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The Neurobiology of Sentence Comprehension

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1. Introduction

On the surface, a sentence (for example, *I am writing this chapter on an aging Sony notebook computer with a sticky Q key that I bought six years ago*) is a linear sequence of words. But in order to extract the intended meaning, the reader must combine the words in just the right way. That much is obvious. What is not obvious is how we do that in real time, as we read or listen to a sentence (see Tanenhaus, this volume). The standard answer to that question, which derives from generative linguistic theories (Chomsky, 1986), is that we combine words at two levels: a level of structure (syntax) and a level of meaning (semantics). In our example sentence, syntactic combination entails assigning the grammatical subject role to *I*, the direct object role to *this chapter*, the object of the preposition role to *aging Sony notebook computer*, and so on. Semantic combination entails identifying who is doing the writing (the *Agent*) and what is being written (the *Theme*). Furthermore, the standard view claims that syntactic combination involves application of phrase structure rules that are abstracted away from individual words. For example, the rule S → NP VP stipulates that every sentence in English is composed of a noun phrase and a verb phrase, in that order, regardless of the individual words in the sentence. These rules define hierarchical relationships within each sentence, in which some phrases or clauses modify others (e.g., the prepositional phrase *with a sticky Q key* modifies the noun phrase *aging Sony notebook computer*). The phrase structure rules are also claimed to contain recursive elements that permit sentences to be glued together (*I am writing this chapter on an aging Sony notebook computer* and *I bought [the Sony notebook computer] six years ago*) to form ever-longer sentences. One result of recursion is the existence of "long-distance dependencies," which can obscure aspects of semantic combination. The clause *that I bought six years ago* does not explicitly provide any indication of what was purchased; nonetheless, every fluent speaker of English immediately recognizes that the purchased item was a Sony computer (or perhaps a sticky Q key). Another claim of the standard model is that syntactic combination precedes and "prepares the way" for semantic combination. Correspondingly, the syntax tells the semantics that the item being purchased was in fact either the Sony computer or the sticky Q key referred to in the main clause. In other words, syntactic combination is claimed to always come first, followed by semantic combination. Finally, because the recursive, hierarchical nature of human syntax seems to be so unique (with respect to other natural communication systems and other aspects of human cognition), syntax is presumed to involve language-specific neural circuits that evolved in humans.

This view of how words are combined has dominated thinking for a long time. The longevity of the standard view is a testament to its elegant explanatory power in terms of linguistic and cognitive modeling. We will argue here, however, that many aspects of this model are very likely wrong, and that its inadequacies become clear when one tries to relate the model to neurobiology. Much of the extant work on language and brain has assumed

some variant of the standard model. We review this work here and conclude that these efforts have met with limited success (for similar conclusions, see Kaan & Swaab, 2002; Stowe, Haverkort, & Zwarts, 2005; for a more optimistic opinion, see Friederici, 2002, or Friederici & Kotz, 2003). In our review, we will attempt to describe which aspects of the model seem to be supported by the neurobiological evidence, and which aspects do not.

We will conclude our chapter by advocating for a research paradigm that is grounded as much in the principles of evolution, genetics, and neurobiological design as it is in the principles of linguistic and psycholinguistic modeling. In his engaging introduction to neurobiology, Gordon Shepherd (1994) comments that "nothing in neurobiology makes sense except in the light of behavior" (p. 9). His point is that no matter how complex a neural circuit might seem, one can always be confident that it is designed to mediate some specific naturally occurring behavior. Generally, the neural circuit becomes more understandable once the relevant behavior is known. It seems reasonable to suggest that the converse is equally true: No matter how complex a naturally occurring behavior might seem, one can always be confident that there is a neural circuit designed to mediate it. The complex behavior will become more understandable once the relevant neural circuits are known. This is because neural circuits (that is, "functional units" of neurobiological organization; Shepherd, 1994) are the likely basis of neurobiological evolution (Jacob, 1974). Knowledge of the relevant neural circuits therefore links the behavior to its evolutionary history and to relevant genetic mechanisms, and will almost certainly lead to a more accurate vision of the behavior. Conversely, a theoretical perspective that imposes *a priori* assumptions concerning language and linguistic structure onto the brain, without due consideration of known neurobiological principles, might lead to a biased and ultimately inaccurate view of human language, language processing, and the evolutionary history of this important behavior.

We believe that a serious effort to understand the neurobiology of language should adopt a neurobiological perspective right from the start. One reasonable assumption is that some principles of neurobiological design are conserved across different functions. If so, then useful analogies might be made between language processing and functions that are better understood at the neural circuit level. One potentially useful concept is the notion of "streams of processing." Visual information, for example, is segregated at the cortical level into multiple parallel streams of processing (e.g., dorsal and ventral streams that process object and spatial aspects of the stimulus, respectively; Ungerleider & Haxby, 1994). These processing streams are thought to be independent in some respects (each stream processes a distinct aspect of the visual world) but highly interactive in others (crosstalk between the streams occurs constantly). Analogously, aspects of sentence comprehension might be segregated into distinct but interacting processing streams (Hickok & Poeppel, 2000; Kim & Osterhout, 2005; Osterhout, McLaughlin, Kim, Greenwald, & Inoue, 2004; Trueswell, Tanenhaus, & Garnsey, 1994).[1] If so, then the primary task would be to identify the processing streams and characterize their interactions. Eventually, we would want to identify the neural circuits that mediate these processing streams and learn something about their evolutionary histories and genetic influences. Ultimately, the goal would be to link the neurobiological evidence with a psycholinguistic theory of language processing.

2. Some commentary on methods of investigation

Progress in this area, as in any other, depends on the appropriateness and utility of the available methods of investigation. The primary methods include the study of brain-damaged patients who have language impairments (the "deficit" approach); non-invasive hemodynamic-based neuroimaging methods such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) (Cabeza & Kingstone, 2001); and methods for recording the brain's electromagnetic activity from the scalp, such as event-related brain potentials (ERPs; Handy, 2005) and magnetoencephalography (MEG; Hämäläinen, Hari, Ilmoniemi, Knuutila,

& Lounasmaa, 1993). Another less frequently used method, transcranial magnetic stimulation (TMS; Walsh & Cowey, 2000), can be used to induce "reversible lesions" in restricted parts of the human brain.[2] Inevitably, none of these methods perfectly reflects the neural processes involved in language comprehension; each method comes with strengths and limitations. We discuss some of the more important ones here.

Deficit studies have been the most important method historically. However, they are complicated by the fact that the lesions are "accidents of nature" and are therefore not controlled in terms of the lesion's location and extent. It is also not at all trivial to properly characterize the behavioral/cognitive deficit resulting from a lesion, or to ascertain the exact type and degree of neurobiological damage.[3] For example, a deficit could result due to damage to the cortical tissue, or to damage to the fiber tracts that lie underneath the gray matter; different conclusions would follow concerning the neural circuits underlying the deficit, depending on what type of damage is assumed. As a consequence, lesion studies can tell us whether an area is essential for some function, but cannot tell us much about the whole circuitry that is involved in the task (cf. Price, Mummery, Moore, Frakowiak, & Friston, 1999). Furthermore, compensatory processes can improve the patient's functioning. This is useful for the patient, but obscures the relationship between neural circuits and specific functions. Each lesion and the resulting deficit tends to be unique to the patient, making it difficult to appropriately group individual patients into larger groups. Some researchers have therefore advocated for single-subject designs (Shallice, 1979). Studies of single subjects are often designed to identify *dissocations* between functions or, even better, *double dissociations*. A dissociation of functions A and B is found if, for Patient Y, A is damaged while B is preserved. A double dissociation exists if, for Patient Z, function B is damaged while function A is preserved (Shallice, 1988). The inferred dissociability of the functions would be supported even if the neural substrates of those functions are not clear. However, single-subject studies also come with caveats, most notably the inability to statistically generalize to larger groups of people. Finally, the location of neurological damage is generally assessed using computerized tomography (CT) or structural magnetic resonance imaging (MRI). These methods identify areas of necrosis, but are less sensitive to the presence of hyperfusion and hypofusion. Hypometabolic cortical areas might not sustain normal function (Caplan, Hildebrandt, & Makris, 1996).

Neuroimaging methods such as PET and fMRI provide relatively good spatial resolution and do not suffer from the caveats associated with deficit studies. However, these methods do not directly measure brain activity but instead index changes in blood flow and blood oxygenation, which are assumed to be useful proxies of neural activity. Although there is some evidence to support this assumption (Mukamel, Gelbard, Arieli, Hasson, Fried, & Malach, 2005), much is unknown about the precise coupling between neural activity and blood flow (Logothetis & Pfeuffer, 2004). Furthermore, changes in blood flow that result from increased activity in the brain are quite sluggish compared to the dynamics of cortical activity. Consequently, these tools suffer from a temporal resolution that is probably at least an order of magnitude worse than the presumed temporal resolution of the processes of interest (tens and hundreds of milliseconds). The hemodynamic response measured with fMRI (BOLD) is delayed several seconds (relative to the event eliciting it) and evolves over 10 to 15 seconds. This contrasts starkly with the fact that in normal fluent conversation, speakers produce (on average) three word, four syllables, and 12 phonemes per second (Levelt, 1999). Furthermore, the processing of a single linguistic unit, such as a word, most likely involves a constellation of processes, each having temporal durations considerably less than 1 second. In other words, under conditions that approximate normal speaking and reading, it is difficult to isolate the hemodynamic response to a particular word embedded within a sentence, much less the (phonological, syntactic, semantic, etc) processing steps that occur in processing that word. Furthermore, because sentence comprehension is inherently an integrative process, one cannot reasonably assume that

successive words and sentences are processed independently. This complicates efforts to isolate the response to particular words in a sentence by using event-related fMRI designs (e.g., Burock, Buckner, Woldorff, Rosen, & Dale, 1998). Event-related designs measure the BOLD response to rapidly sequenced individual events and assume that temporally overlapping BOLD responses summate linearly. Although the independence of overlapping hemodynamic functions has been demonstrated for simple visual stimuli (Dale & Buckner, 1997), the same cannot be said for words in sentences.

Assumptions of linearity and additivity play a crucial role in much of the deficit and neuroimaging literature. Researchers have generally assumed that language processing consists of activation of abstract linguistic codes (e.g., phonological, semantic, and syntactic codes) and computational processes that manipulate these codes (Caplan, 1994; Saffran, 2006). The component processes are assumed to be sufficiently independent (both functionally and neuroanatomically) such that they can be disrupted independently (with brain damage) or methodologically isolated from other components (in neuroimaging studies). If these assumptions are valid, then it should be possible to find patients with deficits that reflect breakdown in a particular component of the model (Saffran, 2006), or brain activations in neuroimaging experiments that reflect the engagement of that particular component (Caplan, 1994).

Unfortunately, these crucial assumptions are difficult to validate, and the invalidity of any one of them would be highly problematic for much of the deficit and neuroimaging literature. Consider, for example, a neuroimaging study designed to isolate the brain areas involved in sentence comprehension. Each subject participates in two conditions, one in which lists of isolated words are presented, and another in which sentences are presented. To isolate the sentence comprehension processes, activations observed in the word list condition are subtracted from activations observed in the sentence comprehension condition. But what function, exactly, does the subtractive method isolate? Many neuroimagers assume that the subtractive method successfully isolated the process of interest, and conclude that the residual activations reflect that process. But this conclusion assumes that the component processes of interest are independent. If that assumption is not valid, then it becomes very difficult to ascertain the function reflected in the residual activations. Because most subtractions are likely to result in some residual activation, this approach suffers from a powerful confirmation bias, in the absence of independent evidence to support the assumption of additivity. There are neuroimaging research designs that mitigate this problem, including conjunction analysis (Price & Friston, 1997; see also Caplan & Moo, 2004) and parametric designs that look for graded activity (Buchel, Homes, Rees, & Friston, 1998; Penny, Stephan, Mechelli, & Friston, 2004; for a general account of fMRI designs, see Petersson, Nichols, Poline, & Holmes, 1999). However, these designs are only now being adopted for use in sentence comprehension studies. It seems likely that these designs will turn out to be more constructive.

Neuroimaging methods based on hemodynamic measures provide a static image of language comprehension, in which the time dimension is collapsed into one image of brain activity. Language comprehension, however, is a highly dynamic process. It would be advantageous to have methods that measure the process of comprehension as it unfolds over time. One such method involves recording ERPs from the scalp. ERPs reflect changes in electrical activity that occur in response to a sensory, cognitive, or motor event. They are thought to reflect the summed, simultaneously occurring postsynaptic activity within neocortical pyramidal neurons. Topographical features of the ERP are referred to as components and can be described in terms of polarity (positive and negative), amplitude, peak latency, and scalp distribution. Because ERPs are multidimensional, they are more likely to be differentially sensitive to different aspects of processing than are other measures. And unlike other methods, ERPs provide a nearly continuous sampling of the brain's electrical activity during the process of sentence comprehension.

However, ERPs are not without disadvantages. All methods for localizing the neural source(s) of a scalp-recorded effect provide relatively low spatial resolution, much worse than the resolution of hemodynamic neuroimaging methods (Slotnick, 2005). Furthermore, the so-called inverse solution (computing the neural source from the scalp activity) is a mathematically ill-posed problem, as any distribution across the scalp can be accounted for by a large number of possible source configurations. Unique solutions are possible given certain limiting assumptions (Michel et al., 2004). The traditional approach to source localization has been to search for point dipole sources (Michel et al., 2004). In general, this entails assuming a small number of dipole sources and iterating through all possible combinations of dipole location, orientation, and strength, looking for the best match between the source model and the observed scalp distribution. This method brings with it numerous limitations and caveats (Halgren, Dhond, Christensen, Van Petten, Marinkovic, Lewine, et al., 2002). More recently developed "distributed source" methods provide a true tomographic analysis analogous to that provided by hemodynamic neuroimaging methods, but with much greater temporal resolution (Dale et al., 2000; Dale & Sereno, 1993; for a review, see Michel et al., 2004). For example, Low Resolution Electromagnetic Tomography (LORETA; Pascual-Marqui, Michel, & Lehmann, 1994) estimates the current distribution throughout the entire three-dimensional cortex. The primary assumption is that dramatic changes do not occur across contiguous areas of cortex (i.e., in adjacent voxels). The primary advantage is that LORETA can provide an estimate of current distribution for each sample of brain activity (i.e., every few msec). The primary disadvantage is a reduced spatial resolution, relative to hemodynamic-based methods.

Clearly, then, each method for relating language to brain brings with it significant limitations. Some of the limitations can be minimized by combining methods. For example, fMRI activations can be used to constrain the inverse solution for ERP or MEG effects (Dale & Halgren, 2001; Dale et al., 2000). Unfortunately, one fundamental limitation cannot be minimized: All of these methods are correlational in nature. Although the antecedent conditions that elicit or modulate some ERP component (or produce some change in hemodynamic response) are relatively easy to determine, the specific cognitive process manifested by the component (or activation) is not. Similarly, although one can assess the correlation between some lesion site and some behavioral deficit, one can never be certain that that lesion site is the "neural home" of that behavior.

3. Segregating language into streams of processing

3.1. Deficit studies

The initial evidence of separable processing streams derived from studies of aphasic patients, in particular the syndromes known as Broca's and Wernicke's aphasia. Broca's aphasics typically produce slow, labored speech; the speech is generally coherent in meaning but very disordered in terms of sentence structure. Many syntactically important words are omitted (e.g., *the*, *is*), as are the inflectional morphemes involved in morphosyntax (e.g., *-ing*, *-ed*, *-s*). Wernicke's aphasics, by contrast, typically produce fluent, grammatical sentences that tend to be incoherent. Initially, these disorders were assumed to reflect deficits in sensorimotor function; Broca's aphasia was claimed to result from a motoric deficit, whereas Wernicke's aphasia was claimed to reflect a sensory deficit. This interpretation was motivated by the proximity of the damaged areas to the left-hemisphere motor and auditory cortices, respectively. Thus, Broca's aphasia was thought to reflect a problem in production and Wernicke's aphasia was thought to reflect a problem in comprehension, perhaps reflecting two processing streams, one for production and one for comprehension. The model was centered around the use of words and had nothing to say about how words are combined to produce or understand sentences.

The standard assumptions about aphasia changed radically in the 1970s. Theorists began to stress the ungrammatical aspects of Broca's

aphasics' speech; the term "agrammatism" became synonymous with Broca's aphasia. Particularly important in motivating this shift was evidence that some Broca's aphasics have a language comprehension problem that mirrors their speech production problems. Specifically, some Broca's aphasics have trouble understanding syntactically complex sentences (e.g., *John was finally kissed by Louise*) in which the intended meaning is crucially dependent on syntactic cues – in this case the grammatical words *was* and *by* (Caramazza & Zurif, 1976). This evidence seemed to rule out a purely motor explanation for the disorder; instead, Broca's aphasia was viewed as fundamentally a problem in using the rules of syntax (or, alternatively, problems in using the function word vocabulary) to produce or understand sentences. Furthermore, it was assumed that Broca's aphasia resulted from lesions to the left inferior frontal gyrus (Brodmann's Area [BA] 44, 45) and that this area was a neural center for syntax. Accounts of agrammatism were very explicitly linked to models of syntactic structure and language processing that derived from linguistic theory and psycholinguistic models (Caplan, 1994, 1995). By contrast, Wernicke's aphasia was assumed to reflect a problem in accessing the meanings of words, and to result from damage to the left posterior temporoparietal region, including the angular gyrus and parts of the inferior parietal lobe (roughly, the posterior part of BA 22, and BA 39 and 40). The standard claim thus became one in which the left inferior frontal gyrus was a center for syntactic aspects of word combination (for both production and comprehension), whereas the left posterior temporoparietal cortex was a center for retrieving semantic knowledge associated with individual words, and perhaps for combinations of words as well. Thus, like the classical model, this model also posits two processing streams: one for dealing with syntactic aspects of word combination, and another for dealing with semantics (meaning).

These claims about the nature of the aphasic disorders are still quite influential. Closer consideration, however, raises many questions (Mohr, Pessin, Finkerlstein, Funkenstein, Duncan, & Davis, 1978; Vanier & Caplan, 1990). Caplan (1995), for example, notes many inadequacies in the agrammatism literature, including inadequate stimuli, overly specific interpretations of data combined with too little testing of the patients, and problems with subject grouping. Many of the problems stem from the difficulty of ascertaining in a precise way what is wrong (if anything) with the patients' ability to comprehend sentences (in contrast to speech production, in which the grammatical problems are overt). More generally, there is now a greater appreciation of the variability in symptoms and of the underlying anatomical complexities. Symptoms often vary considerably across patients, over time within a single patient, and across different tasks (Alexander, 2006; Kolk & Hescheen, 1992; McNeil & Doyle, 2000). "Pure" functional deficits affecting a single linguistically defined function are rare; most patients have a mixture of problems, some of which seem linguistic but others of which seem to involve motor or sensory processing, such as dysarthria or disprosody (Alexander, 2006). Many of the Broca's patients who produce asyntactic output are relatively good at making explicit grammaticality judgments (Linebarger, Schwartz, & Saffran, 1983), suggesting that their knowledge of syntax is largely intact. Similarly, it is not uncommon for Broca's aphasics to have asyntactic output but (seemingly) intact comprehension, bringing into question the claim that Broca's aphasia reflects damage to an abstract "syntax" area used in production and comprehension (Miceli, Mazzuchi, Menn, & Goodglass, 1983; see also Caramazza, Caasso, Capitani, & Miceli, 2005).

With respect to the anatomical correlates of the aphasic syndromes, lesions in the left inferior frontal gyrus are neither necessary nor sufficient to produce problems with syntactic comprehension (Alexander, 2006; Caplan et al., 1996; Dick, Bates, Wulfeck, Utman, & Dronkers, 2001; Dronkers, Wilkins, Van Valin, Refren, & Jaeger, 2004). Instead, lesions to almost any area around the left (and in some cases even the right) sylvian fissure can produce problems with syntactic aspects of sentence comprehension. Controversy continues to exist concerning the lesion sites most likely to produce Broca's aphasia. Some researchers claim that damage to

subcortical structures such as the basal ganglia are essential for producing lasting asyntactic symptoms (Alexander, Naeser, & Palumbo, 1990; Ullman et al., 1997; see also Friederici, Kotz, Werheid, Hein, & von Vramon, 2003). Other researchers have argued that cortical structures are critical and that subcortical structures play no role in the disorder (Nadeau & Crosson, 1995), or that lesions affecting both cortical and subcortical structures (and underlying white matter) are needed (Alexander, 2006). Lesions that produce classic Wernicke's aphasia are generally located in the posterior half of the left temporal lobe, sometimes involving the angular gyrus (Dronkers, Redfern, & Knight, 2000; Dronkers et al., 2004). More recently, Bates et al. (2003) and Dronkers et al. (2004) have used voxel-based lesion-symptom mapping (VBLSM) to evaluate the relationships between areas of injury and performance on language-related behavioral tasks, on a voxel-by-voxel basis, in a wide variety of patients with left-hemisphere strokes. Lesion locations that degraded the ability to combine words at the sentence level included the anterior (BA 22) and posterior superior temporal and angular gyrus (BA 39), and frontal areas BA 46 and 47.

In summary, it is clear that damage to the perisylvian cortex of the left hemisphere (the area surrounding the sylvian fissure), perhaps requiring additional damage to underlying white matter and subcortical structures such as the basal ganglia, is needed to produce a deficit in sentence comprehension. Anterior lesions do seem more likely to produce "agrammatic" symptoms whereas posterior lesions seem more likely to produce the comprehension problems typical of Wernicke's aphasia; furthermore, large lesions are necessary to produce lasting symptoms. What remains unclear are the exact correspondences between lesion site and dysfunction, and also the proper functional characterizations of the observed dysfunctions. On this last point, several theorists have proposed alternative explanations of the asyntactic behavior of Broca's aphasics. For example, in order to account for preserved grammaticality judgments in combination with asyntactic comprehension, several researchers have suggested that patients have limited processing resources that are insufficient for parsing purposes (Kolk & Heeschen, 1990, 1992). This notion is quite different from the claim that specifically syntactic knowledge or processes are lost. Another idea is that patients are able to parse sentences (that is, construct their grammatical structures) but cannot carry out additional operations on the computed structure (for example, mapping from a syntactic representation to thematic roles) (Schwartz, Saffran, & Fink, 1994). Grodzinsky (2000) has proposed that the agrammatism associated with damage to Broca's area reflects a very specific set of syntactic phenomena, specifically the processing of long-distance dependencies in sentences. Many of these ideas are supported (to varying degrees) by the published literature. Nonetheless, the actual nature of "asyntactic" comprehension (and indeed of the aphasias more generally) remains highly controversial (Saffran, 2006), and the problems noted by Caplan (1995) continue to plague the field. Progress has been limited by other factors as well. Many studies have simply summarized radiological reports and/or have displayed lesions on a single transverse section of the brain. Studies reporting more comprehensive radiological investigations have examined relatively few patients (Caplan et al., 1996; Tramo, Baynes, & Volpe, 1988), have not adopted a psycholinguistic approach to defining syntactic deficits (Karbe et al., 1989), or have not specified the boundaries of regions of interest that were analyzed (Dronkers et al., 2004). Taken collectively, the deficit literature provides a confusing picture for those attempting to infer the normal neural organization of language. This confusion is reflected in the disparate and mutually exclusive proposals deriving from the deficit work. Influential proposals include the following:

1) Localizationist models; e.g., Grodzinsky (2000), who claims that Chomskian traces are coindexed in Broca's area.

2) Variable localization models, in which different small areas of the brain support the same function in different individuals (Caplan, 1994).

3) Evenly distributed models; e.g. Damasio and Damasio (1992) and Dick et al. (2001),

who hypothesize that large regions of the brain support a function and usually assume that all parts of the region contribute equally to the function.

4) Unevenly distributed models, in which particular functions are unevenly distributed throughout a region (Mesulam, 1990).

It is not clear how, of even if, the deficit literature will provide the constraints needed to arbitrate between these and other competing ideas.

3.2. Hemodynamic neuroimaging studies

Non-invasive neuroimaging seems, at first glance, to be an excellent tool for identifying separate streams of syntactic and semantic processing, if they exist in the brain. Reassuringly, tasks involving sentence comprehension tend to activate the left perisylvian areas classically associated with aphasic sentence comprehension, including the left inferior frontal gyrus (LIFG) and left posterior superior and middle temporal gyri (Bavelier et al., 1997; Caplan, Alpert, & Waters, 1998, 1999; Just, Carpenter, Keller, Eddy, & Thulborn, 1996; Keller, Carpenter, & Just, 2001; Mazoyer et al., 1993; Price, 2000; Stowe et al., 1999; Stromswold, Caplan, Alpert, & Rauch, 1996). The left inferior parietal region is also frequently activated in sentence comprehension tasks (Awh et al., 1996; Paulesu et al., 1993), as are a number of right-hemisphere sites. In some reports, subcortical structures, most notably the basal ganglia, are activated. An important caveat is that many of these same areas are also activated by lists of words, although the activations are often larger in magnitude for the sentence comprehension tasks (Stowe et al., 1998).Whether or not there are activations that are specific to word combination at the sentence level, for either syntactic or semantic aspects of combination, remains unclear.

One clear implication of the neuroimaging work is that the classic model of aphasia, in which Broca's area subserves language production whereas Wernicke's area subserves language comprehension, seems to be wrong (cf. Stowe et al., 2005). Sentence comprehension often produces frontal as well as posterior activations, and activity in both regions increases when sentences are complex (Caplan et al., 1998, 1999; Stowe et al., 1998).

Less easy to evaluate are implications of neuroimaging work for the revised model of aphasia, in which syntax is mediated by the frontal cortex and semantics is mediated by posterior cortex. In order to evaluate this claim, stimulus or task manipulations are needed that isolate these two putative streams of processing. Some of the strategies used to isolate syntactic processing in sentence comprehension experiments have included the following contrasts: (1) syntactically complex sentences *vs*. syntactically simple ones (Caplan et al., 1998; Caplan et al., 1999; Caplan et al., 2001; Stowe et al., 1998); (2) sentences which contain syntactic structure *vs.* word lists (Kuperberg et al., 2000; Stowe et al, 1998; Stowe et al., 1999); (3) sentences that contain "pseudowords" (e.g., "The blives semble on the plim") *vs.* normal sentences (Friederici et al., 2000; Mazoyer et al., 1993; Moro et al., 2001); and (4) sentences that contain a syntactic anomaly *vs.* sentences that are syntactically well-formed (Friederici, Ruschemeyer, Hahne, & Fiebach, 2003; Kuperberg et al., 2000; Kuperberg et al., 2003; Ni et al., 2000; Newman et al., 2001). The assumptions underlying these contrasts are roughly as follows: (1) syntactically more complex sentences induce more syntactic processing relative to syntactically simple sentences; (2) sentences but not word lists engage syntactic processes; (3) sentences with pseudowords minimize semantic processing but not syntactic processing; and (4) sentences with syntactic anomalies require more syntactic "work." Unfortunately, the reported patterns of activation vary widely across these different contrasts (Kaan & Swaab, 2002; Stowe et al., 2005). For example, in most studies complex sentences elicit more activation in or near Broca's area than do simple sentences, although angular gyrus activations have been reported instead in at least one report (Caplan et al., 2001). Sentences sometimes but not always activate Broca's area more than word lists do; in fact, the converse is sometimes reported (cf. Stowe et al., 2005). Syntactically anomalous

sentences usually activate regions in the temporal lobe more than do well-formed sentences, although frontal activations (generally anterior to Broca's area) have occasionally been reported.

Semantic processing has been isolated in sentence processing experiments by comparing sentences with real words to sentences containing pseudowords (i.e., word-like stimuli with no semantic representations) (Röder, Stock, Neville, Bien, & Rösler, 2002), and well-formed sentences to sentences with semantic anomalies (Friederici et al., 2000; Kuperberg et al., 2000, 2003; Hagoort, Hald, Bastiaansen, & Petersson, 2004; Kiel, Laurens, & Little, 2002). The assumption seems to be that real words will activate semantic processes to a greater extent than pseudowords, and that semantic anomalies will lead to more semantic processing than semantically plausible words. Several of these studies have indicated that sentences containing semantic anomalies evoke more activation in the posterior or middle temporal lobe than do sentences that do not contain anomalies, which is consistent with the revised model of aphasia. However, Kuperberg et al. (2003), Kiel et al. (2002), and Hagoort et al. (2004) report inferior frontal activations to the semantically anomalous sentences either in addition to or in the absence of temporal activation, which is not consistent with the model.

It may be worth pointing out again that fMRI does not isolate online processing at the point of the critical word itself but also images everything that comes before or after that point. While what comes before may wash out in the counterbalancing, what comes afterwards may not. So, in imaging a syntactic anomaly, one is not just imaging what happens at the anomaly itself but all the other consequences of encountering the anomaly, as well as task and decision-related activity (if subjects are required to carry out a task). Such consequences after encountering the anomaly may range from syntactic, to semantic to attentional. One cannot necessarily assume that these processes cancel out in comparing different types of anomalies because the consequences of the anomalies after the word may differ depending on the nature of the anomaly (Kuperberg et al., 2003).

Recently, alternatives to the revised model of aphasia have been proposed, based on neuroimaging results (Bookheimer, 2002; Dapettro & Bookheimer, 1999; Gabrieli, Poldrack, & Desmond, 1998; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997; Hagoort, 2005; Poldrack et al., 1999). For example, Hagoort (2005) proposes that different areas of the left inferior frontal gyrus mediate different levels of combinatory analysis ("integration" in Hagoort's terminology), for both sentence production and sentence comprehension. Specifically, the claim is that more posterior regions of the left inferior prefrontal cortex (BA 44 and the ventral portion of BA 6) integrate phonological units, middle regions of the inferior prefrontal gyrus (BA 45) integrate syntactic units, and the most anterior and ventral regions (BA 47) integrate semantic units. Lexical semantics and structural frames associated with each word are claimed to be retrieved in the posterior superior temporal lobe. This is an interesting model, but the relevant imaging evidence motivating the LIFG claims is mixed (e.g., Barde & Thompson-Schill, 2002; Gold & Buckner, 2002; Thompson-Schill, 2002). One important caveat is that the relevant evidence is comprised mostly of studies involving presentation of individual words rather than sentences. Another is that damage to the relevant LIFG areas does not necessarily disrupt semantic processing (Price et al., 1999), suggesting that the LIFG is not essential for semantic processing (cf. Thompson-Schill, 2002). Furthermore, other neuroimaging evidence seems to indicate that semantic knowledge (and relevant processing) is represented within a widely distributed network encompassing large swathes of the frontal, temporal, parietal, and occipital lobes, rather than in discrete cortical locations (Tyler et al., 2003).

Taken as a whole, then, the imaging literature does not provide clear evidence of anatomically distinct syntactic and semantic processing streams, and does not definitively locate these streams in the brain. This conclusion is reinforced by a recent meta-analysis of PET and fMRI studies designed to isolate phonological, syntactic, and semantic processes in the brain (Vigneau et al., in press).

The meta-analysis shows that (across studies) all three types of processes have been localized to posterior portions of the left frontal lobe, much of the temporal lobe, and the inferior parietal lobe (see Figure 1 in Vigneau et al.). The authors gamely attempt to infer distinct neural circuits for each of the three types of processes based on this evidence. It seems to us, however, that the collective data do not provide compelling evidence of separate processing streams.

A related and very important issue concerns the language-specificity of these activations. Interestingly, listening to or mentally rehearsing music activates many of the same regions activated during sentence comprehension, including the LIFG and left posterior temporal lobe (Halpern & Zatorre, 1999; Hickok et al., 2003; Koelsch, 2005; Koelsch et al., 2002). Non-verbal and non-musical motor planning also activates the LIFG (Binkofski et al., 2000; Lacquaniti et al., 1997). Tasks that require manipulation of sequences over time (Barde & Thompson-Schill, 2002; Gelfand & Bookheimer, 2003) and stimuli that deviate from a familiar patterned sequence (Huettal, Mack, & McCarthy, 2002) produce activation in many frontal areas (including the inferior frontal gyrus) and in the basal ganglia. With respect to the temporoparietal areas, the temporal cortices are often activated in tasks that are not linguistic but that require conceptual processing (e.g., Bar & Aminoff, 2003; Chao et al., 1999; Martin & Chao, 2001). More generally, several reviewers of the neuroimaging-and-language literature conclude that there is little evidence of any truly language-specific neural centers (Patel, 2003; Price, Thierry, & Griffiths 2005; Kaan & Swaab, 2002; Stowe et al., 2005). Instead, these reviewers propose that language might take advantage of a number of domain-general neural circuits. Indeed, a number of proposals associate LIFG and surrounding pre-frontal cortex with functions that extend beyond grammar and language, including working memory (Smith & Jonides, 1998); selection (Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997), cognitive control (Miller & Cohen. 2001).

3.3. Event-related potential studies

Unlike hemodynamic-based methods, ERPs allow one to track changes in brain activity over time with great temporal resolution, as a person is reading or listening to a sentence. ERPs might therefore be ideal for isolating the neural responses to particular critical words in sentences. A particularly fruitful approach has involved the presentation of linguistic anomalies. If syntactic and semantic aspects of sentence comprehension are segregated into distinct streams of processing, then syntactic and semantic anomalies might affect the comprehension system in distinct ways. ERPs (unlike hemodynamic methods) have the temporal resolution necessary to isolate the neural response to the anomalous words. A large body of evidence suggests that syntactic and semantic anomalies do in fact elicit qualitatively distinct ERP effects, and that these effects are characterized by distinct and consistent temporal properties. Semantic anomalies (e.g., *The cat will bake the food …*) elicit a negative wave that peaks at about 400 ms after the anomalous word appears (the *N400 effect*) (Kutas & Hillyard, 1980, 1984; Osterhout & Nicol, 1999). By contrast, syntactic anomalies (e.g., *The cat will eating the food …*) elicit a large positive wave that onsets at about 500 ms after presentation of the anomalous word and persists for at least half a second (the *P600 effect*) (McKinnon & Osterhout, 1996; Osterhout, 1997; Osterhout & Holcomb, 1992, 1993; Osterhout & Mobley, 1995; Osterhout et al., 1996; Osterhout, McLaughlin, Allen, & Inoue, 2002; Osterhout & Nicol, 1999). These results generalize well across types of anomaly (with anomalies involving phrase structure, agreement, verb subcategorization, and constituent-movement all eliciting P600-like effects), types of languages (including word-order languages such as English, Dutch, and French, and case-marked languages such as Italian and Japanese; Angrilli et al., 2002; Inoue & Osterhout, in preparation), and various methodological factors (including modality of the input, rate of word presentation, and presenting isolated sentences and natural prose; Allen, Badecker, & Osterhout, 2003; McKinnon & Osterhout, 1996; Osterhout & Holcomb, 1993; Osterhout et al., 2002). In

some studies, syntactic anomalies have also elicited a negativity over anterior regions of the scalp, with onsets ranging from 100 to 300 ms (the so-called *left anterior negativity*, or LAN, effect; Friederici, 1995; Neville et al., 1991; Osterhout & Holcomb, 1992; Osterhout & Mobley, 1995).

These results seem to indicate that the human brain does in fact honor the distinction between the form and the meaning of a sentence. However, as we note below, there are exceptions to this generalization, and the exceptions tell us quite a bit about how the syntactic and semantic "processing streams" interact with each other during sentence comprehension (Kim & Osterhout, 2005; Kuperberg et al., 2003).

The sensitivity of the N400 and LAN/P600 effects to semantic and syntactic manipulations, respectively, does not necessarily imply that these effects are direct manifestations of semantic and syntactic processing (Osterhout et al., 2004); nor does it indicate that they are in any sense language specific. The available evidence suggests that they are not, in fact, direct manifestations of neural circuits specific to syntactic or semantic aspects of language processing. For example, some types of misspelled words elicit a positive wave that is indistinguishable from the P600 elicited by syntactic anomalies (Kolk et al., in press; Kim & Osterhout, in preparation), suggesting that the P600 effect is not specific to syntax. Furthermore, deviations from expected musical forms (e.g., deviant notes in well-known musical pieces) elicit P600-like effects (Besson et al., 1998; Koelsch, 2005; Patel, 2003; Patel et al., 1998), suggesting that the P600 effect is not specific to language. It is also conceivable that the P600 effect is a member of the P300 family of positive waves elicited by a wide variety of "oddball" stimuli, that is, stimuli that deviate from a preceding sequence (Donchin, 1981; for commentary on this possibility, see Coulson & Kutas, 1998; Osterhout et al., 1996; Osterhout & Hagoort, 1999). What do these categories of anomaly all have in common? One reasonable generalization is that they all deviate from some expected pattern or sequence. That is, the P600 effect conceivably reflects the operation of a neural circuit that mediates "patterned sequence processing." Consistent with this possibility, Dominey and colleagues have shown that violations of syntax-like patterns that are implicitly learned in the laboratory elicit a P600-like positivity (Hoen & Dominey, 2004).

Similarly, the N400 component is sensitive to manipulations that are not explicitly linguistic in nature. In semantic priming studies, smaller N400s are evoked by pictures preceded by related compared to unrelated picture primes (Barrett & Rugg, 1990; Holcomb & McPherson, 1994; McPherson & Holcomb, 1999) and objects that are congruous with their surrounding visual scene evoke a smaller N400 than objects that are incongruous with their surrounding visual scenes (Ganis & Kutas, 2003). Scenes preceded by congruous contexts – written sentence contexts (Federmeier & Kutas, 2001), successively presented static visual scenes conveying stories (West & Holcomb, 2002), or movie clips – evoke a smaller N400 than pictures preceded by incongruous contexts (Sitnikova, Kuperberg, & Holcomb, 2003). All of these experiments involved manipulations of meanings that are not explicitly presented in linguistic codes.[4] Intriguingly, Sitnikova et al. (2003) have shown that events in short silent movies of every-day activities (e.g., a movie clip of a man preparing to shave and then shaving with a rolling pin rather than a razor) that deviate from relevant "event schemas" elicit both N400 and P600 effects. Sitnikova (personal communication) has proposed that the N400 effect reflects the implausibility of the unexpected scene, whereas the P600 effect reflects the deviation from the expected sequence of events within the event schema. If so, then both of these processing streams may be involved in real-world visual comprehension as well as in sentence comprehension.

Ideally, one would like to locate these two processing streams in the brain. Lesion studies have attempted to identify the sites that eliminate or reduce the N400 and P600 effects. The lesion evidence seems to indicate that the N400 semantic context effect is affected by damage to the left temporal lobe and the temporoparietal junction. Importantly, damage to these areas tends to also produce aphasic syndromes characterized by a semantic processing deficit (for a review, see Van Petten & Luka, 2006; Friederici, Hahn, & von Cramon,

1998; Hagoort et al., 1996; Swaab et al., 1997). Conversely, damage to the frontal lobe does not typically affect the N400 effect to semantically inappropriate words Hagoort, Wassenaar, & Brown, 2003). Dipole and distributed source modeling of the magnetic equivalent of the N400 effect (the voltage difference between the semantically anomalous and well-formed conditions) has generally implicated the posterior halves of the left superior and middle temporal gyri and the temporoparietal junction (Helenius, Service, Salmelin, Service, & Connolly, 1998; Simos, Basile, & Papanicolaou, 1997).[5] With respect to the P600 effect, Kotz and colleagues have reported that damage to the basal ganglia can eliminate the P600 effect while leaving the N400 effect intact (Kotz, Frisch, von Cramen, & Friederici, 2003).

Osterhout and Inoue (in preparation) used LORETA to estimate the current distribution associated with *normal* sentence processing (rather than anomalous sentences, which has been the strategy in previously published work) within two critical time windows: the window associated with the N400 component (during which the brain is most robustly sensitive to conceptual aspects of the stimulus) and the window associated with the P600 effect (during which the brain is most robustly sensitive to syntax or, more generally, patterned sequences). The LORETA solutions indicated a posterior distribution for the N400 window (the temporoparietal region, BA 39 and 40), and an anterior distribution for the P600 window (the left inferior frontal gyrus, BA 45 and 47). If the posterior and anterior streams really do mediate certain crucial conceptual and syntactic aspects of word combination, then we would expect to see differences in how words from different grammatical classes engage these streams. Both nouns and verbs should engage the conceptual processing stream, as both types of word are conceptually rich. However, because verbs (in configurational languages like English) specify the structure for the clauses in which they appear, one should expect verbs to engage the anterior processing stream to a greater extent. This prediction was verified: During the N400 window, nouns and verbs both strongly engaged the posterior stream. During the P600 window, however, verbs engaged the anterior stream to a much greater degree than did nouns.

4. Interactions between the processing streams

Assuming that separable processing streams mediate syntactic and conceptual aspects of word combination, the question arises as to how these streams interact during sentence comprehension. This interaction must occur in real time, as a person is reading or listening to a sentence. *A priori*, it seems likely that ERPs will be the most useful tool for studying interactions between the streams. This follows because ERPs (unlike hemodynamic neuroimaging methods) provide dynamic measurement of a dynamic process, and are also differentially sensitive to events occurring within the two streams.

Language processing models have been deeply influenced by the "syntax-first" assumptions of generative linguistics (Chomsky, 1986). A standard assumption has been that comprehension is controlled by an initial stage of purely syntactic processing (Ferreira & Clifton, 1986; Fodor & Ferreira, 1988). As words arrive in the linguistic input, they are rapidly organized into a structural analysis by a process that is not influenced by semantic knowledge. The output of this syntactic process then guides semantic interpretation. This model has been given a neurobiological instantiation by Friederici and her colleagues (Friederici, 2002). They claim that the LAN effect reflects the operation of a rapid, reflexive syntactic processor that precedes semantic analysis (reflected in the N400 component). The P600 effect is claimed to reflect a "reanalysis" of a syntactic string when the sentence is ungrammatical, or when the comprehender chooses the wrong parsing option when confronted with syntactic ambiguity (for a critical assessment of these claims, see Osterhout et al., 2004).

However, the syntax-first processing theory seems at odds with the massively parallel and highly interactive nature of computation in the brain (Fuster, 1995). This general principle of neurobiological design fits better with a second class of psycholinguistic models, a diverse

family of models often referred to as *constraint-based models*. These models posit a probabilistic constraint-satisfaction process in which syntactic knowledge is only one of a number of constraints on interpretation (Trueswell et al., 1994). But the implicit assumption in these models (as for the syntax-first models) is that unless syntactic cues are indeterminate, syntax always controls the direction of processing.

In one of the few studies to dynamically study the real-time interaction of the two processing streams, Kim and Osterhout (2005) presented anomalous sentences that began with a passive structure, for example, *The mysterious crime had been solving ....* . The syntactic cues in the sentence require that the noun *crime* be the Agent of the verb *solving*. If syntax drives sentence processing, then the verb *solving* would be perceived to be *semantically anomalous*, as crime is a poor Agent for the verb *solve,* and therefore should elicit an N400 effect. However, although crime is a poor Agent, it is an excellent Theme (as in *solved the crime*). The Theme role can be accommodated simply by changing the inflectional morpheme at the end of the verb to an active form ("The mysterious crime had been *solved* . . ."). Therefore, if meaning drives sentence processing in this situation, then the verb *solving* would be perceived to be in the wrong syntactic form, and should therefore elicit a P600 effect. Kim and Osterhout observed that verbs like *solving* elicited a P600 effect, showing that a strong "semantic attraction" between a predicate and an argument can determine how words are combined, even when the semantic attraction contradicts unambiguous syntactic cues. Conversely, in anomalous sentences with an identical structure but with no semantic attraction between the subject noun and the verb (e.g., The envelope was *devouring* . . ."), the critical verb elicited an N400 effect rather than a P600 effect. These results show quite clearly that semantics, rather than syntax, can "drive" word combination during sentence comprehension (for related work see Kolk et al., 2003, Kuperberg et al., 2003, and Hoeks et al., 2004).

This method permits detailed investigation of interactions between the two processing streams. For example, because semantic attraction is almost certainly a continuous variable, there must be a "tipping point" (i.e., some amount of semantic attraction) at which the semantics "wins" and the syntax "loses." For example, the introduction of syntactic complexity or ambiguity may weaken the syntactic processing stream, thereby increasing the impact of semantic-thematic factors (Kuperberg et al., in press; Weckerley and Kutas, 1999; see also Ferreira, 2003). Similarly, it might be possible to strengthen the syntactic stream to make it impervious to the effects of semantic attraction. We are currently examining this possibility using a "syntactic priming" paradigm, in which we precede the critical sentence (e.g., *The mysterious crime had been solving* . . .) with sentences that are unrelated in meaning to the critical sentence but which have the same syntactic form. Preliminary data suggest that the syntactic stream can in fact be sufficiently strengthened to make it resistant to even strong doses of semantic attraction.

5. Neural circuits, evolution, and genetics

Let's assume for the moment that the combinatorial properties of sentence comprehension are in fact enabled by two processing streams: An anterior stream processes patterns of sequences that occur over time, with the patterned sequences that comprise the syntax of a human language representing just one particularly salient manifestation of this stream. A posterior stream is crucial for combining words at a conceptual level.[6] These streams run in parallel and are, under some circumstances at least, highly interactive. A truly satisfying understanding of the combinatorial aspects of language would provide answers to questions such as these: Exactly where in the brain are the neural circuits that mediate these processing streams, and how are they organized? How and when did they evolve? What genetic mechanisms might account for the species-specific aspects of human language, in particular its combinatory power? We will argue here that compelling (albeit speculative) answers to each of these questions are readily available, and that the answers are grounded in comparative analyses.

Do we know of an anterior neural circuit that mediates patterned sequence processing in a communicative system? The answer is yes. Songbirds rely on a specialized cortical-basal ganglia-cortical loop to learn, produce, and perceive birdsong (Brenowitz & Beecher, 2005). Disruptions to this circuit disrupt the sensorimotor learning needed to acquire song, and also the sequencing skills needed to produce and properly perceive it. Recent advances in understanding the anatomical, physiological, and histochemical characteristics of this circuitry have revealed a remarkable homology between birds and mammals (Doupe, Perkel, Reiner, & Stern, 2005). The homologous circuit in human and nonhuman primates involves loops connecting many regions in the frontal cortex to the basal ganglia. Afferents from the frontal cortex densely innervate the striatum of the basal ganglia, which also receives inputs from many other areas of the cortex. The striatal output then travels back to the same areas of the frontal cortex via the thalamus, forming a closed loop. The striatum seems to control behavioral sequencing in many species (Aldridge & Berridge, 1998; Graybiel, 1997, 1998). Spiny neurons, the principal cells of the striatum, have properties that make them ideal for recognizing patterned sequences across time (Beiser, Hua, & Houk, 1997). Damage to this loop in primates produces problems with motor and cognitive skills that require planning and manipulating patterns of sequences over time (Fuster, 1995). The striatum is also a major site for adaptive plasticity (Graybiel, 2004). All of these observations lend plausibility to the notion that the basal ganglia play a role in the syntax of human language (see also Lieberman, 2000).

Given the striking homologies between birds and mammals with respect to the cortical-basal ganglia circuitry, it is probably not coincidental that the acquisition of human language and birdsong have compelling parallels (Bolhuis & Gahr, 2006; Doupe & Kuhl, 1999). Humans and songbirds learn their complex, sequenced vocalizations in early life. They similarly internalize sensory experience and use it to shape vocal outputs, through sensorimotor learning and integration. They show similar innate dispositions for learning the correct sounds and sequences; as a result, humans and some species of songbird have similar critical periods for vocal learning, with a much greater ability to learn early in life (Brenowitz & Beecher, 2005). These behavioral parallels are what one would expect, if both species rely on a similar neural substrate for learning and using their communicative systems.

Relevant genetic evidence is also available. The much-discussed *FOXP2* gene is similarly expressed in the basal ganglia of humans and songbirds (Teramitsu, Kudo, London, Geschwind, & White, 2004; Vargha-Khadem, Gadian, Copp, & Mishkin, 2005). *FOXP2* mutation in humans results in deficits in language production and comprehension, especially aspects of (morpho)syntax that involve combining and sequencing linguistic units (Vargha-Kadham et al., 2005). One of the neurobiological effects of the mutation is a 50% reduction in the gray matter of the striatum (Vargha-Kadham et al., 2005). Perhaps, then, the combinatorial aspects of human language were enabled by the preadaptation of an anterior neural circuit that has been highly conserved over evolutionary time and across species, and by a genetic mutation in this circuit that doubled its computational space.

Comparative analyses might also be useful when attempting to identify the neural circuits underlying the posterior stream, and for understanding the nature of the interactions between the two streams. The temporoparietal cortex in humans appears to correspond to polymodal association areas in the monkey, which could plausibly act as the neural substrate of relevant polymodal conceptual representations and processes (Fuster, 1995). Furthermore, the temporoparietal polymodal areas are directly connected to the frontal areas putatively involved in sequence processing by long fibers that are part of the uncinate fasciculus (Fuster, 1995). These connections are robust and reciprocal and provide an obvious mechanism for interaction between the two streams. Of course, this characterization is highly speculative.

6. Conclusions

How are words combined during sentence comprehension? The evidence we reviewed here suggests several conclusions. Different sets of processes combine words syntactically and semantically. The two processing streams operate in parallel but independently most of the time; this enables each stream to pursue an internally attractive analysis even when it is inconsistent with the output of other processes (although usually the two streams converge on the same result). But under certain conditions, either stream can "take charge" of word combination, forcing the other stream to do its bidding. More speculatively, we have suggested that the syntactic processing stream depends on the preadaptation of a highly conserved anterior cortical-basal ganglia circuit for processing patterns of sequences, rather than on language-specific neural circuits that evolved in humans. The relevant conceptual processing may depend on a highly conserved posterior circuit that evolved for representing some aspects of conceptual knowledge.[7]

This account is consistent with several known principles of neurobiological design. Neural circuits tend to be organized hierarchically along the nerve axis, from spinal cord to cortex. They are massively parallel and interactive. They act as the functional unit of evolution; one would therefore expect them to be conserved across species rather than invented out of thin air in humans. "Learning" in neural circuits is generally associative. The model we are proposing here has all of these elements.[5] By contrast, the standard models described throughout this chapter have none of them: These models assume a purely cortical representation for language, serial and modular processes, species-specificity of relevant neural circuits, and a strong belief that associative processes cannot account for language. All of which might be true. But if so, then the neurobiology and evolution of human language remain deeply mysterious.

An advocate of the standard story is likely to object to these conclusions. Surely the syntax of human language is too complex (too highly structured, too recursive, too creative) to be modeled as a simple patterned sequence processor that relies on associative learning mechanisms. In fact, the explanatory burden placed on rule-based, recursive syntax has diminished over recent decades. Modern grammars tend to be lexicalist in nature; that is, much of the knowledge relevant to sentence structure is stored in the lexicon with individual words, rather than being computed by abstract phrase structure rules. Recursion, while clearly a characteristic of human language, is much more limited in actual language usage than would be predicted given the standard model. And, because conceptual knowledge has its own structure (Jackendoff, 1990), it seems plausible that some of the burden for structuring the input rests with the conceptual stream (Jackendoff, 2002). Indeed, this type of influence is precisely what we have recently demonstrated (Kim & Osterhout, 2005; Kuperberg et al., 2003). Thus, multiple theoretical developments converge neatly with the idea that human syntax is processed by a sequence processor relying on associative learning, and indeed sequence learning mechanisms such as hidden markov models and simple recurrent networks are shown to acquire grammatical knowledge and simulate human grammatical behavior (Kim, Srinivas, & Trueswell, 2003). The computational properties of the basal ganglia-frontal cortex circuit are well suited for implementing that type of computational model (Dominey, 1997). The seemingly unique characteristics of human syntax might be partly due to the effects of the human version of the FOXP2 gene, which drastically increased the computational space in this circuit.

We are not claiming that all of the available evidence (or even all of that reviewed in this chapter) is consistent with our conclusions. That is clearly not the case. For example, not all patients with agrammatism have obvious damage to the basal ganglia, and functions other than conceptual processing activate the polymodal areas in temporoparietal cortex. Even so, we think it is essential to try to construct a neurobiologically grounded explanation of sentence comprehension. Part of the problem, as we see it, is that the standard procedure in the deficit and neuroimaging fields (at least as it relates to language studies) has been to take a detailed functional model as a given, and to attempt to use the resulting deficit or imaging data to construct a neurobiological

model that can implement the functional model. In our opinion, this strategy has not led to many genuine advances in our understanding. We believe that the data from these methods needs to be put in a larger context, one that includes consideration of the principles of neurobiology, genetics, and evolution. For example, neuroimaging work has generated many conflicting functional hypotheses concerning the roles of various regions of the frontal cortex during language processing. These hypotheses are usually motivated and evaluated in the context of other neuromaging studies on language or related cognitive functions. Only occasionally are they discussed in the larger context of cortical-basal ganglia neural circuits. But a careful consideration of the neural circuits is absolutely central to discerning the function of some patch of cortical tissue.

Finally, this account of the neurobiology of sentence comprehension is not new. In an amazingly prescient paper, Karl Lashley (1951) proposed a similar model even though much of the evidence favoring it had yet to be discovered. More recently, a number of theorists have advocated for these or related claims (Aldridge & Berridge, 1998; Dominey, 1997; Fuster, 1995; Grossman, 1999; Jackendoff, 2002; Lieberman, 2000, Ullman, 2001).[8] What has changed, in our view, is the quantity and quality of evidence, from diverse fields, that converges on the same explanation for the remarkable combinatorial powers of human language.

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Footnotes

1. Streams of processing operate differently from the "modules" proposed within influential "syntax-first" psycholinguistic models (Frazier & Clifton, 1986). These modules are serially ordered rather than parallel (grammatical analysis precedes semantic interpretation) and informationally encapsulated rather than interactive (grammatical analysis is not influenced by meaning). As we will see, modular and streams-based approaches can lead to very different ways of predicting and explaining brain-based data.

2. To date, MEG and TMS have not been used extensively to study sentence comprehension. We therefore focus on other methods in this review.

3. The difficulty associated with characterizing the functional loss that results from brain lesions is due, in part, to the fact that we do not yet know the neural or cognitive architecture of language in neurologically intact people.

4. More recently, Koelsch (2005) and colleagues report that unexpected switches from one musical piece to another also elicit an N400-like effect. This result stretches the set of antecedent conditions known to modulate the N400, although the theoretical implications are uncertain at the moment.

5. Although we and other recent reviewers (Van Petten & Luka, 2006) conclude that the primary sources of the "semantic integration" N400 effect are located in the left temporal lobe, another reviewer (Marinković, 2004) concludes that the primary sources are bilaterally present in temporal and prefrontal areas. We tend to agree with Van Petten and Kutas that the extant literature strongly supports a temporal source but not significant frontal involvement. For further comment on this debate, see Osterhout et al., 2004.

6. Our idea here is that the posterior stream might be particularly important for aspects of sentence processing that involve the combination of meanings. We recognize that conceptual processing in its entirety is much more than this and probably engages a large and widely distributed network of neural circuits.

7. We should explicitly note that other theorists have proposed that the basal ganglia-frontal cortex loops subserve semantic aspects of language comprehension, rather than or in addition to syntactic aspects (Copland, 2003; Crosson, 1985; Longworth et al., 2005; Wallesch and Papagno, 1988).

8. Our proposal differs a bit from some similar proposals. For example, Ullman (2001) makes a distinction between an anterior "procedural" rule-based system that is mediated by cortico-basal ganglia circuits, and a posterior "declarative memory" system that is mediated by hippocampal-temporal lobe circuits. One difference is that in Ullman's model the anterior circuit mediates language-specific rules of combination. In our model, the anterior circuit is a general patterned sequence processor that learns via associative processes.

Furthermore, Ullman (2001) focuses specifically on the distinction between regular and irregular past tense forms of verbs, and claims that the anterior circuit computes the rule-governed form of regular verbs, whereas the posterior circuit retrieves the irregular form from memory. The evidence to support this specific claim is mixed (cf. Longworth et al., 2005). We are suggesting here that some (or many) of the combinatorial aspects of sentence comprehension rely on the putative anterior circuit but do not have specific suggestions about which aspects those might be, given the paucity of relevant evidence.

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