarger, M., Schwartz, M., & Saffran, E. (1983). Sensitivity to grammatical structure in so-called agrammatic aphasics. *Cognition*, **13**, 361-392.

Lüders, H., Lesser, R., Dinner, D., Morris, H., Wyllie, E., and Godoy, J. (1991). Localization of cortical function: New information from extraoperative monitoring of patients with epilepsy. *Epilepsia*, **29** (Suppl. 2), S56-S65.

Lüders, H., Lesser, R., Hahn, J., Dinner, D., Morris, H., Resor, S., and Harrison, M. (1986). Basal temporal language area demonstrated by electrical stimulation. *Neurology*, **36**, 505-509.

Lüders, H., Lesser, R., Hahn, J., Dinner, D., Morris, H., Wyllie, E., and Godoy, J. (1991). Basal temporal language area. *Brain*, **114**, 743-754.

MacWhinney, B. (1991). Implementations are not conceptualizations: Revising the verb-learning model. *Cognition,* **40,** 121 - 157.

MacWhinney, B. & Bates, E. (Eds.) (1989). *The crosslinguistic study of sentence processing.* New York: Cambridge University Press.

Marchman, V. (1993). Constraints on plasticity in a connectionist model of the English past tense. *Journal of Cognitive Neuroscience* , **5:2**, 215-234..

Marchman, V., Miller, R. and Bates, E. (1991). Babble and first words in children with focal brain injury. *Applied Psycholinguistics,* **12,** 1-22.

Menn, L. & Obler, L.K. (Eds.). (1990). *Agrammatic aphasia: Cross-language narrative sourcebook.* Amsterdam/Philadelphia: John Benjamins.

Merzenich, M., Nelson, R., Stryker, M., Cynader, M., Schoppmann, A. & Zook, J. (1984). Somatosensory cortical map changes following digit amputation in adult monkeys. *Journal of Comparative Neurology*, **224**, 591-605.

Milberg, W. & Albert, M. (1991). The speed of constituent mental operations and its relationship to neuronal representation: An hypothesis. In R.G. Lister & H.J. Weingartner (Eds.), *Perspectives on cognitive neuroscience.* New York: Oxford University Press.

Milner, B. (1993, April). *Carotid-amytal studies of speech lateralization and gesture control.* Paper presented at the FESN Study Group on Evolution and Neurology of Language, Geneva.

Newport, E. (1990). Maturational constraints on language learning. *Cognitive Science,* **14,** 11 - 28.

Norman, D.A., & Shallice, T. (1980). *Attention to action: Willed and automatic control of behavior.* Center for Human Information Processing (Technical Report No. 99). (Reprinted in revised form in R.J. Davidson, G.E. Schwartz & D. Shapiro [Eds.] [1986], *Consciousness and self-regulation* [Vol. 4]. New York: Plenum Press.)

Ojemann, G.A. (1991). Cortical organization of language. *Journal of Neuroscience*, **11:8**, 2281-2287.

Petersen, S.E., Fiez, J.A. & Corbetta, M. (1992). Neuroimaging. *Current Opinion in Neurobiology - Special Issue on Cognitive Neuroscience*, **2**, 217-222.

Petitto, L. and Marentette, P.F. (1991). Babbling in the manual mode: Evidence for the ontogeny of language. *Science,* **251**, 1493-1499.

Pinker, S. (1991). Rules of language. *Science. 253*, 530 - 535.

Pinker, S. and Prince, A. (1988). On language and connectionism: An analysis of a parallel distributed processing model of language acquisition. *Cognition,* **28,** 73 - 193.

Plunkett, K. and Marchman, V. (1991). U-shaped learning and frequency effects in a multi-layered perceptron: Implications for child language acquisition. *Cognition,* **38:1,** 43 - 102.

Plunkett, K., & Marchman, V. (1993). From rote learning to system building: Acquiring verb morphology in children and connectionist nets. *Cognition,* **48,** 21-69.

Posner, M.I. & Driver, J. (1992). The neurobiology of selective attention. *Current Opinion in Neurobiology - Special Issue on Cognitive Neuroscience,* **2,** 165-169.

Rakic, P. (1975). Timing of major ontogenetic events in the visual cortex of the rhesus monkey. In N. Buchwald & M. Brazier (Eds.), *Brain mechanisms in mental retardation.* New York: Academic Press.

Rasmussen, T., & Milner, B. (1977). The role of early left-brain injury in determining lateralization of cerebral speech functions. *Annals of the New York Academy of Sciences,* **299,** 355-369.

Reilly, J., Stiles, J., Larsen, J., & Trauner, D. (1994). *Affective facial expression in infants with focal brain damage.* Manuscript submitted for publica-tion.

Rom, A. and Leonard, L. (1990). Interpreting deficits in grammatical morphology in specifically language-impaired children: Preliminary evidence from Hebrew. *Clinical Linguistics and Phonetics,* **4:2,** 93 - 105.

Rumelhart, D., McClelland, J. and the PDP Research Group (1986). *Parallel distributed processing: Explorations in the microstructure of cognition, Vol. 1.* Cambridge, MA.: MIT/Bradford Books.

Savage-Rumbaugh, S., Murphy, J., Sevcik, R., Brakke, K., Williams, S., & Rumbaugh, D. (1993). Language comprehension in ape and child. *Monographs of the Society for Research in Child Development,* Serial #233, Volume **58**, 3-4, 222-242.

Schneider, W., & Shiffrin, R. (1977). Controlled and automatic human information processing: 1. Detection, search and attention. *Psychological Review,* **84**, 321-330.

Sereno, M. (1990). Language and the primate brain. *Center for Research in Language Newsletter, 4:4.* L a Jolla: University of California, San Diego, Center for Research in Language.

Shankweiler, D., Crain, S., Gorrell, P., & Tuller, B. (1989). Reception of language in Broca's aphasia. *Language and Cognitive Processes,* **4:1**, 1 - 33.

Stiles-Davis, J. (1988). Spatial dysfunctions in young children with right cerebral hemisphere injury. In J. Stiles-Davis, M. Kritchevsky & U. Bellugi (Eds.), *Spatial cognition: Brain bases and development.* Hillsdale, NJ: Erlbaum.

Stiles-Davis, J., Janowsky, J., Engel, M., & Nass, R. (1988). Drawing ability in four young children with congenital unilateral brain lesions. *Neuropsychologia,* **26,** 359-371.

Stiles, J. & Nass, R. (1991). Spatial grouping activity in young children with congenital right- or left-hemisphere brain injury. *Brain & Cognition,* **15**, 201-222.

Stiles, J. & Thal, D. (1993). Linguistic and spatial cognitive development following early focal brain injury: Patterns of deficit and recovery. In M. Johnson (Ed.), *Brain development and cognition: A reader.* Oxford: Blackwell Publishers.

Tallal, P., Stark, R. and Mellits, D. (1985). Identification of language-impaired children on the basis of rapid perception and production skills. *Brain and Language,* **25,** 314 - 322.

Tallal, P., Townsend, J., Curtiss, S. and Wulfeck, B. (1991). Phenotypic profiles of language-impaired children based on genetic/family history. *Brain and Language,* **41,** 81 - 95.

Thal, D., Bates, E., & Bellugi, U. (1989). Language and cognition in two children with Williams Syndrome. *Journal of Speech and Hearing Research*, **3**, 489-500.

Thal, D., Marchman, V., Stiles, J., Aram, D., Trauner, D., Nass, R. and Bates, E. (1991). Early lexical development in children with focal brain injury. *Brain and Language,* **40,** 491-527.

Thal, D., Tobias, S. and Morrison, D. (1991). Language and gesture in late talkers: A one-year follow-up. *Journal of Speech and Hearing Research,* **34,** 604 - 612.

Vargha-Khadem, F. and Passingham, R. (1990). Speech and language defects. *Nature,* **346,** 226.

Vargha-Khadem, F., Isaacs, E., Papaleloudi, H., Polkey, C. and Wilson, J. (1991). Development of language in six hemispherectomized patients. *Brain,* **114,** 473 - 495.

Wulfeck, B. (1987). *Sensitivity to grammaticality in agrammatic aphasia: Processing of word order and agreement violations.* Doctoral dissertation, UCSD.

Wulfeck, B., & Bates, E. (1991). Differential sensitivity to errors of agreement and word order in Broca's aphasia. *Journal of Cognitive Neuroscience,* **3**, 258-272.

Wurm, S.A. (1993, April). *Language contact and unusual semantic features: Some ideas on language and thought.* Paper presented at the FESN Study Group on Evolution and Neurology of Language, Geneva.

Zurif, E. & Caramazza, A. (1976). Psycholinguistic structures in aphasia: Studies in syntax and semantics. In H. & H.A. Whitaker (Eds.), *Studies in neurolinguistics* (Vol. I). New York: Academic Press.

Zurif, E., Swinney, D., & Garrett, M. (1990). Lexical processing and sentence comprehension in aphasia. In A. Caramazza (Ed.), *Cognitive neuropsychology and neuro-linguistics: Advances in models of cognitive function and impairment.* Hillsdale, NJ: Erlbaum.