The Competition Model and Language Disorders

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Introduction

To understand language disorders, we need to contrast disordered language processing with normal language processing. In one sense, this is easy to do. We see that people with language disorders struggle with articulation, lexical access, syntactic structure, comprehension, and other language functions. But simply observing these behavioral differences is not enough. To understand the dynamics of communication disorders we need to articulate a processing model that explains in mechanistic terms how and why disordered processing differs from normal processing. Moreover, because communication abilities continue to develop throughout the lifespan, the model must also consider properties of language acquisition.

The Competition Model {MacWhinney, 2008 #10341} addresses this challenge by providing a functionalist account of how languages are learned across the lifespan and how they are processed in real time. Three decades of research based on this model have shed light on aspects of first language learning, second language learning, bilingual processing, developmental language disorders, and language loss in aphasia.

The fundamental claim of the model is that alternative interpretations compete online during language processing. To probe these various competitions, researchers

have used multifactorial experimental designs to measure the process of cue competition. As summarized in MacWhinney {, 2008 #10341} and elsewhere, the predictions of the model have been uniformly supported across three decades of research involving 15 different languages {MacWhinney, 1989 #5822}. In this chapter, we will focus on the ways in which this model can help explain behavioral and neural patterns of language disorders.

Readers of this volume are well aware of the many challenges involved in studying language disorders. Perhaps the biggest challenge is that that these disorders come in so many alternative forms. Given the complexity of language, there are good reasons to expect that the patterns of language disorders should be at least as diagnostically complex as disorders of any other sophisticated biological system (such as the immune or the circulatory system). We know that the human brain is an extremely complex object {Buzsaki, 2006 #10635} and that no two human brains are totally alike. Rather, our brains differ substantially in terms of sizes, connectivity, and microstructure. Language is a complex and distributed process overlaid on this individually variable and complex system. Although language is a species-specific ability, the detailed shape of that ability involves many mutations across millions of years that have promoted a gradual and continual growth in communication skills {MacWhinney, 2008 #10355}. These changes have impacted dozens of traits relating to the size of the brain, patterns of neural connectivity, styles of neural processing, gestural expression, and the structure of the vocal apparatus.

Given this immense complexity, we might wonder how one could even begin to understand language disorders. Fortunately, language itself provides two powerful

searchlights for our exploration. The first is that language use is grounded on social convention. This means that, no matter how variable our brains, we all learn to use the same socially shared system for communicating meaning. To illustrate this, Witttgenstein {, 1953 #4435} compared language use to the structure of hedges in a formal garden. Viewed from the inside, each hedge has its own idiosyncratic branching structure. Viewed from the outside, all of the hedges have the same straight edges. Social convention serves as the metaphorical gardener, making sure that each of us uses language in accordance with tightly specified patterns. No matter how individualistic our intentions or divergent our thought patterns, we all must end up conforming to the same grammatical rules. These core linguistic patterns are called "cues" in the Competition Model, and studies in that framework show that cues are acquired bit by bit during childhood. However, by adulthood, normal native speakers have acquired and coordinated all the relevant cues, weighting each cue by its normative strength. In this way, ensuring a common communicative system, normally developing speakers end up with figuratively nice, straight hedges.

There is also a second way language properties facilitate our exploration of language disorders. By its very nature, language rests on at least six separable data-processing systems: audition, articulation, lexicon, syntax, perspective switching, and mental model construction. A modular view of language {Pinker, 1997 #10385;Fodor, 1983 #1330} views these systems, and others, as executing in isolation and as represented in discrete local neuronal regions. This model suggests that we might expect to find easily distinguishable neural patterns in patients with language disorders.

In contrast, the Competition Model views these separable systems as interconnected and interactive {McClelland, 1989 #7102}. From the viewpoint of the Competition Model, developmental language disorders should arise primarily from disturbances in the connections between these partially separable systems, rather than damage or malformation of particular areas. At the same time, we must recognize that some disorders, particularly in the aphasias, can arise from malformations, including lesions, within specific brain areas.

Why use a processing model?

Some have argued that specific linguistic deficits are the basis for language disorders {Rice, 1996 #10652}. In some of these accounts, deficits are associated with specific brain areas that are damaged in aphasia {Caplan, 1992 #6052} . In other accounts, the deficit is linked to some specific mutation that is thought to impact language functioning {van der Lely, 1996 #7735}. However, analyses have often oversimplified the actual patterns of disruptions in the linguistic system. Language is a complex process, consisting of many component skills. Semantic, lexical, phonological, and syntactic information must be processed on-line to produce or comprehend language. Viewing language as a dynamic interaction between many local brain areas is consistent with the available neuroanatomical and behavioral data {Bookheimer, 2002 #10367}. One classic contrast emphasizes the role of Wernicke's area for lexical processing and the role of Broca's area for syntactic processing. Work in neuroimaging {Booth, 2001 #7987;Just, 1996 #7660} has supported aspects of this analysis. This work has shown that there is language task differentiation in neural tissue.

Why, then, would we suggest that research go beyond mapping particular competencies to specific disorders (such as SLI) or anatomical injuries (as in aphasia)? First, evidence suggests that even specific damage can exert broad and varying effects on language functioning {Bates, 1991 #4977}. The reverse is also true; similar symptoms in language production and comprehension can be elicited from different types of damage {Bates, 1991 #4977}. More fundamentally, it is a mistake to think that local areas operate in a simple and uniform way when involved with other areas online. Neuroimaging with fMRI can underestimate the dynamic real-time flexibility and complexity of the system. A processing approach, on the other hand, emphasizes the potential for system-wide deficits to stem from varying or multiple causes. Moreover, as Wittgenstein's analysis suggests, the specific symptoms of a language disorder will depend on how the linguistic processing network is configured. For example, because different languages are represented differently, we would expect noticeably different patterns of impairment in different languages. These observations underline the importance of a processing approach to communicative disorders.

An Introduction to the Competition Model

The Competition Model provides a processing account for both comprehension and production. In order to map form to meaning during comprehension, or meaning to form in production, a language user must use a set of cues specific to that language. Each of these cues has a certain *validity*, or general usefulness of a cue in the input. More specifically, we can think of cue validity in terms of the dimensions of *reliability* and *availability*. The availability of a cue is the degree to which it is accessible in the input.

The reliability of a cue is the probability with which a cue leads to correct usage or understanding.

The original framing of the Competition Model {Bates, 1982 #228} relied exclusively on the concepts of reliability and availability. At that time, we viewed competition in terms of its final results, such as sentence role interpretation, often revealed in decisions made after subjects had finished hearing whole sentences. However, once experimenters began making online measurements of processing {MacWhinney, 1988 #3335;Kempe, 1998 #7912}, it became clear that additional dimensions needed to be included in the model. This additional variance was described in terms of *cue cost*, a measure of the processing effort needed to make use of that cue during comprehension or production. Among the factors affecting cue cost, the most notable is *working memory load* {King, 1991 #5462;Gupta, 1997 #6908}. Other factors include *detectability* and *systematicity*. For cues to compete, they must be maintained together for the short term, necessitating some working memory or attentional focus mechanism. This integration is important because it plays a large role in predicting the effects of disorders in language.

Over the years, as the model has been extended to an increasingly wider range of phenomena in both first and second language learning, it has been necessary to add additional processing dimensions. The current version of the model, called the Unified Competition Model, provides a singular account of both first and second language learning. The Unified Model retains competition as the core mechanism by which form and meaning are mapped in comprehension and production. The model is described in

terms of seven additional dimensions of cognitive processing that modulate this core process of competition, as illustrated in Figure 1.

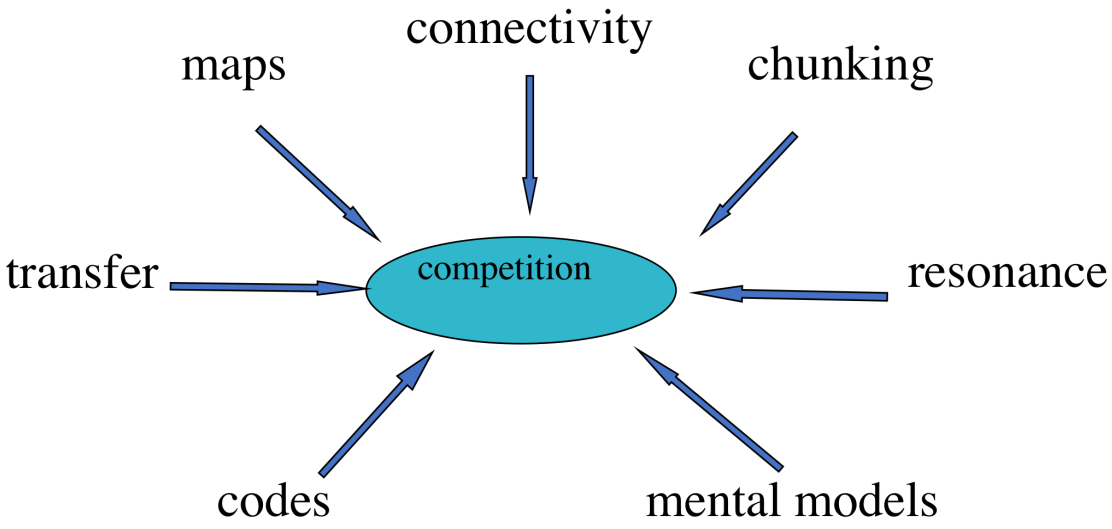

Figure 1. Unified Competition Model (MacWhinney, 2008a for further details).

In the Unified Model, competition arises between alternatives within specific cortical *maps*. The relevant maps represent cortical areas that encode patterns, across six areas language processing: audition, articulation, lexicon, syntax, perspective taking, and mental models. These maps are broadly tied to specific brain areas in the model: auditory cortex for audition, motor cortex for articulation, Wernicke's area for lexicon, Broca's area for syntax, dorsolateral prefrontal cortex for perspective taking, and more dorsal areas for mental models. In addition, morphological usage coordinates processing in posterior lexical areas and anterior syntactic areas. In lexical representation, there is a further distinction between localized phonological representations and distributed semantic representations. For details regarding the emergence and connectivity of lexical maps please consult Li, Zhao, & MacWhinney {, 2007 #10203}.

The strength of a given competitor within a map is determined by its resting strength and the additional activation it receives from other items from *connectivity* both within and between brain areas. For example, the competition between the two readings of *port* in (1) and (2) can be resolved by connections to other lexical items (*captured* and *drank*). However, the competition between the two readings of *raced* in (3) and (4) is determined by syntactic patterns that extend beyond single lexical items.

1. The sailors captured the port at night.

2. The sailors drank the port at night.

3. The horse raced past the barn.

4. The horse raced past the barn fell.

The dimension of *connectivity* likewise influences the ways in which local areas communicate with each other. For example, the lexical cortex in Wernicke's area must be connected in some way to the motor cortex that produces articulatory output. The study of speech errors has shown that the lexicon maintains a function that assures that output forms are at least whole words. To do this, there must be some reciprocal connection between motor and lexical brain areas.

Within local maps, items that occur together frequently can become unified into *chunks*. For example, the phrase *as they say* functions as a single lexical item that is functionally equivalent to *reportedly*. Other chunks operate on the phonological level. For example, in Japanese, there are only 70 possible syllables and each of these, such as *na*, *ko*, or *ku* operate as single chunks. In syntax, chunks have a more flexible structure.

For example, as phrase such as *what I really wanted to say was* can be processed as the same syntactic chunk that would produce *what I really meant to say was.*

The patterns of connection between local areas are further influenced by *resonance* in neural activation. During processing, attentional areas in the frontal cortex {Botvinick, 2001 #9544} maintain resonant activation in the local maps. This type of online resonance is particularly relevant to the study of language disorders, because it implements both the process of working memory and that of gating, which are necessary to language models. Working memory operates by maintaining a pattern of activation in an area or across areas. Gating operates in speech production to allow a candidate pattern in the output buffer to actually be produced {Levelt, 1989 #4614}. Both of these aspects of resonance rely heavily on accurate timing of gating during production and preservation of material while it is still needed for sentence processing. If this connectivity is poorly wired or if resonance is inaccurate, errors in timing result, and the whole complex process of language production can fall apart.

Resonance also operates during language learning to consolidate new forms and chunks in memory. For example, when we read a new word, we represent that new word in terms of resonance between sound, meaning, and orthography. The hippocampus and other subcortical areas provide temporary support for these resonant connections {Wittenberg, 2002 #10207}. The smooth functioning of resonance involves precise activation between corresponding linked areas. For example, a given lexical item in auditory cortex is linked to a corresponding representation in articulatory/motor cortex. This mapping between the two areas retains the fundamental property of resonance: auditory features must be mapped in a traceable way onto articulatory features. Another

pathway of resonant connection links lexicon and syntax. Words that occupy a certain position in the lexical map also operate in similar ways in the syntactic map, and connectivity between the two areas is necessary. If these patterns of connection between areas are jumbled or disordered, it will be difficult to achieve smooth control of this type of resonance.

The Unified Model includes three additional dimensions. Two of these dimensions –transfer and code selection – are primarily important for the study of bilingualism and second language learning. The final dimension, mental model construction, is relevant primarily for those speakers with conceptual communication disorders {Craig, 1993 #5836}. Mental model construction includes skills such as perspective shifting, theory of mind, imagery, and narrative construction. Problems with mental model construction have often been implicated in disorders such as autism {Pelphrey, 2005 #10250}, schizophrenia {Rochester, 1979 #3492}, and Williams Syndrome {Karmiloff-Smith, 1997 #7899}.

Predictions of the Model

Having reviewed the seven dimensions that control competitive processing in the Unified Model, we can now return to the original question. Specifically, how can a processing model add to the understanding of language disorders and treatment of patients diagnosed with those disorders? There are two main possibilities. First, understanding the greater system-wide features of these disorders can increase our ability to predict patient behavior. Second, this increased predictive power can influence how treatment can be designed and evaluated.

Research in communication disorders addresses three main theoretical questions: 1) To what degree can we characterize impairments as localized vs. global? 2) Are the problems encountered by patients exclusively linguistic, or are domain-general cognitive processes also affected? 3) How much of language is "hard-wired"; that is, genetically specified and available independent of experience and learning? Let us examine how the Unified Model addresses each of these questions.

Localized vs. Global Impairment

Reacting against his failure to locate the *engrams* of memory, Karl Lashley {, 1951 #2455} proposed that all cognitive functioning is global. However, given what we now know about the details of neural connectivity {Van Essen, 1990 #10343;Schmahmann, 2007 #10636}, it is difficult to deny that different neuronal areas have different functions. However, this functional differentiation does not invalidate the concept of global cognition. In the case of language impairments, we can think of language processing as an acrobat who is simultaneously juggling across seven separate dimensions. At any given moment, there is a contribution from attentional areas, lexical processing, links from lexicon to syntax, and often elaboration of a mental model. If processing in any one of these coordinated areas suddenly "crashes" or breaks down, then the larger process is disrupted. In the case of normal speakers, the juggler is so skillful that this seldom happens, and when it does, there is a quick recovery. In a speaker with impairments, problems in any area can impact the whole system. Because of this, the Unified Model places an emphasis on overall patterns of cognitive *cost* or cognitive *load*. If stress to the system causes failure primarily in a highly "vulnerable" or costly area of language, then within a language, there should be a common tendency *across disorders*

for similar structures and processes to be harmed. That is, aphasics and SLI patients should be similar in *which* elements of language are impaired, either in comprehension or production. Moreover, a similar pattern of these impairments (albeit to a lesser degree) might be produced with normal language users under cognitive load. We will see that findings from Broca's and Wernicke's aphasics, as well as SLI patients, show support for this Competition Model prediction.

An important piece of evidence for the systemic properties of language disorders comes from non-disordered individuals under cognitive load. First, we know that marked increases in cognitive load can impair normal comprehension {Just, 1992 #5180}. Moreover, varying the type and quality of cognitive load creates a performance profile in normal college students that closely resembles the one found in aphasics {Dick, 2001 #9700}. Because we know there is no *systematic* physiological or genetic damage to the language system in these control participants, results like these support a model of language as a broad, complex, resource-intensive system that depends on smooth coordination between diverse local resources.

Moreover, aphasics and SLI patients have similar deficits in the specific elements of language that are impaired, both in comprehension or production. Most importantly, this prediction shifts the emphasis in language disorders from specific competency deficits (e.g., inflectional morphology in Broca's aphasics) and moves it to the *cause* of those deficits (i.e., less reliable, less frequent, more costly parts of the system, such as inflectional morphology, are more vulnerable to deficits across the board). The resemblance between the areas of language affected under cognitive load and those affected in SLI is a good example of the benefits of a processing model in general.

The Competition Model does not suggest there are no differences among different disorders. On the contrary, these dissociations are very informative in understanding neural specialization and other properties of language. However, a model in which injuries to the language system create a system-wide decrement in performance that disproportionally affects the "weakest" parts of the system predicts some commonalities among different disorders. By examining some disorder-specific predictions, we further show the advantage of a Competition Model approach.

Specific Language Impairment

Specific language impairment is a disorder that is defined by normal cognitive function combined with outlying poor performance on language tasks. As such, the disorder is a logical testing ground for hypotheses about the domain-generality of language as well as the genetic origins vs. learning basis of grammatical competency.

Specific Language Impairment and FOXP2

Some researchers have argued that SLI is a genetic disorder resulting in a phenotypically unified competency deficit. For example, the model of Extended Optional Infinitive {Rice, 1996 #10652} proposes that SLI is centrally a failure to develop verb agreement, delaying competent syntactic production. Similarly, the G-SLI model {Van der Lely, 2005 #10655} proposes that there is at least some subgroup of SLI patients whose essential deficit is in grammatical processing; more specifically, there must be impairment in grammar but not in word learning, phonology, or working memory. However, we suggest that full application of these models requires logical steps that lack strong biological evidence and general plausibility.

The strongest phrasing of these arguments includes each of three main tenets: 1) the cause of SLI is genetic in origin, 2) the deficits seen in SLI are fundamentally domain-specific, and 3) SLI has a common set of diagnostic criteria that are based on linguistic competency, and these criteria mark the fundamental difference between SLI and comparison individuals. Let us examine each of these tenets.

1. The cause of SLI is genetic in origin.

In order to characterize SLI specifically as a disorder with a single genetic cause, several inferences are needed. First, the argument requires an identifiable genetic source of the disorder. For example, in the KE family {Marcus, 2001 #10658}, the prevalence of language disorder mapped onto a predicted pattern of inheritance for a mutation in a dominant gene. This family seems to illustrate a pattern in which a language deficit could be linked to a known mechanism of action for the gene base pairs in question, which is the model of a mutation disorder, such as sickle cell anemia. Further research in SLI proposed the FOXP2 gene as a likely location for such a specific mutation.

What is known about FOXP2, however, does not lend itself to such an easy explanation. First, the fact that the gene exists in large concordance across species {Enard, 2002 #10311} makes it is necessary to differentiate what part of the FOXP2 gene is uniquely human. Second, a large-scale study of 270 4-year-old language-impaired children from a general population sample of 18,000 children {Meaburn, 2002 #10650} did not find the hypothesized FOXP2 mutation in any participants. Therefore, there must be some alternative etiology that leads to language impairment, beyond a simple mutation in FOXP2.

Moreover, mutations of FOXP2 in patients are also associated with small-scale orofacial motor control. Thus, behavioral deficits in these individuals extend beyond functional language processing to motor control (including motor control that is necessary for speech). Vargha-Khadem and colleagues {, 1995 #10656} note that the disorder in affected members of the KE family "indicates that the inherited disorder does not affect morphosyntax exclusively, or even primarily; rather, it affects intellectual, linguistic, and orofacial praxic functions generally" (p. 930). Given the complex range of deficits, it is unclear how a mutation in this area could yield a phenotypically unified disorder such as that proposed by van der Lely et al. {, 1998 #10654}.

This is not to say that such specific and mutation-based disorders are impossible; indeed, sickle cell anemia is a clear case of a disorder that is both phenotypically identifiable and genetic in origin. However, this example makes it clear that the level of specificity involved in describing such a disorder is much higher than that currently used in SLI. Genetic specification of the 'one gene, one mutation' variety is unlikely. A more complex model, involving interactions between genetic factors, seems more probable. Recently, Vernes et al. {Vernes, 2008 #10660} traced the down-regulation of FOXP2 on CNTNAP2, a gene that encodes a neurexin that influences cortical development. Looking at a British database of 847 individuals from families with at least one child with SLI, this group then focused on nine CNTNAP2 polymorphisms. Each of these nine had a significant association with non-word repetition scores. The most powerful association was for a haplotype labeled ht1 linked to a lowering of non-word repetition scores by half a standard deviation. This same pattern is also heavily associated with autism. This new research illustrates the growing contribution of genetic analysis and the complexity of

genetic interactions involved and the ways in which they impact the formation of connections between areas in early brain development

Van der Lely has emphasized the extent to which she can identify a highly specified subgroup of SLI language users. However, attempts to replicate this selection specificity {Bishop, 2000 #9134} have not succeeded. Moreover, even if such a distinct subtype were identified, and even if there were some statistical association between that disorder and some genetic mutation or set of mutations, we would still need a cognitive or neural model by which the mutations could be linked mechanistically to the disorder in question.

2. SLI deficits are domain-specific

Claims of specific competence deficits in children with SLI have been used to support nativist views regarding the "faculty of language" {Hauser, 2002 #9733}. The idea is that the specificity of this disorder implies that language learning and processing depend on a separate linguistic module, rather than on domain-general processes, and that damage to the module causes highly specified symptoms as hypothesized in SLI. However, the comorbidity of non-linguistic task difficulties for children with SLI {Barry, 2006 #10637} calls this interpretation into question.

Many studies have found deficits in non-linguistic tasks in SLI patients, seemingly disputing the definition of SLI as an exclusively linguistic (or exclusively grammatical) disorder. The findings that SLI patients have impaired phonological short-term memory {Evans, 1999 #7796}, that the KE family and others have comorbid motor problems {Vargha-Khadem, 1995 #10656}, and other trends toward cross-domain SLI

symptoms are supportive of a richer understanding of SLI than that which restricts the impairment to one grammatical competency.

Finally, in a gating task of word identification from incomplete auditory data, an SLI group took longer to produce only the correct response consistently {Mainela-Arnold, 2008 #10649}. These findings suggest a prolonged process of competition, and these data help connect a potential perceptual deficit with the accompanying processing impairment.

3. SLI is a deficit in linguistic competence

Van der Lely & Christian {, 2000 #9479}) describe the difference between processing models and competence deficit models as the difference between whether or not "impaired input processes and processing capacity cause SLI" (p. 35). That is, within a competence deficit model, any negative effects that stem from SLI should be restricted to the linguistic domain, and basic cognitive capacity (such as working memory) should remain within the normal range. More concretely, these competence models of SLI predict that genetic changes cause domain-specific effects, and that those effects consist of competency deficits such as the Extended Optional Infinitive stage. However, there is real variability in the symptoms and deficits showed by SLI children, and multiple cognitive limitations could be the source of these varied deficits.

First, as noted earlier, SLI is characterized by a variety of co-morbid impairments {Norbury, 2002 #10659}, such a phonological and oro-facial motor control disorders. The data from the KE family of language-impaired individuals is characterized by just such comorbidity {Bishop, 2002 #10638}. Motor control problems, while clearly

implicated in deficits in language production, do not fit the profile of a uniquely human mutation-based impairment in grammatical usage, as in a competency model. Van der Lely et al. {, 1998 #10654} differentiate between these comorbid impairments and the root cause of SLI by selecting for participants who fall within normal range in these other language-related skills. This exclusion certainly increases the likelihood that there is a common etiology for the impairment in the Grammatical SLI subgroup. However, there is no room in such a model to explain the non-linguistic deficits of the many individuals excluded during this process. Unless there is some plausible explanation for the rest of these SLI sub-types, it is difficult to accept a model dependant on restricting any variance in the patient population.

Second, the competency impairments that serve as *cause* in models such as Rice & Wexler {Rice, 1996 #10652} and van der Lely et al. {, 1998 #10654} could in fact be the *result* of impairments in processing, which need not be domain-specific. For example, the Competition Model account would suggest that at least some children with SLI have problems with long-distance neural connectivity. Such problems could have a particularly strong impact on the coordination of information between posterior lexical areas and anterior syntactic areas. These problems would not impact linguistic competence, but rather the speed and accuracy of processing during resonant communication between these two separate areas. This emphasis on the vulnerability of between- area communication is in accord with the Competition Model emphasis on processing cost. According to the Competition Model, the SLI patient is performing a complex task with limited cognitive resources, and the limitation of those resources

creates predictable and consistent negative effects for the most resource-intensive aspects of language processing.

Third, there is substantial evidence that the SLI diagnosis can be further sub-divided based on whether the impairment in language competence extends to receptive as well as expressive language use {Evans, 1999 #7796}. It is difficult to see how a competence account alone can explain this further dissociation. However, the Competition Model can account for this asymmetry as a result of differences in connectivity. Varieties of SLI that are exclusively expressive function much like Broca's aphasia. In typical speakers, Broca's area serves to gate the firing of lexical items during production. In expressive SLI, as in Broca's aphasia, disruption in the connectivity between Broca's and Wernicke's areas interrupt the smooth gating of lexical items for production. This gating is only important during production and is not involved in comprehension. In the case of receptive-expressive SLI, then, we would expect to see a different, more general problem of information exchange between brain areas, affecting connections between Broca's area, DLPFC, Wernicke's area, and attentional areas generally.

Aphasia

These questions can be further expanded when considering aphasic patients. Aphasia arises when a brain lesion from trauma or stroke produces a linguistic impairment. Traditionally, aphasia has been divided into three main categories: Broca's, or nonfluent aphasia; Wernicke's, or fluent aphasia; and anomia, or problems with word finding. Additional types include global and conduction aphasia. Because the etiology

of aphasia is much clearer than that of SLI, and because the injuries are easier to map, aphasia provides a useful counterpoint to SLI. In SLI, the functional deficits are well defined but etiology remains unclear. In aphasia, the opposite is true.

Although aphasia has a clear etiology, lesion site is not a strong predictor of symptom pattern. Two patients with lesions in very different areas will often have similar linguistic profiles. Similarly, patients with lesions in the same area often end up with very different profiles in language performance. Moreover, if a person with Wernicke's aphasia is impaired in grammaticality judgment in a way that resembles a person with Broca's aphasia, this does not necessarily mean that Broca's and Wernicke's areas perform the same processing tasks, or that they are neurally identical. Rather, it means that grammar is a complex computational task with certain high-risk components that can be impaired in similar ways through damage to various parts of the language network. In this way, aphasia often teaches us more about the nature of the language processing system than it does about the brain.

Crosslinguistic studies of aphasia {Bates, 1991 #4977} have illustrated and validated this approach. There is a rich literature demonstrating differences between Broca's aphasics who are native speakers of different languages. For example, the use of agreement in aphasic patients whose native language is Italian is relatively less impaired than in comparison patients whose native language is English. This result is predictable in a Competition Model framework, given the strength of agreement cues in Italian compared to English. In both Broca's and Wernicke's aphasics, obligatory structures such as SVO word order in German and Italian patients are preserved (Bates et al., 1988). These structures are also the most valid, least costly (as defaults in the language), and

most highly frequent. Similarly, when Turkish speakers become aphasic, they still maintain the use of SOV word order, which is the standard in Turkish. As Elizabeth Bates would say, "You can take the Turks out of Turkey, but you can't take the Turkish out of the Turks." So are the properties that aphasics fail to use or comprehend contingent on the language of the patient. Overall, this research shows that the major determinant of cue survival in aphasia is the relative strength of the cue in the language of the aphasic.

The status of competence accounts in aphasia is similar to its status in SLI. In SLI, competence accounts look to a simple causal association between a damaged component (such as a specific mutation) and a language deficit. In aphasia, these accounts also require that a specific lesioned local area or module be the root cause of the aphasic disability. In both cases, the competence approach fails to consider the broader context of the language system, wherein levels of processing (semantics, syntax, lexicon, audition, comprehension) interact within a distributed functional neural network of brain areas {Bookheimer, 2002 #10367}.

In the Competition Model analysis, the effects of lesions must be understood in terms of the damage inflicted on both grey matter and white matter. Damages to grey matter impact the content of the representational maps that are at the core of the system. In one view, these maps could be viewed as encoding specific linguistic competence. However, when grey matter is damaged, there is usually accompanying damage to the white matter that connects the local map with other processing regions. Thus, actual patterns of aphasia relate not just to the processing in local maps, but also disorders in

connectivity and processing that occurs as two or more maps attempt to work in synchrony.

Gupta et al. {, 2003 #9529} showed that, in children who had had early focal lesions, learning was quantitatively delayed in word learning, non-word repetition, and serial recall tasks. Although the level of performance was impaired overall, the relation between measures of verbal working memory and word learning was maintained, and those relations were similar to the control group. These data are consistent with the finding that children with focal lesions are able to achieve functional language use, although their overall reaction times are often slower than that of controls {MacWhinney, 2000 #7795}.

A similar, and perhaps even more striking, finding comes from Wilson and Saygun {, 2004 #10657}. They report evidence in direct contradiction to models that hypothesize that Broca's area is the unique site for comprehension of maximal trace projections {Grodzinsky, 2000 #10646}. In Wilson and Saygun's study, *all patient groups*, including anomics, shared a general impairment pattern, although the quantitative performance of the patients varied, as expected. These results show that a number of injuries to the language network can create similar performance profiles. These data fit well with the analysis of the Competition Model.

Finally, data suggest that non-aphasic patients with left-hemisphere lesions have sentence comprehension and free word recall deficits compared to similar right-hemisphere lesion patients {Vallar, 1988 #10653}. The left hemisphere patients had lexical and syntactic deficits, as well as a decrement in verbal long-term memory.

Consistent with the view of language as a complex and distributed system, the varied lesion sites all had some broad effect on linguistic *processing* as demonstrated through varied behavioral measures.

Summary and Conclusion

This chapter has presented ways in which the Competition Model can be useful in understanding SLI and aphasia. The Competition Model framework suggests that there are multiple pathways that can produce SLI. As we have argued, impairment in processing capacity can result in symptoms that reflect weakened "vulnerable" linguistic structures. Such a processing deficit, however, could have multiple causes, consistent with accounts of decreased verbal working memory {Gathercole, 1993 #6961}, phonological processing impairment {Tallal, 1974 #5223}, and orofacial motor impairment {Vargha-Khadem, 1995 #10656}. In this sense, SLI can be viewed as linked to an *endophenotype* {Gottesman, 2003 #10644} in which a complex set of genetic variations produce a phenotypically consistent cognitive outcome. The current literature suggests that at the core of the SLI endophenotype is a set of individual variations that can influence the operation of verbal working memory. We must note that verbal working memory is not a single cognitive process. On the one hand, the six local cortical maps that support language each maintain some type of local memory through competitive activation patterns. However, this local activation is not enough to effectively control higher levels of language processing. Once an item is activated locally, it must receive additional support from other areas and it must also trigger activation in other areas.

For example, a word such as “more” may maintain activation in the posterior lexical area. This activation constitutes a certain level of local memory. However, this item must then activate Broca’s area to trigger combination with a noun, as in “more campers”. Once this phrase links up with a verb, as in “more campers visited the park”, activation then spreads to frontal areas that encode perspective (MacWhinney, 2008) and overall mental models. The distributed and interactive nature of this information flow requires smooth white matter connections between each of the areas involved in processing. This suggests that an SLI endophenotype involves disruptions that interrupt the timing or accuracy of this information flow.

This approach suggests that we should not imagine working memory as a discrete neural storage area. As Mainela-Arnold, Evans, & Coady {, 2008 #10649} write: “Current developments in connectionist modeling and neuroscience suggest that what has been referred to as working memory capacity may be comprised of global competition of activation in large-scale neural networks with a top-down attentional bias from prefrontal cortex (PFC) circuits” (p. 390). This is consistent with other domain-general models of working memory function {Schneider, 2003 #9880}.

The Unified Competition Model is designed to interface with perceptual and memory processes and integrates working memory buffers at each level of processing. In this way, the model is aligned with cognitive models such as ACT-R {Anderson, 1998 #7972} that maintain local buffers. These accounts fit behavioral data that show competition and interference at multiple levels in online language processing. Because language depends on the integration of multiple inputs to produce either intelligible outputs or comprehension, describing the precise nature of this coordination is crucial for

neurally grounded language models. By (for example) mapping connections between language areas with diffusion tensor imaging (DTI) {Schmahmann, 2007 #10636}, or by using functional connectivity analyses {MacDonald, 1992 #5474}, we can provide further articulation of this account.

Although the Competition Model emphasizes the structural integrity of language, it also emphasizes the complexity of neurolinguistic processing. Although we expect a wide variety of lesions or endophenotypes to produce similar symptom patterns, we also expect that careful scientific work can eventually separate out the relative contributions of the six separate local processing regions and the complex patterns of white matter connections between them. We also expect that some symptom patterns will arise not from lesions, but from poor mappings between resonant areas and cellular-level problems with neuronal firing and consolidation. In this sense, we would agree with van der Lely {, 2005 #10655} when she notes "it is only by identifying pertinent . . . phenotypes that we can illuminate functionally specialized cognitive systems" (p.53). There are many contrasts that are illuminative in this way; for example, some aphasics are expressive (fluent), while others are non-fluent. Some subjects have affected prosody and labored articulation, whereas others do not. Similarly, in SLI, some subjects have reduced working memory and others are closer to normal. However, we do not want to use these dissociations to link impairments to modules. Rather, we need to look at the overall dimensions of cue strength and cue cost as the linguistic backdrop against which processing limitations should be measured. Only by collecting a rich set of measures of performance in both experimental and naturalistic contexts can we achieve clearer understandings of the various ways in which this integrated system can be impaired.

Language, though unique in the types and complexity of the necessary calculations, is in the end a cognitive process, and this simple fact leads to a more complete understanding of language disorders.

For Further Reading:

Bishop, D. (2002) 'The role of genes in the etiology of specific language impairment', *Journal of Communication Disorders*, 35: 311-328.

MacWhinney, B. (2008a) 'A Unified Model', in P. Robinson & N. Ellis (Eds.), *Handbook of Cognitive Linguistics and Second Language Acquisition,* Mahwah, NJ: Lawrence Erlbaum Associates.

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