5

Revisiting Modularity: Using Language as a Window to the Mind*

SUSAN CURTISS

5.1 Introduction

This chapter takes a new look at modularity, both so-called 'big modularity' (BMod) and 'little modularity' (LMod). In it, I reconsider the viability of the BMod claims that language, and in particular, grammar, represents a domain-specific mental faculty, one that rests on structural organizing principles and constraints not shared in large part by other mental faculties and in its processing and computation is automatic and mandatory, and the LMod assertion that language, itself, is comprised of distinct submodules, each of which can be seen to develop and function separately. The first part of the chapter concentrates on BMod issues, the second part on LMod. I will at the end summarize what I believe I have shown and draw a final conclusion.

Having written my dissertation on the Genie case, it is no surprise that I have devoted much of my research to following up on ideas this case led me to consider. Her cognitive profile—a severely limited grammar that lacked functional structure, including all I- and C-system functional elements and the syntactic operations, *Move* and *Agree*, alongside excellent vocabulary learning ability, good ability to initiate and sustain topics, excellent ability to apprehend complex hierarchical structure outside the realm of grammar, good ability to logically sequence pictures into stories, ability to count, ability to draw in silhouette and capture in drawing juxtapositions of objects and events that she could not communicate verbally, bafflingly powerful non-verbal communicative ability, and superior visual and spatial cognition—compelled me to

* In deciding what contribution I could make to this volume that honors Carol's work and reflects influences her work had on my own, I determined that her work's greatest contributions to my own were, perhaps, in encouraging me to think 'out of the box', to look where others had not looked to find data that spoke to those issues I have designed my research program to address, and to take a new look at individuals and populations that can teach us something about both language and human nature. Carol was always concerned about the humanity of those she studied and worked with, and we all know how wonderfully well she managed to do this. Her example has inspired me throughout my career to try to do the same.

explore the issue of modularity further and to attempt, throughout my career, to generate empirical data which bore on issues of modularity.

This chapter brings to bear empirical data from a wide variety of sources that speak to the questions involved. The first part of this chapter focuses on data that speak to BMod, drawing from both normal and atypical data to demonstrate non-trivial dissociations between grammar and other mental faculties. To this end, I discuss relevant data from ERP and imaging studies of the neural basis for language, cases of linguistic isolation, mentally retarded children, normally developing children acquiring ASL as a first language, children and adolescents with Specific Language Impairment (SLI), with Turner's syndrome and with other developmental anomalies, and adults with acquired aphasia and progressive dementia.

The second part of this chapter addresses LMod. I again examine data from studies pertinent to the neural mediation of language and from children and adolescents with SLI, as well as data from adolescents and men with Klinefelter's syndrome, adults with acquired aphasia, and adults with progressive dementia. In both parts some of the data examined come from research done by others; some are data drawn from my own work.[1]

5.2 Big Modularity

What would it mean for language to be a distinct module of the mind/brain, based on domain-specific organizing principles? What kind of evidence would support such an idea? To begin, language, like all of cognition, lives in the brain. Is there evidence for the tenets of BMod from work examining the neurology of language? It should be noted that even if it were the case that grammar-dedicated brain tissue could *not* be segregated from brain regions not dedicated to the representation or processing of grammar, there is, I would argue, abundant evidence supporting the existence of language/grammar as a discrete, separable, functional human biological system. However, there *are* neurological arguments to be made supporting BMod. Space constrains me to mention but a small fraction of this ever-growing body of work that suggests that notions underlying the claims of BMod are correct.

5.2.1 *The neurology of language*

To begin, there is the columnar organization of neurons in the cytoarchitecture of the human cortex (e.g., Roland and Zilles 1998 and sources therein). Columnar neural cytoarchitecture is not, in and of itself, evidence that the mind is modular, but some experimental research appears to strongly support functional modularity, and it is telling that much of this research examines what might be predicted to be most closely

[1] A list of tests used in collecting my data on Genie, Chelsea, TS children, KS individuals, and mentally retarded children and adolescents is presented in the Appendix, which can be found on my website at <http://www.linguistics.ucla.edu/people/curtiss/index.html>. The Appendix also includes example items from many of these tests.

aligned with and inseparable from spoken language representation and processing neurally—namely, acoustic processing.

5.2.1.1 *Phonological processing separable from (non-linguistic) acoustic processing* A number of studies appear to show that distinct columns of neurons or neural tissue respond to phonological categories and are different from those that respond to acoustic distinctions (e.g., Phillips et al. 2000; Dehaene-Lambertz et al. 2002; Dehaene-Lambertz and Pena 2001). These studies and numerous others have looked specifically at the neural instantiation of acoustic processing and whether it can be shown to underlie phonetic/phonological processing or even be prior to and/or inseparable from it, or whether evidence can be generated that argues for their separability. One technique studies have used to examine this issue is to look at the timing of neural responses to sounds or sound combinations utilizing Event Related Potentials ERPs, Optical Scanning, or MEG. Using Oddball paradigms[2] that generate Mismatch responses ('Mismatch Negativities', MMNs) to signals perceived to be distinct from others in a series, experimenters have manipulated acoustic signals such that while the outlier is distinct acoustically, it may or may not be phonologically.

One set of studies of this type has examined the MMN reflex of categorical perception. In these studies the 'Standards' are stimuli that are distinct acoustically but not phonologically; i.e., they are acoustically distinct but within phonological category (WC) signals. This series of WC stimuli is followed by an equivalently acoustically distinct signal, but one that crosses phonemic category boundaries (an AC stimulus) (e.g., Dehaene-Lambertz 1997; Dehaene-Lambertz and Gliga 2004). Such studies uniformly find that the AC signals generate a robust MMN, while the WC stimuli do not. The WC stimuli generate a weaker, slower, and spatially different neural response.

Building on this idea, Phillips et al. (2000) devised an especially compelling experiment to elicit a neural response that would unequivocally demonstrate a distinction between phonological and acoustic processing. Phillips et al. constructed a set of CV stimuli to demonstrate an MMN to the abstract phonological category, the feature [voice], realized differently in stop consonants of different places of articulation (POA). Given that acoustically the signals corresponding to [-voice] or [+voice] in stop consonants at different POAs are very different and can be treated as the same only at the level of phonology, the result that the brain indeed treats these quite distinct acoustic signals as the same across labial, alveolar, and velar stops is persuasive evidence that phonological representation and processing is both cognitively and neurally distinct from acoustic representations and processing.[3] Furthermore,

[2] The Oddball paradigm utilizes a design wherein a series of signals is presented which has the design SSSD—where the Ss (the 'Standards') are the same along some dimension, and the Ds ('Deviants') differ from the Ss along that dimension.

[3] The MMN disappeared when the S and D stimuli all fell into either the – or + voice category. Thus the MMN response could only have been a phonological and not an acoustic response, since all the stimuli differed acoustically.

comparing brain activation to sublexical units for both sign and spoken language, Pettito et al. (2000) report that specific neural tissue is sensitive to phonological patterning regardless of modality.

Other studies of adults whose findings have the same implications abound. As but a few examples, Zatorre et al. (1992) and Burton et al. (2000) both report that the same CVC sequence (thus the same acoustic stimulus) elicits different lateralized neural responses depending on whether the task is a phonological one (e.g., discriminating onsets, codas, or rimes) or a non-linguistic acoustic task (i.e., discriminating pitch). In addition, Jacquemot et al. (2003), again using the Oddpall paradigm, studied speakers of French and Japanese and examined their neurological responses to stimuli that did or did not conform to phonotactically permissible sound sequences. In some sequences, the Ds were sequences that involved long vowels, phonemic in Japanese but not in French; in others the Ds involved a consonant cluster, permissible in French but not Japanese. They found (1) that there was a faster response to a sound sequence that conforms to the phonotactic constraints of one's native language, than to acoustically distinct items that do not constitute a possible phonological sequence; and (2) that the phonological task elicited a spatially different neural response from the acoustic one.

We find the same response pattern in the brains of infants (Dehaene-Lambertz 2000; Dehaene-Lambertz and Baillet 1998; Dehaene-Lambertz and Gliga 2004; Dehaene-Lambertz and Pena 2001; Dehaene-Lambertz et al. 2006; Pena et al. 2003). Dehaene-Lambertz et al. (2002), measuring brain activation of awake and sleeping three-month-olds evoked by forward and backward speech, were able to show that the infant cortex is already structured into several functional regions sensitive to forward but not backward speech. This finding suggests that the precursors of adult cortical language areas are already present and active in infants well before the onset of speech production, despite the fact that synaptogenesis and myelination of these areas are not at all yet mature!

The fact that discrimination of phonological categories takes place more quickly and via different neural 'networks' than the processing of acoustic distinctions in both adults and infants (e.g., Dehaene-Lambertz 1997; Dehaene-Lambertz and Pena 2001) suggests that the widely argued assertion that an acoustic mapping is both prior to and more basic than a direct phonological mapping of discriminable signals is incorrect. More pertinently, it illustrates that discriminating among sounds that are not linguistically relevant is cognitively and neurally distinct from discriminating among sounds that are; i.e., phonology and phonetic discrimination is not reducible to acoustics, even for the sleeping neonate (Dehaene-Lambertz and Pena 2001).

5.2.1.2 *Event Related Potential (ERP) evidence for BMod* An early ERP component referred to as the ELAN (Early Left Anterior Negativity) is a component associated with automatic syntactic processing and structure building and is present in both adults and children, including children as young as two (Hahne and Freiderici 1999;

Hahne et al. 1999, 2004; Oberecker et al. 1995; Pulvermüller and Shtyrov 2003; Pulvermüller et al. 2008).

The ELAN is not only a reflection of automatic processing, it is insensitive to task demands or violation frequency (number of syntactic violations; Pulvermüller et al. 2008; Pulvermüller and Shtyrov 2006; MacGregor et al. forthcoming). Using ERPs to measure MMNs to syntactic violations, Pulvermüller et al. asked subjects to listen to sentences, some of them ungrammatical, some of them grammatical, while performing a demanding acoustic signal detection task.[4] Their subjects had to listen for grammaticality and not only point out when they detected a grammatical violation, but correct the error. Pulvermüller et al. found first, that the syntactic MMN was extremely rapid, occurring at or before 150 msecs following the point at which the relevant information occurred, and second, that the magnitude of the MMN response was unaffected by attention load. This early time window appears to be very narrow, but is robustly present as an index of automatic syntactic processing, a key characteristic of a task-specific, modular response.

Similar findings have been reported for MMN responses to detecting native segments and syllables and word semantics while subjects concurrently carry out a difficult, attention-demanding task. Using, in the first case, native and non-native speech sounds and phonotactically possible or ungrammatical syllables, and in the second, pseudowords and real words, a number of different labs looking at responses to different languages report that neurophysiological signatures of language-specific phonological responses and language-specific, word-specific memory circuits/cell assemblies are activated in the human brain in a largely automatic and attention-independent fashion (e.g., MacGregor et al. 2012, forthcoming; Pulvermüller et al. 2009).

5.2.2 *Language separable from spatial cognition*

5.2.2.1 *Sign language and spatial cognition* It is well known that in the 'modal' brain, (that of the right-handed, typically male individual), spatial cognition is mediated by the right hemisphere, while computational linguistic tasks asymmetrically engage the left hemisphere. This pattern in itself provides clear evidence that these two aspects of human cognition are separate at both the cognitive and neural level. The extent to which these two cognitive domains might become interrelated and therefore less dissociable when the language involved is in part a spatially coded system, i.e., a sign language, has also been explored. Such research in normals largely comprises studies comparing sign language processing with spoken language processing and these, with brain areas activated during spatial tasks. For example, McGuire et al. (1997) found that (outside of motor cortex) the same brain regions were activated by

[4] Pulvermüller et al. divided their subjects into two groups and used two tasks. One group had to watch a silent video while listening to sentences; the other had to determine if a tone was briefly attenuated—a task with a high attentional load, whose stimuli were in the same modality as the language stimuli.

Deaf signers mentally articulating British Sign Language sentences as those activated by hearing speakers when silently articulating sentences of English, in neither case involving areas of the right hemisphere activated during non-linguistic visual/spatial processing. Emmorey (2002) provides a survey and discussion of much of the relevant research in this area. We return to this topic in 5.2.3 below.

5.2.2.2 *Turner's syndrome (TS)*[5] Behavioral data also speak to this issue. Studies of TS, for example, provide compelling evidence for dissociations between language and non-linguistic spatial cognition. TS is a genetic disorder occurring in females that arises from partial or complete absence of an X chromosome. TS is associated with a peculiar cognitive profile characterized by normal grammatical development and function, *enhanced* reading ability compared to age-matched normals in childhood, including the ability to read irregularly spelled words and long unfamiliar regular words (Temple and Carney 1996), normal arithmetic abilities, but impaired number reasoning and severely impaired visual and spatial cognition (Bruandet et al. 2004; Money 1963, 1973; Money and Alexander 1966; Pennington et al. 1985; Rovet 1998 and references therein; Silbert et al. 1977; Waber 1979), a profile that persists from early childhood through adulthood (Temple and Shephard forthcoming).

In my own work, I have studied a number of TS children, including a mentally retarded girl, V, with an IQ of 68, tested over the course of several months (from 9;6–10;0)[6] (Curtiss and Yamada 1981). One of the most notable aspects of the cognitive profile associated with TS is the discrepancy between the absence of visual and spatial defects in the realms of reading, writing, and performing arithmetic calculations alongside pervasive non-language visual and spatial deficits. To wit, despite V's inability to copy a simple square or circle, to draw representationally (see Figure 5.1), to string colored beads in accordance with a visually present model, to build even a simple bridge with blocks or copy any hierarchical stick structure, a preschool level performance on the block design, object assembly, and picture completion subtests of the WISC, a 'defective' level score on visual memory, below all norms performance on the Mooney Faces test, a performance in the 'defective' range on the Thurstone Mental Rotation test and Thurstone Closure Speed test, inability to do either embedded figures task, a preschool level of drawing and copying, the absence of Piagetian conservation in every area except possibly number and an inability to perform the

[5] While there is no debate regarding substantial visual and spatial cognitive deficits in Williams syndrome (WS), TS provides a clearer example of the relevant dissociation than does the WS population, I would argue. For there is ongoing controversy over how intact language is in the WS population. Some research indicates lexical impairments (e.g., Jones 2007; Clahsen and Almazon 2001) or syntactic anomalies (e.g., Karmiloff-Smith et al. 1997; Perovic and Wexler 2007). Other research indicates largely intact syntax, even of complex structures such as complex nominal compounds and relative clauses (Zukowski 2005, forthcoming). However, there is no such controversy over the language or language development in TS.

[6] This work was carried out with J. Yamada.

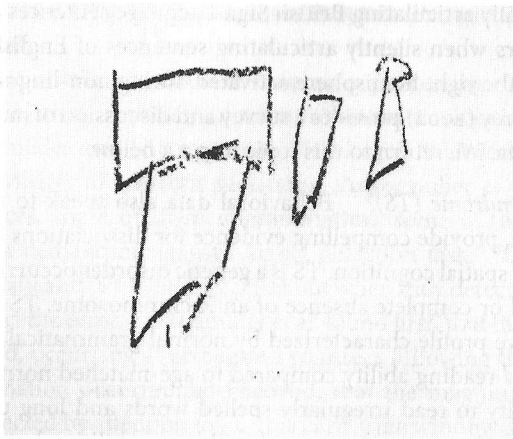

FIGURE 5.1 V's drawing of a house. The two smaller squares alongside the larger one represent windows of the house.

Localization of Topographical Stimuli task, V not only had an essentially mature grammar, but she could read and spell and do simple arithmetic problems, both in her head and on paper. (See Curtiss and Yamada 1981 for further details of this case.)

The profile V displayed is characteristic of that displayed by TS girls and women. Only within the language-related domains of reading and writing and in calculation does one see no evidence of a visual or spatial deficit. Outside these domains, one finds marked visual and spatial impairment.

TS raises the issue of the role of the sex chromosomes in verbal ability, one we will turn to again below in our discussion of research with Klinefelter's syndrome (KS) adolescents and adults. Of relevance here is that it provides empirical evidence for a clear dissociation between language, particularly grammar and language-dependent abilities such as reading, and a number of aspects of non-linguistic cognition, including visual memory, visual constructive ability, number reasoning, visual cognition, and spatial cognition.

5.2.3 *Language separable from both spatial cognition and non-linguistic communication*

Another population demonstrating striking dissociations between linguistic and non-linguistic visual and spatial cognition is brain-damaged Deaf signers. Several studies have documented the relevant dissociations (Bellugi et al. 1993; Corina et al. 1992; Emmorey 2002 and references therein), including also a dissociation between (1) the use of and ability to copy meaningless non-linguistic gestures alone and in combination and (2) linguistic gesture (sign). Importantly, comparing brain-damaged signers

with and without acquired aphasia, one finds a double dissociation[7] between language on the one hand and non-linguistic communication and spatial cognition on the other, with aphasics showing normal visual and spatial cognition outside of the use of the spatialized syntax of ASL, and right-hemisphere-damaged non-aphasic signers displaying impaired visual and spatial cognition.[8] A striking illustration of this double dissociation, reported in Bellugi et al. (1989), is left-hemisphere-damaged signers producing via aphasic utterances accurate descriptions of the spatial layout of the furniture in a room of their house, contrasting with right-hemisphere-damaged, *non*-aphasic signers producing in grammatical sentences, descriptions of the arrangement of furniture in a manner that reflects hemi-neglect (neglect of the left side of space). Notably, their spatial cognitive deficits do not translate into deficits in comprehending or producing spatially transmitted aspects of sign grammar.

Thus we see deficits in the apprehension of linguistic gesture not transferring to the realm of *non*-linguistic gesture, communicative or not, and deficits in visual and spatial cognition not transferring to the realm of the use of space for grammatical purposes—the double dissociation referred to above.

The dissociation between linguistic and non-linguistic gesture seen in brain-damaged signers, and therefore between grammar and non-verbal communication, is also seen in the acquisition of sign vs communicative gesture in normally developing children. Pointing as a communicative gesture comes in prelinguistically, typically around the end of the first year of life (Bates et al. 1987; Pettito 1987). Completely isomorphic pronouns in ASL, however, emerge later, in correspondence with the timing of the acquisition of pronouns in English and other languages. This fact is rather striking, since not only are pronouns formationally isomorphic to the pointing gestures already established as part of the child's communicative repertoire far earlier, but also because ASL pronouns are iconic (i.e., a point to the speaker means 'you', a point to oneself means 'me', etc.). Children appear to process pronouns as formal linguistic units, as functional units of grammar, ignoring the iconicity and isomorphism they could readily exploit if grammatical development were driven by mechanisms shared by those underlying communicative development.

A similar picture is found with the acquisition of possessive determiners and negation in ASL (Jackson 1984). Again we find that ASL-learning children ignore the communicative isomorphism with the earlier learned and used communicative 'head-shaking' gesture of the negative or the transparency of the possessive determiner for first and second person 'my/mine' or 'your'. Rather, they master these structures in timing and manner so as to indicate that they are developing a grammatical system,

[7] A double dissociation between two phenomena is the neuropsychological benchmark for determining their cognitive independence.

[8] Recent research emanating from Helen Neville's lab at the University of Oregon suggests that cerebral organization of these systems may not be uniform across the deaf population. That, however, is not the issue under discussion.

with functional structure that is independent of their communicative repertoire, blind to iconicity as a cue for grammatical acquisition.

5.2.4 *Dissociations between grammar and non-linguistic communication*

A double dissociation can also be found between grammar and non-linguistic communication in adults and children, in that one finds selective deficits in one and not the other. Autism is defined in part by impairments in social interaction and communicative competence, such that regardless of where an individual may fall on the autism spectrum, some impairment in social interaction and communicative competence is always present. However, high-functioning autistic individuals and those with related Asperger's syndrome develop and maintain normal grammars. The opposite profile is seen in many acquired aphasics, in Deaf individuals who have little knowledge of a first language, with late L1 learners as exemplified by Genie and Chelsea and in most individuals with SLI. In these instances, we find impaired grammars alongside good non-verbal communication abilities.

5.2.5 *Multiple dissociations between grammar and other mental faculties*

Examining a number of different populations, I and others have demonstrated a double dissociation between grammar and non-linguistic cognition including communication.[9] In one direction I have reported on several cases of mentally retarded children and adolescents (e.g., Curtiss 1982, 1988a,b, 1995, 2011) with an island of intact function, namely, grammar, within a sea of pervasive and comprehensive non-grammatical deficits, including deficits in areas some have hypothesized to underlie or be necessary for language development—sequencing, ability to construct and apprehend hierarchical structures, normal auditory/verbal short-term memory, symbolic thought as revealed through play or drawing, categorization, rule formation or generalization, to name just some. (See above references for details.)

Individuals with spina bifida (see Stough et al. 1988) and hydrocephalus (see Tew and Laurence 1979a,b) have also been reported to show a profile of good grammatical function coupled with mental retardation and atypical communicative behavior, giving rise to the terms 'chatterbox syndrome' and 'cocktail party syndrome'.

An additional study describing a case of intact grammatical development and function in the face of substantial non-linguistic impairments in non-grammatical domains is the case of Françoise (Rondal 1995). Françoise, a woman with Down Syndrome with an IQ of 64/65 and an MA of 7;4 when she was in her early to mid-thirties, like the MR children I report on, appears to have an intact, mature, age-appropriate grammar. Moreover, though Françoise displays short-term memory and vocabulary performance comparable to her MA-matched peers, she shows intact,

[9] Again, tests used in my research are listed in the appendix to this paper, found at <http://www.linguistics.ucla.edu/people/curtiss/index.html>.

fully adult working memory for sentence processing and sentence repetition and although unable to make semantic plausibility judgments, can make grammaticality judgments of long, syntactically complex sentences.

The mentally deficient individuals with intact grammars I and others have documented, though not autistic, could be considered Linguistic Savants, with savant abilities in grammar. So, too, is Christopher (e.g., Smith and Tsimpli 1995), who is autistic, and whose pervasive non-linguistic retardation makes his intact computational linguistic abilities quite remarkable. (At last estimate Christopher could speak and understand sentences in more than twenty languages, including British Sign Language.) An additional case is that of Daniel, a 31-year-old high-functioning autistic whose savant areas include number and language. Daniel is reported to speak eleven languages, and to have learned to speak Icelandic in a matter of days (for a television interview). He has also written an autobiography of sorts (Tammet 2007).

Establishing a double dissociation between grammar and non-linguistic cognition, we see the flip side to the profile of Françoise, the mentally retarded children I have written about, and cases like Christopher and Daniel in Genie, who has no clear deficits outside of grammar and psychosocial function, and in Chelsea, an adult linguistic isolate who shows a grammatical profile even more impaired than Genie's (Chelsea appears to have no grammatical system at all but evidences robust vocabulary learning[10] (Curtiss 1995) alongside non-grammatical functioning between a 10–11-year-old level. (This profile is evidenced to a less extreme degree by Grammatical SLI children; see section 5.2.7 below.) Like Chelsea, other deaf adults who were not exposed to a sign language until adulthood (e.g., Newport 1990) also manifest significant grammatical deficits together with normal (even superior) non-verbal communication and normal cognitive function, even normal number knowledge and arithmetic ability.

5.2.6 *Dissociations between language and number*

Chelsea can add, subtract, multiply, and divide, manipulate money well enough to conduct restaurant and shopping transactions, keeps a correctly reconciled checkbook (Glusker, personal communication) and can tell time—all without a grammar (Grinstead et al. 1998, 2002).

There is evidence from other cases as well for the independence of language and number, both in development and breakdown. There is abundant literature documenting acquired impairments in language with the number faculty spared as well as acquired acalculia with no aphasia. In addition, there is other behavioral evidence that the domain of number can develop or remain functional despite the absence of language and grammar. Cases of individuals who appear to have fully developed

[10] Genie has deficits in sociocultural aspects of discourse; namely, using the cultural rituals of discourse, including conversational operators and turn-holding devices that differ from culture to culture, while Chelsea has no difficulty in this area but shows difficulties in conversational aspects of discourse, such as contributing to a conversation's progress, most probably because of her poor comprehension.

number faculties; i.e., knowledge of how numbers work—knowing how to perform arithmetic operations at will, from counting to multiplication, despite a complete lack of language—are reported by Schaller (1991). A fascinating study described by Schaller of a community of immigrant deaf adults without language, with a focus on a particular case study within this community, documents how number systems can be developed or readily learned despite the total absence of language, even vocabulary. Galvan (reported in Schaller) describes another deaf man, who despite having no language, learned to tell time and utilize his knowledge of time to negotiate bus schedules. Both of these cases and others like them point to number being its own distinct mental module, separate from language. (See Curtiss 2011 and Grinstead et al. 1998, 2002 for more details and discussion of such cases.) A bit more on number is presented in section 5.3.7 below.

5.2.7 *Specific Language Impairment (SLI)*

Anywhere from 2 to 19 percent of children are estimated to have developmental language impairment not associated with hearing loss, mental retardation, frank neurological damage, or social/emotional impairments (Nelson et al. 2006). Although we will return to this population in section 5.3, the G-SLI subgroup within this population, so called because they present with selective grammatical (G) impairments, while non-linguistic cognition remains intact, offer another source of compelling data in support of BMod and the double dissociation displayed by cases and populations already discussed.

The most relevant research on G-SLI has been done largely by van der Lely and colleagues.[11] Van der Lely and her colleagues have demonstrated normal non-linguistic cognitive function not only by their selection criteria but also by demonstrating that G-SLI individuals perform like age-matched controls in their ability to solve logical problems as tapped by a modified game of *Cluedo* (van der Lely and Battell 2003) and the ability to perform a complex visual inference task (van der Lely et al. 2004). Van der Lely and her colleagues have also established that individuals with G-SLI are typically unimpaired in the acoustic processing that some researchers have hypothesized underlies SLI itself (Tallal 1976, 2000; Rosen et al. 2009; van der Lely et al. 2004)[12].

In addition, heritability studies of children with G-SLI (and others) suggest genetic factors dedicated to grammar separable from non-language cognition (see, for example, Stromswold 2001 for a general review; Bishop, North, and Donlan 1996; van der Lely and Stollwerck 1996; van der Lely 2004). G-SLI children are found to have a higher incidence of family histories with language impairments than normally developing children. Taken together with data characterizing G-SLI as a domain-specific deficit, this then provides another source of evidence that there is a brain system dedicated to grammar alone.

There is increasingly abundant data indicating the heritability of language both in children with a variety of language or language-related deficits from twin studies showing greater heritability of language impairments in monozygotic (MZ) than dizygotic (DZ) twins, from familial aggregation studies showing significantly higher family histories of language or reading problems in families of children with SLI than in normally developing children and from adoption studies that show a significantly higher correlation in language abilities between adopted children and their biological parents than with their adopted parents (Bishop et al. 1996, 1999a,b; Felsenfeld and Plomin 1997; Hohnen and Stevenson 1999; Stromswold 2007). These data also constitute evidence for genetic factors dedicated to language alone. This is not surprising to anyone working in the field of grammatical development, since normal language development is characterized by uniformity in timing of emergence and of major developmental linguistic milestones across the species. Given the additional force of poverty of stimulus arguments, normal language development itself can be seen as strong evidence that language is a biological system that is part of our genetic endowment as humans, and therefore, genetically transmitted. It is simply because of the selective deficits manifested by the SLI population, that evidence regarding the heritability of language and its subsystems by those with language impairments may seem more persuasive as evidence for a domain-specific, genetically determined mental module we call 'language'. I return to this topic briefly in section 5.3.5 below.

5.2.8 *Acquired aphasia and Dementia of the Alzheimer's Type (DAT)*

5.2.8.1 *Acquired aphasia and intelligence* With agrammatic (non-fluent) aphasia, one typically sees intact intelligence (Bay 1964; Varley and Siegal 2000), another noteworthy dissociation of grammar from extra-grammatical cognition. The manuals for and findings from clinical tests given worldwide for classifying aphasic disorders take as a given at this point in time, that in aphasias whose predominant characteristics are anomia or belabored and halting production with particular difficulty with functional elements, intelligence remains largely intact. This fact is reflected in the depression that is a frequent artifact of these aphasias, a depression based on such aphasics' keen awareness of their functional loss. Largely intact intelligence alongside acquired language loss provides, then, yet another source of evidence that language and non-language cognition are not of a piece, but rather are separate and separable.[13]

[11] Many other linguists have done important, even seminal work characterizing the specific linguistic impairments of individuals with SLI. I concentrate on van der Lely and her colleagues' research here, for its particular relevance to BMod issues.

[12] The discovery of SLI in a signer (Morgan et al. 2007; Mason et al. 2010) is further evidence against the hypothesis that an impairment in the ability to process rapidly changing acoustic information underlies SLI, since in the sign signal, information is presented at a far slower rate.

[13] Work demonstrating equivalent intelligence (little 'g') of the right and left hemispheres also provides evidence of the independence of language and intelligence (e.g., Zaidel, Zaidel, and Sperry 1981).

5.2.8.2 *Dementia of the Alzheimer's Type (DAT)* With agrammatic aphasics and adults with DAT we have two populations that together reveal a double dissociation of grammar and non-grammatical mental faculties. Alongside cognitive dissolution, progressive dementia is characterized by lexical and other forms of semantic loss, even early on. While non-linguistic cognition and extra-grammatical aspects of language (e.g., lexicon and discourse functions) deteriorate, however, phonological and morphosyntactic knowledge appears to remain largely intact, often until late stages of DAT (Kempler 1984; Kempler et al. 1987). I return to this population in section 5.3.3, where I concentrate on the selective preservation of submodules of language; however, I mention the DAT population here, as they reflect in breakdown an adult parallel to developmentally retarded individuals with selectively intact grammatical development, much as adult acquired aphasia is in many respects a breakdown parallel to SLI.

5.2.9 *Selectively impaired non-linguistic cognition*

Selective developmental and acquired impairments in non-linguistic cognitive domains are well established in the clinical literature and include the domains of number, spatial cognition, facial recognition, and other visual agnosias, visual cognition in general, proprioception, non-linguistic communication, and music. Selective impairments of a number of different cognitive domains or submodules within those domains will be discussed in more detail in section 5.3.6, but the fact that so many cognitive domains in addition to language can be selectively spared or damaged both in breakdown and development points to a model of the mind comprised of a number of distinct faculties which rest on task-specific principles, constraints, and mechanisms for processing domain-relevant information. As we will see, each of these can be fractionalized and subsystems within them develop abnormally or become impaired. Space constraints prevent me from elaborating on this additional, important area of evidence supporting the basic claims of BMod; however, I mention them here to point out that the issue of modularity of mind is one that speaks not just to language and mind, but to the broader issue of the nature of mind in general.

5.3 Little Modularity

Above I enumerated a variety of populations and kinds of evidence in support of the basic claims of BMod. In this section of the chapter we will do the same for the claims of little modularity (LMod); namely, that language (like other cognitive systems) is not all of a piece, and that different subsystems within language—lexicon, pragmatics, and the computational system (the grammar)—can be selectively impaired in development and breakdown.

5.3.1 *Acquired aphasia*

Lack of space prevents me from richly explicating relevant findings from studies of acquired aphasia, but linguistic aphasiology has documented selective impairments of (1) the lexicon, even semantic and syntactic category-specific deficits within lexical loss (e.g., Hart et al. 1985; Jodzio et al. 2008; Caramazza 1988); (2) morphology (Thompson et al. 2002), including selective impairments differentially affecting derivational and inflectional morphology (e.g., Miceli and Carramazza 1988); and (3) syntax (Grodzinsky 1986; Grodzinsky and Finkel 1998; Friedmann et al. 2006; Bastiaanse and van Zonnefeld 1998; Bastiaanse and Thompson 2003; Buchert et al. 2008).[14] A cottage industry seems to be devoted just to the selective loss of 'closed-class' elements in the lexicon (Bradley, Garrett, and Zurif 1980) and at which level of grammar or processing the relevant generalization regarding what is impaired can best be captured (e.g., Kean 1980; Grodzinsky 1986; Golston 1991). Researchers have also noted selective deficits in pragmatics (e.g., Bottini et al. 1994; Champagne-Lavau and Joanette 2009), typically following RH damage, where the grammar and lexicon remain essentially intact, but 'non-ordinary' language (e.g., appreciating metaphor, jokes) is affected.

Studies of rare forms of aphasia reveal additional interesting patterns of selectively impaired vs selectively intact pieces of language. Both mixed transcortical aphasics and transcortical sensory aphasics spontaneously and unconsciously (i.e., automatically and mandatorally) correct minor phonological or morphosyntactic errors (e.g., Whitaker 1976; Davis et al. 1978), but appear impervious to the semantic plausibility of the sentences they are asked to repeat.

Linguists have focused particular attention on agrammatism, with most attempting to delimit exactly which syntactic principles or piece(s) of computational machinery are affected in agrammatism. Many have suggested, for example, that the operation *Move* is selectively impaired (Bastiaanse and van Zonnefeld 1998; Bastiaanse and Thompson 2003), providing a striking adult parallel with S-SLI (Friedmann, Gvion and Novogrodsky 2006). There is ongoing debate as to whether the agrammatic's loss is one of being able to compute the entire syntactic tree, with all of the relevant functional heads and internal functional structure (e.g., the Trace Deletion Hypothesis (Grodzinsky, 1986); the Tree-Pruning type Hypotheses (Hagiwara 1985; Friedmann 2001; Friedmann and Grodzinsky 1994, 1997)), or is one of selective loss either for parts of that functional structure (e.g., elements/features marking finiteness) or of the operation *Move* triggered by such features (e.g., Friedmann 2001; Friedmann and Grodzinsky 1994, 1997; Grillo 2009; Grodzinsky and Finkel 1998; Friedmann

[14] It is intriguing that, to my knowledge, deficits in phonology unaccompanied by other deficits are not documented. One can speculate as to why this is the case, taking into consideration the interdependence and interrelation of phonological processes and the lexicon or between phonology and morphological realization, among other factors. It remains a curiosity, nonetheless.

et al. 2010). It is striking that problems with *Move* are noted for both the G-SLI/S-SLI population and agrammatics.[15]

Whatever the correct analysis, the acquired aphasias provide clear evidence that language is decomposable into separate and separable submodules or subcomponents, the principles of which can be selectively damaged or remain intact, including the piece of the parser by which grammatical form is filtered for errors—all evidence supporting the fundamental tenets of LMod.

5.3.2 *Klinefelter's syndrome (KS)*

In TS we saw the potential influence of the sex chromosomes, in particular, the X chromosome, on language function. In KS we see another instance implicating the X chromosome in language. In TS, there was the partial or complete absence of one of the X chromosomes, resulting in *enhanced* reading abilities. KS is a genetic disorder in which males have an extra X chromosome and have a sex chromosomal make-up of forty-seven XXY. There is very little linguistic research on KS, but the little research that exists on language, reading, and spelling in KS reports that KS boys are developmentally dyslexic, have spelling deficits, and are frequently mildly retarded, with verbal IQ typically lower than Performance IQ (e.g., Bender et al. 1986; Netley and Rovet 1982; Rovet et al. 1996; Boone et al. 2001). We see an influence of the X chromosome again here, but this time an extra X has the effect of causing reading difficulties—the opposite profile to that seen in TS girls.

I have been conducting research on KS adolescents and men[16] with the objectives (1) to determine how long such deficits persist across the lifespan in KS males and (2) to conduct a comprehensive linguistic-theoretically driven investigation of language production and comprehension in KS.[17] Our subjects to date comprise twelve adolescents and men with KS, none retarded. Eight are highly educated professionals (engineers, lawyers, accountants, teachers, insurance brokers), two are high-school students, two are college students. Subjects are presented in Table 5.1 below.

Our findings to date are summarized in Table 5.2 below. Surprisingly, we have not found any traces of persistent reading difficulties, at least at the level of the word, even

Table 5.1. KS subjects by age

Subj.	MS	GS	TR	JK	GS	MC	FR	SP	PG	GM	CM	WL
CA	14	18	32	34	68	16	22	28	47	49	36	17

[15] This raises the possibility that *Move* itself and/or the principles that trigger or underlie it might represent a separable submodule within the syntax.

[16] This research has been done in collaboration with S. de Bode and D. Geschwind and is still ongoing.

[17] Some of the subjects overlap with those studied by Boone et al. (2001), yet our findings diverge quite a bit from theirs—the result most probably of the nature of the tests and tasks used.

Table 5.2. Test performance of KS subjects

Subj.	MS	GS	TR	JK	GS	MC	FR	SP	PG	GM	CM	WL
SS Relatives	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
SO Relatives	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
OS Relatives	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
OO Relatives	4/5	5/5	3/5	5/5	5/5	3/5	5/5	5/5	5/5	5/5	3/5	5/5
Obj. Clefts	4/5	5/5	3/5	5/5	4/5	4/5	4/5	5/5	5/5	5/5	2/5	5/5
S-V Agreement	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20
Negative Scope	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5	5/5
Binding* Items	2/4	1/4	2/4	0/4	1/3	1/3	0/3	0/4	1/4	2/4	1/3	2/4
Control* Items	2/6	0/6	1/6	3/6	0/6	0/4	0/5	1/4	1/3	0/3	1/5	2/6
Presupp.* Items	2/4	1/4	2/4	3/4	1/4	3/4	0/4	0/4	4/4	0/4	4/4	2/4
Entlmt. Items	1/4	3/4	4/4	1/4	2/4	0/4	1/4	4/4	3/4	2/4	4/4	4/4
Homophones	18/18	18/18	18/18	18/18	18/18	18/18	18/18	18/18	18/18	18/18	18/18	18/18
Reading Homophones	30/30	29/30	30/30	29/30	30/30	30/30	29/30	29/30	30/30	30/30	30/30	30/30
Reading Rhymes	28/30	29/30	30/30	30/30	29/30	30/30		29/30	28/30	30/30	30/30	30/30
Reading Nonsense Words	18/20	19/20	20/20	18/20	17/20	20/20	20/20	18/20	20/20	19/20	17/20	17/20
Curtiss Agreement	32/32	32/32	32/32	28/32	32/32	32/32	32/32	32/32	32/32	32/32	32/32	32/32
CELF-PFS	>norms	>norms	>norms	>norms	>norms	>norms	>norms	>norms	>norms	>norms	>norms	>norms
Gopnik Plural	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20	20/20

*From the Binding, Control, and Presupposition Test

in our youngest subjects. All twelve read the words on our Reading Nonsense Words Test like the normal adults we used as controls, making at most three 'errors' (an error defined as a pronunciation that none of our twenty native English speaker controls produced), three of the twelve made one error on the Reading Homophones Test, and only two made two errors on the Reading Rhymes task[18], with the remainder making either no errors (eight) or one error (two). Additionally, all twelve have normal speech and conversational abilities and frequently produce sentences with complex syntactic structures, including passives and embedded clauses of all types, including relative clauses with object extractions.

However, 'to a man', all twelve show difficulty with Binding and Control, with some also doing poorly on tasks exploring Presupposition and Entailment as well. A few have also shown difficulty with the Object Clefts and OO relatives subtests of the CYCLE-R, and one subject made four errors with nonce items on the Curtiss Agreement Test, failing to add the /-s/ 3rd singular marker to stems that ended in a sibilant, and changing one stem from [pɛ̃m] to [pɛ̃nz].

The Binding and Control items of the BCP Test provoked comments like, 'This is giving me a headache. Can we please stop?' from almost all the subjects, and other comments like, 'I have no idea' or 'Maybe' or 'How should I know?' were made repeatedly throughout the test on these items. Given how well these same subjects performed on the Sentence Judgement Test and the large battery of comprehension and elicited production tests of grammar, which require correct sentence interpretation because of the foils on the comprehension tasks put there to test just that and control of the large number of grammatical structures tested on the production subtests, our KS subjects appear to have a quite selective and specific 'hole' in their grammars—one that involves ready access to the Binding Principles or the relevant C-Command relations for Principles A and B and construal of nominals. Some also appear to have deficits in the complex verb semantics of presupposition and entailment, all of these, perhaps, deficits at the level of the syntax/semantics interface. Such selective deficits are evidence that the computational component is divisible into subsystems far more specific than the submodules of phonology, morphology, syntax, and semantics (logical form). They provide support for a linguistic theory that incorporates interface levels of computation and representation, after the fashion of Minimalist models, as an example. In any event, these findings do not corroborate those of others who have worked on this population, though it should be stated that ours may be the first linguistically informed investigation into the grammar of the KS population.

5.3.3 *DAT*

In research with Kempler, we studied a population of twenty adults with DAT who varied along a continuum of severity (as measured by the Mini Mental Status exam).

[18] All of these reading tests were designed to *elicit* errors by including different words with different pronunciations for the same letter sequence (*said–paid*), same pronunciation for very different letter sequences (*write–right*), or by requiring close attention to orthographic content (*dose–does, loose–lose*).

We conducted several behavioral experiments on this population, including two that I will briefly describe here. (See Kempler et al. 1987 for a full description of this research.) In one study we examined spontaneous speech to determine whether the range of syntactic structures used, the frequency of a predetermined set of syntactic structures used, and the type and frequency of morphosyntactic vs lexical/semantic errors in our DAT samples differed, and if so, in what way(s) from that found in comparably sized samples of speech by SES, age, and sex-matched controls. The two groups differed significantly in the number of lexical/semantic errors made, with the DAT group making significantly more sem./lexical errors ($T = 3.351, p < 0.005$). The number of morphosyntactic errors made by the DAT subjects ranged from none to three, with four of the twenty subjects making no such errors. In contrast, all DAT subjects made at least three semantic errors, with three subjects making more than twenty-six such errors. Fourteen of the twenty normal controls made no errors of any kind; the remaining six made only six errors in total, four syntactic and two lexical. Moreover, the number of lexical/semantic errors made correlated significantly with severity of illness ($r = .7057, p < 0.025$), while morphosyntactic errors appeared to be independent of disease stage ($r = .1287, p > 0.05$).

Additionally, the frequency with which specific construction types were used was rank-ordered and compared and was almost identical between the two groups ($r = .9833, p < 0.0000$) and the structural complexity embodied in the sentences used was also compared (with complexity indexed by constituent movement and number of embedded clauses), and again no significant differences were found ($t = -.664$, $p > 0.05$). In contrast, the DAT population showed difficulty with conversational pragmatics, often drifting off-topic or interjecting inappropriate comments.

The second experiment involved examining disambiguating differently spelled homophones by means of a semantic vs a syntactic cue (using the Curtiss and Kempler Written Homophones Test). Words were spoken aloud in pairs and subjects asked to write the words just spoken. A word semantically related to one member of the homophone pair accompanied half the items; a word providing a syntactic cue occurred with the other half. (Each word was presented with each type of cue (e.g., *lake*-sea, *the* sea; *look*-see, *I*-see).) There was a significant difference in the ability of the DAT subjects to make use of the two kinds of disambiguating cues, with the ability to utilize syntactic cues far more preserved than the ability to make use of the semantic cues ($t = 6.147, p < 0.001$). These findings paint a striking picture—morphosyntax is significantly more preserved than is the lexicon or phrasal semantics. This finding again points to the decomposability of the linguistic system, with semantics (lexical and phrasal) and pragmatics in this case far more impaired than the computational system.

5.3.4 *SLI*

In section 5.2.7 I discussed the selective impairment to the computational system evidenced by G-SLI children. However, SLI is an umbrella label for a heterogeneous

population, one that can readily be divided into subgroups with differing impairments. For many individuals with SLI, the linguistic deficits involved are widespread and affect linguistic performance seemingly across the board, from word learning, to articulation, to grammatical development, to pragmatic function. But for many with SLI their deficits are much more circumscribed. In a study examining just this, Friedmann and Novogrodsky (2008) identified SLI children who had only a phonological deficit ('PhoSLI'), only a syntactic deficit ('SySLI'), a selective lexical impairment ('LeSLI'), or a selective pragmatic deficit ('PraSLI'). A few children in their study showed deficits in more than one area, but the majority of the children in their sample did not. Together with the G-SLI children studied by van der Lely and colleagues, all of whom are reported to have deficits implicating the computational component, we find in this single developmental population evidence for selective disruption of the computational component as opposed to the lexicon or pragmatics (the G-SLI children), and a variety of SLI types, each of which manifests selective deficits in only one part of the linguistic system, including all its subsystems, phonology, morphosyntax, lexicon, and/or pragmatics.

Many other linguists studying SLI have identified the deficit in the children they studied as being very discrete, for example, involving only the marking of finiteness, or only tense, or only agreement, or primarily a deficit in affixal morphology, or verb movement, or the operation *Move* more generally, or hierarchical complexity (defined as a branching structure, regardless of the subsystem of the computational component involved). Although at this time there is no agreement among linguists as to the best characterization of SLI, the current state of the art in research on SLI from a theoretical linguistic perspective is that this population is one that illustrates LMod in a strong form. The impairments in the SLI population demonstrate that language can be divided into many distinct components and subsystems, and within these, particular principles or operations can, themselves, be selectively impaired or spared.

5.3.5 *Evidence for LMod from studies on the genetics of language*

Although still relatively new, the study of the genetics of language already provides support for LMod. Though there are many kinds of studies that comprise this field (e.g., concordance studies, familial aggregation studies, linkage studies), most of the relevant studies for our purpose come from studies of MZ twins, one or both of whom have SLI. These studies examine the family histories of these twin pairs to determine if there is a greater incidence of language or language-related impairments in such twins than is found in the families of MZ twins, neither of whom have SLI.

Stromswold (2007) conducted a meta-analysis of twin studies and reports that for both language-impaired and normal twins, genetic factors are found to affect vocabulary the least, syntax a bit more, articulation and phonology even more, and general language function most of all. Interestingly, there is little genetic overlap between babbling and other areas of linguistic development, suggesting that different genetic factors underlie babbling than other aspects of language.[19] Her meta-analysis of twin studies indicates a 68 percent heritability rate for MZ twins' phonological development, a 56 percent heritability rate for MZ twins' syntactic development and performance, and a 40 percent heritability rate for MZ twins' lexical and lexical access abilities.

Twin studies of this kind provide clear evidence of a significant heritable factor for language, indicating, as stated in section 5.2.7 above, that genetic factors play a non-negligible role in language, both the language of normals and those with SLI. Moreover, these studies demonstrate that genetic factors affect all aspects of language, though I report here only the findings for phonology, morphosyntax, and the lexicon. While clearly there are genetic factors that are not specific to language that together with others may influence language development, it appears that some genetic factors *are* specific to language and include genetic factors specific to only *pieces* of language. Thus, here is another source of data that speak to LMod and support its basic tenets.

5.3.6 *Evidence for LMod from studies of the neurology of language*

Recent electrophysiological evidence provides fascinating evidence in support of LMod. Using intracranial electrophysiological (ICE) recordings, a technique that allows microscopic temporal and spatial measurement of brain activity, Sahin et al. (2009) found robust evidence for distinct neural processing of lexical, morphological, and phonological processing. In distinct, neighboring regions of Broca's area, three individuals revealed the very sequential computational processing hypothesized by models of lexical processing (e.g., Levelt et al. 1991). As the authors state, their findings 'suggest that a linguistic processing predicted on computational grounds is implemented in the brain in fine-grained spatiotemporally patterned activity.'

ERP evidence also supports LMod (as well as BMod). As noted above in section 5.2.1.2, an early ERP component referred to as the ELAN is a component associated with automatic syntactic processing and structure building present in both adults and children. This component, however, has been shown to be absent in G-SLI children. Comparing eighteen G-SLI children and adolescents with language-matched controls, age-matched controls, and normal adults, Fonteneau and van der Lely (2008) found that the ELAN was absent only in the G-SLI group. Moreover, as has been found with agrammatic aphasics (Hagoort et al. 2003), Fonteneau and van der Lely found that their population, impaired in specific syntactic computations, appears to attempt to compensate for their syntactic deficits by using extra-syntactic processing, in this case leading to the utilization of neural circuitry associated with semantic processing.

[19] This is not to suggest that babbling is not part of language development and maturation of the language faculty, a fact that work by Pettito and Marentette (1991) and others has clearly established.

An ever-growing number of fMRI studies evidence distinct neural responses for different components of language. Dapretto and Bookheimer (1999), examining syntactic and semantic processing, reported distinct fMRI activation patterns for each. Indefrey et al. (2001) found lexical and syntactic processing to elicit distinct and separable neural responses. Yamada and Neville (2007) and many others report robust neural processing of syntactic form in the absence of meaning, and therefore a clear separability of the two both neurally and cognitively. Newman et al. (2001) also found both temporally and spatially distinct patterns of activation for semantic vs syntactic acceptability judgments using fMRI and ERP. Moreover, the fMRI patterns temporally mirrored the distinct ERP patterns for the same stimuli—providing corroborating evidence that syntactic and semantic processing depend on distinct neurolinguistic processes and neural substrates. Even within the subsystem of syntax, we find neural reflexes of distinctions that syntactic theory posits. For example, Ben-Shacher et al. (2004) and several others report a consistent, spatially defined neural response to syntactic movement, and Santi and Grodzinsky (2010) demonstrate spatially distinct neural processing of movement vs clausal embedding.

Like the aphasic literature, the imaging literature is at this point too vast to cover here. What is important for the thesis of this chapter is that there are a plethora of studies from laboratories across continents with converging findings that demonstrate clear distinctions between syntactic and semantic processing, including many studies suggesting differential brain circuitry for different classes of syntactic structures (e.g., those involving object extraction vs subject-verb agreement) vs violations as well as different brain activation patterns for phonological processing as opposed to other levels of linguistic processing.

Though too numerous to mention or discuss, the findings of these studies support two fundamental tenets of LMod: (1) the separability of language into submodules, each of which can be differentiated from the others in terms of neural circuitry as well as on linguistic-theoretic grounds, and (2) the further decomposability of the major components of language: phonology, morphology, syntax, or lexicon into even smaller, discrete subsystems.

5.3.7 *Non-linguistic evidence for LMod: the fractionation of other mental domains into submodules*

Almost all of the above discussion has revolved around language as a test case for the viability of the Modularity hypothesis. Yet if the human mind is indeed modular in its make-up, one should find evidence of this regardless of which particular domain one examines. As many have asked, if language is like it is, then what does that indicate the mind must be like more generally? In addressing this question, one finds that language serves as a window into the nature of mind more broadly. When one examines cognitive domains outside of language, one finds that they, too, appear to be modular in composition. And, as with language, this is typically most visible from the purchase of impairment—the atypical case.

One can find instances of quite specific agnosias, for example, agnosias for specific objects or categories of objects (*Apperceptive agnosia* or 'category-specific agnosia'), agnosia for faces (prosopagnosia), for words ('pure word deafness'), or quite specific categories of words, for numbers or for colors (achromatopsia). Moreover, non-linguistic cognitive domains can be fractionated into submodules, most apparent when the system is impaired, with clinical literature providing the necessary evidence that these pieces can be selectively impaired or spared.

The system underlying our body awareness and sense of self (proprioception) is another cognitive domain that can be fractionated, such as with loss of one's sense of body posture, not uncommon in Parkinsons, or failure to recognize one's body parts as one's own (*asomatagnosia*), or the opposite, phantom limbs, in which one experiences as present a body part that is no longer there or was never there (e.g., Melzack 1992; Ramachandran and Blakeslee 1998; Sacks 1987; Shreeve 1993).

Spatial cognition can similarly be fractionated into discrete submodules. Clinical cases of selective impairments of spatial transformation (including selective deficits in processing mirror images or other degrees of mental rotation) and spatial location have been documented (e.g., Bricolo et al. 2000), and neglect of one/half of space (hemi- or unilateral neglect) is not at all uncommon, typically, though not always, following right hemisphere parietal damage.

Selective deficits within the domain of number knowledge and processing have also been documented; in addition to developmental dyscalculia, a selective impairment with the number faculty alongside otherwise normal cognitive function, deficits in procedural dyscalculia and number facts dyscalculia as separate impairments have also been described (e.g., Temple 1991).

Visual cognition can be fractionated as well. Visual closure ('Gestalt Perception'), color agnosia, the ability to apprehend the relation between a part and the whole of which it is a part (e.g., arc to circle), the ability to locate embedded figures within other figures, the ability to recognize particular visual configurations, meaningful or not, the ability to reproduce (copy or construct) particular visual configurations, meaningful or not, and all the various visual agnosias are all part of the mental faculty of visual cognition.

A subcomponent of visual cognition is facial recognition, which in and of itself is decomposable. The system of facial recognition can be impaired broadly (*prosopagnosia*), resulting in extensive dysfunction in the otherwise automatic ability to recognize a face as a face (one form of visual agnosia), and such an impairment can be developmental (as in congenital prosopagnosia) or acquired (typically after right hemisphere damage). However, the cognitive faculty of facial recognition can be fractionated into more discrete pieces, such as the ability to recognize familiar faces, an impairment of which can have serious psychosocial consequences (as in Capgras syndrome). The facial recognition system can also be affected after brain damage in such a way as to lead to abnormal hyperemotional responses to unfamiliar faces, as in Fregoli syndrome (Ramachandran and Blakeslee 1998; Feinberg 2001).

The domain of music cognition, too, can be fractionated into discrete subsystems, each of which can be selectively impaired or spared, both developmentally and as a result of acquired brain damage. Deficits in rhythm, timbre, melody, and harmony have all been reported (e.g., Sacks 2007), and while recent research reveals tantalizing relationships and similarities between music and spoken language, one can be impaired while the other remains intact—amusia without aphasia and aphasia without amusia (e.g., Sacks 2007). Both patterns have been documented frequently in DAT (e.g., Cuddy et al. 2005; Cuddy and Duffin 2005; Piccirilli et al. 2000). Moreover, at least aspects of music cognition can be seen in the brains of newborns (Perani et al. 2010). Similar to brain imaging results for aspects of linguistic processing, Perani et al.'s results indicate that within the first hours of life the infant brain shows (right) hemispheric specialization for music and is differentially sensitive to subcomponents of music, in this case, to differences in consonance and dissonance and to changes in tonal key. As with language, before brain regions responsible for such processing in the adult are at all mature, the neural architecture underlying such processing appears to be hardwired into the species.

5.4 Summary and Conclusions

The primary objective of this chapter has been to take a new look at the question of modularity of mind, bringing to bear evidence from a wide array of sources from which to examine its basic tenets—evidence from studies on the neurology of language, the genetics of language, from cases of atypical development, cases of genetic anomalies, language breakdown, from cognitive dissolution, and from a variety of cognitive domains in addition to language. I am well aware of efforts to debunk this view, the arguments raised toward that end, and the disfavor into which a modular view of the mind has fallen. I believe, however, that when one considers the vast array of relevant evidence, new and old, only a small amount of which I was able to include here, there is strong reason to conclude that language, and in particular, grammar, is a mental faculty that rests on structural organizing principles and constraints not shared in large part by other mental faculties and in its processing and computation is automatic and mandatory. Further, the evidence I have described strongly indicates that language, itself, is comprised of distinct submodules which can be selectively reflected, impaired, or spared, neurologically and cognitively.

A look at domains outside of language further supports the fundamental notion of a modular mind. The various discrete agnosias and fragmentations of other mental systems only briefly mentioned provide potent non-linguistic evidence that the mind is composed of a set of mental faculties, which under normal circumstances intricately interact in a beautiful dance that we recognize as normal human function, or being human, but when examined carefully, can be seen as separable pieces that together comprise the human mind—a modular mind.

6

Every Child an Isolate: Nature's Experiments in Language Learning

LILA GLEITMAN AND BARBARA LANDAU

In this chapter we will concentrate our attention on two specific issues that are implicit in Carol Chomsky's challenging work: to understand how children come to know as much as they do about language and its interpretation onto the world, when the information they receive is paltry. The first concerns the robustness of language acquisition to variability in learners' access to input that would seem crucial to the function being acquired, as dramatized by studies of language in people who became both deaf and blind during infancy. The second concerns the abilities of children to reconstruct the meanings of sentences with covert structure, as in Carol Chomsky's landmark studies of whether blindfolded dolls might be hard to see. These two themes are crucially related, of course, for both exemplify the general problem known as 'the poverty of the stimulus'; in the present case, how humans reconstruct linguistic form and meaning from the blatantly inadequate information offered in their usable environment (cf Plato 380BC; N. Chomsky 1965; J. A. Fodor 1981, inter alia).

6.1 *See* and the Blind Learner

Children ordinarily acquire their native tongue in circumstances where they can listen to speech that refers to the passing scene. To use a famous example, a lucky learner might hear 'Lo! Rabbit!' just as a rabbit hops by. Not only Quine (1960) but serious commentators of every theoretical persuasion are at pains to emphasize that simply alluding to this word–world pairing leaves us light years from the specifics of vocabulary acquisition; indeed exposing the class of problems here is the very purpose of discussing rabbits spied by vexed field linguists (in related regards, see particularly N. Chomsky 1957; Goodman 1951). All the same, it is safe to say that the sensible pairing of sound to circumstance is a crucial precondition for learning, playing a causal role for both vocabulary and syntax acquisition, and most especially at early

References

Bastiaanse, R. and Thompson, C. 2003. Verb and auxiliary movement in agrammatic Broca's aphasia. *Brain and Language*, *87*, 286-305.

Bastiaanse, R. and van Zonneveld, R. 1998. On the relation between verb inflection and verb position in Dutch agrammatic aphasics. *Brain and Language*, *64*, 165-181.

Bates, E., Friederici, A. and Wulfeck, B. 1987. Comprehension in aphasia: A cross-linguistic study. *Brain and Language*, *32*, no. 1, 19-68.

Bates, E., Friederici, A. and Wulfeck, B. 1987. Grammatica morphology in aphasia: Evidence from three languages. *Cortex, 23*, 545-574.

Bates, E., Benigni, L., Bretherton, :I. Camaioni, L. and Volterra, V. 1977. From gesture to the first word: On cognitive and social prerequisites. In M. Lewis and L. Rosenblum (Eds.) *Interaction, Conversation and the development of language*. New York: John Wiley.

Bay, E. 1964. Aphasia and intelligence, *Int J. Neurology, 4* no. 3. 251-64.

Bellugi, U., Poizner, H. and Klima, E. 1989. Language modality and the brain. *Trends in the Neurosciences, 12*, 380-388.

Bender, B., Puck, M., Salbenblatt, J. and Robinson, A. 1986. Dyslexia in 47 XXY boys identified at birth. *Behavior Genetics, 16*, no. 3. 343-354.

Bishop, D.V.M, Bishop, S.J., Bright, P., James, C., Delaney, T. and Tallal, P. 1999. Different origin of auditory and phonological processing problems in children with language impairment: Evidence from a twin study. *Journal of Speech, Language, and Hearing Research*, *42*, 155-168.

Bishop, D.V.M., Carlyon, R.P., Deeks, J.M. and Bishop, S.J. 1999. Auditory temporal processing impairment: Neither necessary nor sufficient for causing language impairment in children. *Journal of Speech, Language, and Hearing Research*, *42*, 1295-1310.

Bishop, D.V.M., North, T. and Donlan, C. 1996. Nonword repetition as a behavioural marker for inherited language impairment: Evidence from a twin study. *Journal of Child Psychology and Psychiatry*, *37*, 391-403.

Boone, K., Swerdloff, R., Miller, B., Geschwind, D., Razani, J., Lee, A., Gaw, I., Gonzalo, I., Haddal, A. and Rankin, K. 2001. Neuropsychological profiles of adults with Klinefelter's syndrome. *Journal of the International Neuropsychological Society*, *7*, no. 4, 446-456.

Bradley, D., Garrett, M.F., and Zurif, E.B. 1980. Syntactic deficits in Broca's aphasia. In D. Caplan (Ed.), *Biological studies of mental processes*, 269-286.

Bricolo, E., Shallice, T., Priftis, K. and Meneghello, F. 2000. Selective space transformation deficit in a patient with spatial agnosia. *Neurocase*, *6*, no. 4, 307-319.

Brown, J. 1977. *Mind, Brain and Consciousness: The Neuropsychology of Cognition*. New York: Academic Press.

Brunellière, A., Franck, J., Ludwig, C. and Frauenfelder, U.H. 2007. Early and automatic syntactic processing of person agreement. *Neuroreport, 18*, no. 6, 537-541.

Buchert, R., Thomasius, R., Wilke, F., Petersen, K., Nebeling, B., Obrocki, J., Schulze, O. and Schmidt, U. 2008. Sustained effects of ecstasy on the human brain: a prospective neuroimaging study in novel users *Brain*, *131*, no. 11, 2936-2945.

Burton, M.W., Small, S., and Blumstein, S.E. 2000. The role of segmentation in phonological processing: An fMRI investigation. *Journal of Cognitive Neuroscience*, *12*, 679-690.

Caramazza, A. 1988. Some aspects of language processing as revealed through the analysis of acquired aphasia: the lexical system. *Annual Review of Neuroscience, 11*, 395-391.

Cholfin, J., Curtiss, S. and Geschwind, D. 2007. A dominantly inherited speech and language disorder not linked to the SPCH1 locus. Revised and resubmitted 2010

Clahsen, S. and Almazon, M. 2001. Compounding and inflection in language impairment: Evidence from Williams Syndrome (and SLI)*. *Lingua, 111*, no. 10, 729-757.

Cohen, M.S. and Bookheimer, S.Y. 1994. Functional magnetic resonance imaging. *Trends in Neurosciences, 17*, no. 7, 268-77.

Corina, D., Poizner, H., Bellugi, U., Feinberg, T., Dowd, D. and O'Grady-Batch, L. 1992. Dissociations between linguistic and nonlinguistic gestural systems: A case for compositionality. *Brain and Language, 43,* 414-447.

Curtiss, S. 2011. Revisiting Modularity. In the 11th Annual Proceedings of the Tokyo Conference on Psycholinguistics.

Curtiss, S. 1995. Language as a cognitive system: Its independence and selective vulnerability. In C. Otero (Ed.), *Noam Chomsky: Critical Assessments: 4*. New York: Routledge.

Curtiss, S. 1988a. The special talent of grammar acquisition. In L. Obler & D. Fein (Eds.), *The Exceptional Brain*. New York: The Guilford Press, 364-386.

Curtiss, S. 1988b. Abnormal language acquisition and grammar: Evidence for the modularity of language. In L. Hyman & C. Li (Eds.), *Language, Speech, and Mind: Studies in Honor of Victoria A. Fromkin*. New York: Routledge, Kegan and Paul, 81-102.

Curtiss, S. 1982. Developmental dissociations of language and cognition. In L. Obler & L. Menn (Eds.), *Exceptional Language and Linguistics*. New York: Academic Press, 285-312.

Curtiss, S. and Yamada, J. 1981. The relationship between language and cognition in a case of Turner's syndrome. *UCLA Working Papers in Cognitive Linguistics, 3*, 93-116.

Dapretto, M. and Bookheimer, S. 1999. Form and content: Dissociating syntax and semantics in sentence comprehension. *Neuron*, *24*, 427-432.

Davis, L., Foldi, N., Gardner, H. and Zurif, E. 1978. Repetition in the transcortical aphasias. *Brain and Language*, *6*, 226-238.

Dehaene-Lambertz, G. 2000. Cerebral specialization for speech and nonspeech stimuli in infants. *Journal of Cognitive Neuroscience*, *12*, no. 3, 449-460.

Dehaene-Lambertz, G. 1997. Electrophysiological correlates of categorical phoneme perception in adults. *NeuroReport*, *8*, no. 4, 919-924.

Dehaene-Lambertz, G and Baillet, S. 1998. A phonological representation in the infant brain. *NeuroReport, 9*, no. 8, 1885-1888.

Dehaene-Lambertz, G., Dehaene, S., Anton, J-L., Campagne, A., Coiciu, P., Dehaene, G. P., Denghian, I., Jobert, A., LeBihan, D., Sigman, M., Palier, C., and Poine, J-B. 2006. Functional segregation of cortical language areas by sentence repetition. *Human Brain Mapping, 27*, 360-371.

Dehaene-Lambertz, G., Dehaene, S. and Hertz-Pennier, L. 2002. Functional neuroimaging of speech perception in infants. *Science, 298,* 2013-2015.

Dehaene-Lambertz, G., Dupoux, E. and Gout, A. 2000. Electrophysiological correlates of phonological processing: A cross-linguistic study. *J. of Cognitive Neuroscience, 12,* no. 4, 635-647.

Dehaene-Lambertz, G. and Gliga, T. 2004. Common neural basis for phoneme processing in infants and adults. *Journal of Cognitive science, 16*, no. 8, 1375-1387.

Dehaene-Lambertz, G., Hertz-Pennier, L. and Dubois, J. 2006. Nature and nurture in language acquisition: Anatomical and functional brain-imaging studies in infants. *Trends in Neuroscience, 29,* no. 7, 367-373.

Dehaene-Lambertz, G , Hertz-Pennier, L , Dubois, J, Mériaux, S., Roche, A., Sigman, M. and Dehaene, S. 2006. Functional organization of perisylvian activation during presentation of sentences in preverbal infants. *PNAS, 103*, no. 38, 14240-14245.

Dehaene-Lambertz, G., Pallier, C., Serniclaes, W., Sprenger-Chharole, L., Jobert, A. and Dehaene, S. 2005. Neural correlates of switching from auditory to speech perception. *Neuroimage, 24*, no. 1, 21-33.

Dehaene-Lambertz, G,. Pena, M., Christophe, A. and Landrieu, P. 2004. Phenome perception in a neonate with a left sylvian infarct. *Brain and Language*, *88*, 26-38.

Dehaene-Lambertz, G. and Pena, M. 2001. Electrophysiological evidence for automatic phonetic processing in neonates. *NeuroReport, 12*, no. 14, 3155-3158.

Emmorey, K. 2002. *Language, Cognition and the Brain: Insights from sign language research*. Mahwah: Lawrence Erlbaum Associates.

Feinberg, T. 2001. *Altered Egos: How the Brain Creates the Self*. Oxford: Oxford University Press.

Felsenfeld, S. and Plomin, R. 1997. Epidemiological and offspring analyses of developmental speech disorders using data from the Colorado adoption project. *Journal of Speech, Language, and Hearing Research, 40*, 778-791.

Fonteneau, E., and van der Lely, H.K.J. 2008. Electrical brain responses in language-impaired children reveal grammar-specific deficits. *PLOS ONE, 3,* no. 3, 1-6.

Friedmann, N., Gvion, A., Biran, M. and Novogrodsky, R. 2006. Do people with agrammatic apahasia understand verb movement? *Aphasiology*, *20*, 136-153.

Friedmann, N., Gvion. A. and Novogrodsky, R. 2006. Syntactic movement in agrammatism and S-SLI: Two different impairments. In A. Belletti, E. Bennati, C. Chesi, E. Di Domenico & I. Ferrari (Eds.), *Language Acquisition and Development*. Newcastle, UK: Cambridge Scholars Press/CSP, 197-210.

Friedmann, N. and Novogrodsky, R. 2008. Subtypes of SLI: SySLI, PhoSLI, LeSLI, and PraSLI. In A. Gavarró, & M. João Freitas (Eds.), *Language Acquisition and Development*, 205-217.

Friedmann. N. and Novogrodsky, R. 2007. Is the movement deficit in syntactic SLI related to traces or to thematic role transfer? *Brain and Language, 101*, no. 1, 50-63.

Golston, C. 1991. *Both Lexicons*. Unpublished Ph.D. dissertation. UCLA.

Gopnik, M., Dalalakis, J., Fukuda, S. and Hough-Eyamic, W. 1997. The biological basis of language: Familial language impairment. In M. Gopnik (Ed.), *The Inheritance and Innateness of Grammars*. New York: Oxford University Press, 111-140.

Grodzinsky, Y. 1986. Language deficits and the theory of syntax. *Brain and Language*, *27*, 135-159.

Grodzinsky, Y. and Finkel, L. 1998. The neurology of empty categories. *Journal of Cognitive Neuroscience, 10*, no. 2, 281-292.

Hagiwara, H. 1995. The breakdown of functional categories and the economy of derivation. *Brain and Language. 50*, 92-116.

Hagoort, P., Wassenaar, M.E.D. and Brown, C.M. 2003. Syntax-related ERP-effects in Dutch. *Cognitive Brain Research*, *16*, no. 1, 38-50.

Hahne, A., Eckstein, K. and Friederici, A.D. 2004. Brain signatures of syntactic and semantic processes during children's language development. *Journal of Cognitive Neuroscience*, *16*, 1302-1318.

Hahne, A. and Friederici, A.D. 1999. Electrophysiological evidence for two steps in syntactic analysis: Early automatic and late controlled processes. *Journal of Cognitive Neuroscience, 11*, 194-205.

Hart, J., Berndt, R.S., Caramazza, A. 1985. Category-specific naming deficit following cerebral infarction. *Nature, 316*, 439-440.

Hohnen, B. and Stevenson, J. 1999. The structure of genetic influences on general cognitive, language, phonological, and reading abilities. *Developmental Psychology*, *35,* no. 2, 590-603.

Indefrey et al, 1998. *NeuroImage*...

Itti, E., Gaw, I., Gonzalo, I., Pawlikowska-Haddal, A., Boone, K.B., Mlikotic, A., Itti, L., Mishkin, F.S. and Swerdloff, R.S. 2006. The structural brain correlates of cognitive deficits in adults with Klinefelter's syndrome. *Journal of Clinical Endocrinology and Metabolism, 91*, no. 4, 1423-1427.

Jackson, C. 1984. Language acquisition in two modalities: Person deixis and negation in ASL and English. Unpublished Masters Thesis, UCLA.

Jacquemot, C., Pallier, C., LeBihan, D., Dehaene, S. and Dupoux, E. 2003. Phonological grammar shapes the auditory cortex: A functional magnetic resonance imaging study. *Journal of Neuroscience, 23*, 9541-9546.

Jodzio, K., Biechowska, D. and Leszniewska-Jodzio, B. 2008. Selectivity of lexical-semantic disorders in Polish-speaking patients with aphasia: Evidence from single-word comprehension. *Archives of Clinical Neuropsychology, 23*, no. 5, 543-551.

Jones, N. 2007. The Use of Deictic and Cohesive Markers in Narratives by Children with Williams Syndrome. Unpublished Ph.D. dissertation, UCLA.

Kean, M-L. 1980. Grammatical representations and the description of language processes. In D. Caplan (Ed.), *Biological Studies of Mental Processes*. Cambridge, MA: The MIT Press.

Kempler, D. 1984. Syntactic and symbolic abilities in Alzheimer's disease. Unpublished Ph.D. dissertation, UCLA.

Kempler, D., Curtiss, S. and Jackson, C. 1987. Syntactic preservation in Alzheimer's disease. *Journal of Speech and Hearing Research, 30*, 343-350.

Kessels, R., Hendriks, M., Schouten, J., van Asselen, M. and Postma, A. 2004. Spatial memory deficits in patients after unilateral selective amygdalohippocampectomy . *Journal of the International Neuropsychological Society, 10*, no. 6, 907-912.

Levelt, W. J. M., Schriefers, H., Vorberg, D., Meyer, A. S., Pechmann, T., and Havinga, J. 1991. The time course of lexical access in speech production: A study of picture naming. *Psychological Review*, *98*, 122-142.

Levy, Y. and Schaeffer, J. (Eds.). 2003. *Towards a Definition of Specific Language Impairment*. Mahwah: Lawrence Earlbaum Associates.

Lewis, B. 1992. Pedigree analysis of children with phonology disorders. *Journal of Learning Disabilities, 25*, no. 9, 586-597.

McGue, M. and Broen, P. 1995. Familial aggregation of phonological disorders: Results from a 28-year follow-up. *Journal of Speech and Hearing Research, 38*, 1091-1107.

McGuire, P.K., Robertson, D., Thacker, A., David, A.S., Kitson, N., Frackowiak, R.S.J. and Frith, C.D. 1997. Neural correlates of thinking in sign language. *NeuroReport, 8*, no. 3, 695-698.

Money, J. 1973. Turner's syndrome and parietal function. *Cortex, 9*, 385-393.

Money, J. and Alexander, D. 1966. Turner's syndrome: Further demonstrations of the presence of specific cognitional deficiencies. *Journal of Medical Genetics, 3*, 223-231.

Morgan, G., Herman, R. and Woll, B. 2007. Language impairment in sign language: breakthroughs and puzzles. *International Journal of Language and Communication Disorders*, *42*, no. 1, 97-105.

Naatanen, R., Lehtokovski, A., Lennes, M., Cheour, M., Huotilainen, M. and Ivonen, A. 1997. Language-specific phoneme representations revealed by electric and magnetic brain responses. *Nature, 385*, 432-434.

Nelson, H.D., Ngyren, P., Walker, M. and Panoscha, R. 2006. Screening for speech and language delay in preschool children: Systematic evidence review for the US Preventive Services Task Force. *Pediatrics*, *117*, no. 2, 298-319.

Newman, A.J., Pancheva, R., Ozawa, K., Neville, H.J. and Ullman, M.T. 2001. An event-related fMRI study of syntactic and semantic violations. *Journal of Psycholinguistic Research, 30*, no. 3, 339-364.

Netley, C. and Rovet, J. 1982. Verbal deficits in children with 47 XXY and 47 XXX karyotypes: A descriptive and experimental study. *Brain and Language, 2*, 10–18.

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

Oberecker, R., Friedrich, M. and Friederici, A. 2005. Neural correlates of syntactic processing in two-year-olds. *Journal of Cognitive Science, 17*, no. 10, 1667-1678.

Pena, M., Maki, A., Kovacic, D., Dehaene-Lambertz, G, Koizumi, H., Bouquet, F. and Mehler, J. 2003. Sounds and silence: An optical topography study of language recognition at birth. *PNAS, 100*, no. 20, 11702-11705.

Pennington, B., Heaton, R., Karzmrak, M., Pennington, M., Lehman, R. and Shucard, D. 1985. The neuropsychological phenotype in Turner's syndrome. *Cortex, 21*, 391-404.

Petitto, L.A. 1987. On the autonomy of language and gesture: Evidence from the acquisition of personal pronouns in ASL. *Cognition, 27*, 1-52.

Pettito, L.A., Zatorre, R., Gauna, K., Nikelski, E.J., Dostie, D. and Evans, A. 2000. Speech-like cerebral activity in profoundly deaf people while processing signed languages: Implications for the neural basis of human language. *Proceedings of the National Academy of Science*, *97*, no. 25, 13961-13966.

Phillips, C. 2001. Levels of representation in the electrophysiology of speech perception. *Cognitive Science, 25*, 711-731.

Phillips, C., Kazanina, N. and Abada, S. 2005. ERP effects of the processing of syntactic long-distance dependencies. *Cognitive Brain Research, 22*, 407-428.

Phillips, C., Pellathy, T., Marantz, A., Yellin, E., Wexler, K., Poeppel, D., McGinnis, M. and Roberts, T. 2000. Auditory Cortex Accesses Phonological Categories: An MEG mismatch study. *Journal of Cognitive Neuroscience, 12*, 1036-1055.

Pulvermüller, F., Shtyrov, Y., Hastings, A. and Carlyon, R. 2008. Syntax as a reflex: Neurophysiological evidence for early automaticity of grammatical processing. *Brain and Language*, *104*, no. 3, 244-253.

Pulvermüller, F. and Shtyrov, Y. 2003. Automatic processing of grammar in the human brain as revealed by the mismatch. *Neuroimage, 20*, no. 1, 159-172.

Ramachandran, V.S. and Blakeslee, S. 1998. *Phantoms in the brain: Probing the mysteries of the human mind*. New York: Quill William Morrow.

Roland, P. K. and Zilles, R. 1998. Structural divisions and functional fields in the human cerebral cortex. *Brain Research Reviews*, *26*, nos. 2-3, 87-105.

Rondal, J. 1995. *Exceptional Language Development in Down Syndrome*. Cambridge: Cambridge University Press.

Rosen,S., Adlard,A. and van der Lely,H.K.J. 2009. Backward and simultaneous masking in children with grammatical specific language impairment: no simple link between auditory and language abilities. *Journal of Speech, Language, and Hearing Research*, *52,* no. 2, 396-411.

Rovet, J. 1998. Turner's syndrome. In B.P. Rourke (Ed.), *Syndrome of Nonverbal Learning Disabilities: Neurodevelopmental Manifestations*. New York: The Guilford Press, 351-371.

Rovet, J. and Ireland, L. 1994. The behavioral phenotype in children with Turner's syndrome. *Journal of Learning Disabilities, 26*, 333-341.

Rovet, J., Netley, C., Keenan, M., Bailey, J. and Stewart, D. 1996. The psychoeducational profile of boys with Klinefelter Syndrome. *Journal of Learning Disabilities, 29*, no. 2, 180-196.

Sacks, O. 2007. *Musicophilia.* New York: Albert Knopf.

Sahin, N., Pinker, S., Cash, S., Schomer, C. and Holgren, E. 2009. Sequential processing of lexical, grammatical, and phonological information within Broca's area. *Science, 326*, no. 5951, 445-449.

Sakai, K., Noguchi, Y., Takeuchi, T. and Watanabe, E. 2002. Selective priming of syntactic processing by event-related transcortical magnetic stimulation of Broca's area. *Neuron, 35*, 1177-1182.

Schaller, S. 1995. *A Man Without Words.* Berkeley: University of California Press.

Schmitt, B.M., Schiltz, K., Zaake, W., Kutas, M. and Münte, T.F. 2001. An electrophysiological analysis of the time course of conceptual and syntactic encoding during tacit picture naming. *Journal of Cognitive Neuroscience, 13*, no. 4, 510-522.

Silbert, A., Wolff, P., and Lillienthal, J. 1977. Spatial and Temporal processing in patients with Turner's syndrome. *Behavior Genetics, 7*. 11-21.

Smith, N.V. and Tsimpli, I.M. 1995. *The Mind of a Savant: Language Learning and Modularity*. Oxford: Blackwell, Inc.

Stromswold, K. 2007. A gene linked to speech and language, in developing human brain. *American Journal of Human Genetics*, *87*, 1144-1157.

Stromswold, K. 2006. Biological and psychosocial factors affect linguistic and cognitive development differently: A twin study. *Proceedings of the Thirtieth Annual Boston University Conference on Language Development*, *2*, Somerville, MA: Cascidilla Press, 595-606.

Stromswold, K. 2001. The heritability of language: A review and meta-analysis of twin, adoption and linkage studies. *Language, 77*, no. 4, 647-723.

Tallal, P. 1976. Rapid auditory processing in normal and disordered language development. *Journal of Speech and Hearing Research, 19,* 561-571.

Tallal, P. 2000. Experimental studies of language learning impairments: From research to remediation. In D.V.M. Bishop and L.B. Leonard (Eds.), *Speech and Language Impairments in Children: Causes, Characteristics, Intervention and Outcome*. Hove, UK: Psychology Press, 131-155.

Tammel, D. 2007. *Born on a Blue Day: Inside the Extraordinary Mind of an Autistic Savant*. Free Press. (available: http://www.google.com/search?client=firefox-a&rls=org.mozilla%3Aen-US%3Aofficial&channel=s&hl=en&source=hp&q=Daniel+Tammet%2C+%E2%80%9CBorn+on+a+Blue+Day%3A+Inside+the+Extraordinary+Mind+of+an+Autistic+Savant%E2%80%9D+%28Free+Press%29&btnG).

Temple, C. 1991. Procedural dyscalculia and number facts dyscalculia: Double dissocation in developmental dyscalculia. *Cognitive Neuropsychology, 8*, 155-176.

Temple, C and Carney, R. 1996. Reading skills in children with Turner's syndrome: An analysis of hyperlexia. *Cortex, 32*, no. 2, 335-345.

Thompson, C., Fixa, S. and Gitelman, D. 2002. Selective impairment of morphosyntactic production in a neurological patient. *Journal of Neurolinguistics, 15*, nos. 3-5, 189-207.

Tomblin, J. and Buckwalter, P. 1998. Heritability of poor language achievement among twins. *Journal of Speech and Hearing Research, 41*, 188-199.

van der Lely, H.K.J. 2005. Domain-specific cognitive systems: Insight from grammatical specific language impairment. *Trends in Cognitive Sciences, 9*, no. 2, 53-59.

van der Lely, H.K.J. 2005. Grammatical-SLI and the computational grammatical complexity hypothesis. *Revue Frequences, 17*, no. 3, 13-20.

van der Lely, H.K.J. 2004. Evidence for and implications of a domain-specific grammatical deficit. In L. Jenkins (Ed.) *Variations and Universals in Biolinguistics*. Oxford: Elsevier, 117-145.

van der Lely, H.J.K. and Battell, J. 2003. Wh-movement in children with grammatical SLI: A test of the RDDR hypothesis. *Language*, *79*, 153-181.

van der Lely, H.K.J. and Maranis, T. 2007. On-line processing of wh-questions in children with G-SLI and typically developing children. *International Journal of Language and Communication Disorders, 42*, no. 5, 557-582.

van der Lely, H.K.J., Rosen, S. and Adlard, A. 2004. Grammatical language impairment and the specificity of cognitive domains: Relations between auditory and language abilities. *Cognition, 94*, no. 2, 167-183.

van der Lely, H.K.J., Rosen, S., McClelland, A. 1998. Evidence for a grammar specific deficit in children. *Current Biology*, *8*, 1253-1258.

van der Lely, H. and Stollwerck, L. 1996. A grammatical specific language impairment in children: An autosomal dominant inheritance? *Brain and Language, 52*, 484-504.

Varley, R. and Siegal, M. 2000. Evidence for cognition without grammar from causal reasoning and theory of mind in an agrammatic patient. *Current Biology*, *10*, no. 12, 723-726.

Waber, D. 1979. Neuropsychological aspects of Turner's syndrome. *Developmental Medicine and Child Neurology, 21*, 58-70.

Whitaker, H. 1976. A case of the isolation of the language function. In H. Whitaker and H.A. Whitaker (Eds.), *Studies in Neurolinguistics, Volume 2*, 1-58.

Zatorre, R.J., Evans, A.C., Meyer, E. and Gjedde, A. 1992. Lateralization of phonetic and pitch processing in speech perception. *Science*, *256*, 846-849.