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GENES AND LANGUAGE

Why Should One Expect Genes to Play an Important Role in Language?

Unlike offspring of any other species, ordinary human children routinely acquire complex language, characterized by open-ended vocabularies and productive syntax. This cannot be a result of input alone because juveniles of other closely related primate species, such as chimpanzees, do not develop human-like languages even with extensive tutelage (Terrace, Petitto, et al. 1980). From the perspective of biology, the question is not *whether* genes play roles in language but *how* (Fisher and Marcus 2006).

Clearly, the words and grammar that are specific to any particular language are learned through exposure to appropriate models. Nevertheless, the peculiar human capacity to acquire and use language depends on a rich mixture of neural systems that must be biologically constrained. These include mechanisms that need to simultaneously coordinate **SYNTACTIC**, **SEMANTIC**, **PHONOLOGICAL**, and **PRAGMATIC** representations with one another, with motor and sensory systems, and with both speaker's and listener's knowledge of the world. The functional properties of the relevant neural systems are largely determined by the cellular architecture of the human brain, which is itself the product of ongoing interactions between genes and the environment (Marcus 2004). Genes contribute to the birth, migration, differentiation, patterning, and connectivity of neurons during embryogenesis and development, and they continue to contribute to online functions in the mature brain, for example, by mediating changes in the strengths of connections between neurons. It is likely that hundreds or even thousands of genes participate in the development and maintenance of the neural systems that underlie language, some in ways that may be tailored to linguistic functions, others (like housekeeping genes that govern the basic metabolic processes of all cells) that clearly are not.

As yet, it is unknown how many of the genes in the human genome are closely tied to language, but studies of developmental syndromes that primarily disrupt speech and/or language skills give strong reason to believe that such genes are there to be found (Fisher, Lai, et al. 2003). Speech and language disorders are repeatedly observed to cluster in families, and twin studies indicate that they are highly heritable (Bishop 2001). In recent years, geneticists have successfully located chromosomal sites within the human genome that are likely to harbor genetic risk factors involved in developmental language disorders (e.g., The SLI Consortium 2002). Moreover, they have even been able to zero in on a specific gene, *FOXP2*, that is implicated in one particular disorder affecting speech and language (Lai, Fisher, et al. 2001).

FOXP2: What Is It and How Was It Discovered?

The *FOXP2* gene, found on human chromosome 7, codes for a special type of regulatory protein, technically known as a forkhead-box (or FOX) transcription factor. This class of proteins helps govern when and where genes are expressed (switched on and off) during embryogenesis, in postnatal development, and in the mature organism (Lehmann, Sowden, et al. 2003). Each FOX protein contains a special structure, called a forkhead-box domain, which enables it to bind to the DNA of a target gene and affect how much of the product of that target gene is made in the cell. Transcription factors like these may affect many downstream targets in chorus and, thus, represent central components of gene regulatory networks that are important for implementing developmental programs, allowing cells to respond to signals, and so on.

The discovery of the human *FOXP2* gene originated in studies of a large three-generational family (the KE family) suffering from a rare form of speech and language impairment (Hurst, Baraitser, et al. 1990; Gopnik and Crago 1991). The disorder is characterized primarily by severe difficulties in the learning and production of sequences of mouth movements that are necessary for fluent speech, usually referred to as *developmental verbal dyspraxia* or *childhood apraxia of speech* (Vargha-Khadem, Watkins, et al. 1998). Affected individuals simultaneously display problems in a wide range of language-related abilities, in both oral and written domains, with impact on receptive as well as expressive skills (Watkins, Dronkers, et al. 2002; Vargha-Khadem, Gadian, et al. 2005). All 15 of the affected people in the KE family have inherited a mutation altering a single nucleotide letter in the DNA code of the *FOXP2* gene (Lai, Fisher, et al. 2001). This change affects the structure of the encoded FOXP2 protein and prevents it from functioning properly (Vernes, Nicod, et al. 2006).

Although the mutation in question is private to the KE family, different mutations disrupting FOXP2 function have been discovered in other families, showing comparable problems with speech and language acquisition (MacDermot, Bonora, et al. 2005). In all cases identified thus far, the mutations have been heterozygous; that is, people with the disorder have a mutation in only one copy of *FOXP2*, while the other copy is intact. (Humans are diploid organisms, carrying two copies of every gene, one inherited from the father, the other from the mother, with a few exceptions such as the genes on the sex chromosomes.) The consistent observations of heterozygosity in different cases of FOXP2-related disorder suggest that affected people have reduced amounts of working FOXP2 protein in brain circuits that are important for speech and language. Therefore, the

amount (dosage) of FOXP2 may be a critical factor in the proper development of speech and language skills.

Does That Make *FOXP2* the "Language Gene"?

No. Although studies of people carrying damaged versions of *FOXP2* are consistent with a role (or roles) in the development and/or processing of language, it is already apparent from genetic studies that no single gene is exclusively responsible for this distinctive human trait. Indeed, *FOXP2* is implicated (thus far) only in one rare form of disorder, and not mutated in people diagnosed with more common variants of **SPECIFIC LANGUAGE IMPAIRMENT** (SLI) (Newbury, Bonora, et al. 2002). Instead, most developmental language disorders are likely to be multifactorial: the product of multiple genetic risk factors, their interactions with one another and interactions with the environment (Fisher, Lai, et al. 2003). It is also worth noting that mutation of *FOXP2* impairs not only aspects of speech and language but also aspects of nonlinguistic orofacial motor control (Watkins, Dronkers, et al. 2002; Vargha-Khadem, Gadian, et al. 2005).

More broadly, given what we know about the fundamentals of genetics, developmental biology, and neuroscience, it is highly unlikely that there is a single human-specific gene whose sole purpose is to endow our species with the capacity to acquire language. Individual genes do not specify particular behavioral outputs or aspects of cognitive function. Rather, they contain the codes for assembling individual molecules that act in a highly interactive fashion with other molecules in order to build and maintain a working human brain (Marcus 2004). Often, a gene will have a primary function that is very clearly defined at the cellular level – for example, by encoding an enzyme, structural protein, ion channel, signaling molecule, or receptor – but the pathways that link the gene to higher-order brain function will nevertheless be complex, indirect, and difficult to disentangle (Fisher 2006).

The "language gene" shorthand is also misleading because most, if not all, of the genes that are involved in language are likely to play other roles, elsewhere in the brain and/or in other tissues of the body. The expression of *FOXP2* is not confined to classical language-related regions of the cortex, or even to the brain. Instead, it extends to additional brain structures, such as the **BASAL GANGLIA, THALAMUS,** and **CEREBELLUM** (Lai, Gerrelli, et al. 2003), and to other parts of the body (e.g., the lungs [Shu, Yang et al. 2001]); it also has close counterparts in all vertebrates, as discussed in the section on evolution. In sum, *FOXP2* can properly be a called "a gene that participates in language" but not "the language gene" or even a gene that participates exclusively in language.

How Representative Is *FOXP2*? Are Other Genes Involved in Language Likely to Act in Similar Ways?

It is difficult to say for sure; thus far, the *FOXP2* gene represents the *only* known example where point mutations have been linked to a developmental disorder which primarily affects speech and language. However, since disruptions of *FOXP2* are found in only a very small subset of people with language-related disorders (Newbury, Bonora, et al. 2002; MacDermot, Bonora, et al. 2005), it is clear that there must be other genetic effects that remain to be discovered.

Genetic studies of typical forms of specific language impairment have identified other genomic regions that are likely to be relevant to language, and researchers are focusing considerable attention on those chromosomal sites in the hope of pinning down particular genes (e.g., The SLI Consortium 2002). For developmental dyslexia, a disorder primarily characterized by reading disability, but underpinned by subtle persistent deficits in language processing, it has been possible to home in on several candidate genes (*DYX1C1*, *KIAA0319*, *DCDC2*, and *ROBO1*). These genes differ from *FOXP2* in that there have been no specific causal mutations identified – instead, it is thought that the increased risk of dyslexia stems from as-yet unknown variants in the *regulatory* parts of those genes that govern their expression (Fisher 2006). Nevertheless, there are some striking parallels with *FOXP2*; each of the dyslexia candidate genes shows widespread expression patterns in multiple circuits in the brain, and each is active in additional tissues, not only the brain. None of the genes is unique to humans; for example, highly similar versions of each are found both in other primates and in rodents. At this stage, there is little understanding of why alterations in these genes should have relatively specific effects on reading abilities, although their basic neurobiological functions are beginning to be defined; three of the genes (*DYX1C1*, *KIAA0319*, and *DCDC2*) have been linked to neuronal migration, and the fourth (*ROBO1*) codes for a receptor protein involved in signal transduction, which helps regulate axon/dendrite guidance.

At the time of writing, there are indications that alterations in gene dosage may emerge as a general theme underlying overt speech/language deficits. For example, it has been recently shown that duplications of a specific region on chromosome 7 (far away from the site of the *FOXP2* gene) can cause speech deficits (Somerville, Mervis, et al. 2005). What is especially interesting about this finding is the fact that the relevant part of chromosome 7, which contains several different genes, corresponds to the region that is most commonly *deleted* in cases of Williams syndrome, a well-studied disorder in which language skills can be relatively well preserved as compared to other abilities. In other words, while deletion of that part of chromosome 7 (i.e., reduced gene dosage) tends to spare language, duplication of this same set of genes (increased gene dosage) leads to speech disruptions. Similarly, there is evidence to suggest that the number of functional copies of a chromosome 22 gene called *SHANK3*, recently implicated in autism spectrum disorders, may be critical for speech development (Durand, Betancur, et al. 2007). Language, like many aspects of biology, is likely to depend on a precise balance among many different molecules.

What Can Genes Tell Us about the Evolution of Language?

Genes, like species, are the product of the process that Darwin called "descent with modification." Each gene has an evolutionary history, with its current function a modification of earlier functions. To the extent that the language system is the product of descent with modification, most genes that are associated with language can be expected to have counterparts in nonlinguistic species. As such, comparisons of gene sequences and expression patterns in different species can help cast light on language evolution, identifying which of the relevant neurogenetic pathways are shared with other species and which have been modified

on the lineage that led to modern humans (Fisher and Marcus 2006).

FOXP2 again appears representative in this regard. Following the discovery of the gene, molecular studies have shown that it is present in similar form in many vertebrates, including mammals, birds, reptiles, and fish, where it is expressed in corresponding regions of the brain to those observed in humans (reviewed by Vargha-Khadem, Gadian, et al. 2005; Fisher and Marcus 2006). On the basis of such data, it appears that *FOXP2* is evolutionarily ancient, shared by many vertebrate species, regardless of speech and language ability, where it may have conserved functions in brain circuits involved in sensorimotor integration and motor-skill learning (Fisher and Marcus 2006). For example, the striatum in the **BASAL GANGLIA** is a conserved site of high *FOXP2* expression, which shows reduced gray matter density in humans carrying *FOXP2* mutations (Vargha-Khadem, Gadian, et al. 2005). It is intriguing that in songbirds, changes in expression of the gene in striatal Area X – a key nucleus of the brain system involved in song learning – appear to relate to alterations in vocal plasticity (see White, Fisher, et al. 2006).

Despite such notable conservations across distantly related species, a comparison of the locus in different primates has demonstrated that there was accelerated change in the FOXP2 protein sequence during human evolution, most likely due to positive selection (Enard, Przeworski, et al. 2002). Mathematical analyses of genomic sequences from diverse human populations suggest that the version of FOXP2 now ubiquitous in modern humans arose within the last 200,000 years, concordant with several archaeological estimates of the time of emergence of proficient spoken language (ibid.). We may never know for certain why these modifications spread throughout the population, but it seems plausible that they proliferated due to some advantage inherent in enhanced vocal communication, perhaps achieved through modification of pathways already involved in motor-skill learning. Still, this does not mean that changes in FOXP2 were the sole reason for the appearance of speech and language, even if they did represent an important factor in the evolution of human communication.

What's on the Horizon?

Technological advances provide one reason for optimism. Techniques for characterizing genes and genomes are quickly becoming more rapid, cost-effective, and efficient – which can only speed up ongoing searches for genes involved in speech and language. For example, it has recently become possible to simultaneously screen hundreds of thousands of genetic markers in people with a disorder of interest, and compare the data to those obtained from a control set of unaffected individuals. Given an adequate sample size, this kind of approach could uncover subtle genetic differences that are correlated with developmental language disorders. Before long, it will even be feasible to sequence the entire genome of every person participating in a study. We might also expect to see developments in the ways we can image gene expression patterns in the human brain, with the hope that it may one day be possible to observe on-line changes in gene expression in neural circuits during language processing. Still, it is also clear that we will need major conceptual advances in order to make sense of the vast quantities of sequence and expression data that will soon emerge, both in terms of sheer data analysis and in terms of relating these data to linguistic functions.

Another exciting prospect is the use of genetic manipulation in order to find out more about the functions of genes that are involved language, for example, by examining the function of nonhuman counterparts to those genes (White, Fisher et al. 2006). In this way, individual genes may provide the first molecular entry points into neural pathways involved in human communication, and a direct way to understand how the twin processes of descent and modification led to the remarkable and uniquely human faculty for complex language.

– Gary F. Marcus and Simon E. Fisher

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GESTURE

"... no ideas, just irritable mental gestures."

(remark attr. to Lionel Trilling,
New York Times, June 21, 2006, A1)

What Is Gesture?

Lionel Trilling, in this non-motto, invokes an all-too-common view of gesture. The very phrase "hand waving" suggests triviality. But let us imagine Trilling's own gesture. It would have been (we can predict) what Cornelia Müller has called the "palm up open hand" (PUOH), the hand seeming to hold a "discursive object," holding, in fact, Trilling's view. These kinds of gestures have been linked to the *conduit metaphor* – the metaphor whereby language or cognition is a container holding some content.

The PUOH is also one of a species of gesture termed by Kendon "gesticulation," one of several kinds of gesture he distinguished and that I arranged on "Kendon's Continuum":

Gesticulation → Speech-Linked → Pantomime → Emblems → Sign Language

Even though gesticulation is only one point on the continuum, in storytelling, living-space descriptions, academic discourse (including prepared lectures), and conversations, gesticulation is the overwhelming gesture type – 99+ percent of all gestures – and it is the gesture offering the greatest penetration into language itself. As one moves from gesticulation to **SIGN LANGUAGE**, two reciprocal changes take place. First, the degree to which speech is an obligatory accompaniment of gesture *decreases*. Second, the degree to which gesture shows the properties of a language *increases*. Gesticulations are obligatorily accompanied by speech but have properties unlike language. Speech-linked gestures, such as "the parents were fine but the kids were [finger across throat]," are also obligatorily performed with speech but relate to speech as a linguistic segment – sequentially, rather than concurrently, and in a specific linguistic slot (standing in for the complement of the verb, for example). Pantomime, or dumb show, by definition is not accompanied by speech. Emblems such as the "OK" sign have independent status as symbolic forms. Signs in American Sign Language (ASL) and other sign languages are *not* accompanied by speech and while simultaneously speaking and signing is possible for ASL-English bilinguals, this is not typical, and the languages themselves have the essential properties of all languages.

Clearly, therefore, speech and gesticulations (but not the other points along Kendon's Continuum) combine properties that are unalike, and this combination of unalikes occupies the same psychological instant – a fact of importance for creating an imagery–language dialectic. I use *gesture*, rather than *gesticulation*, in the remainder of this entry.

Figure 1. Gesture combining entity, upward movement, and interiority in one symbol. (Computer art by Fey Parrill.)

Table 1. Gesture-speech binding resists interruption

Domain	Phenomenon
Delayed auditory feedback	Does not disrupt speech-gesture synchrony.
Stuttering	Gesture stroke onsets resist stuttering; stuttering cancels ongoing strokes.
Blindness	Gestures occur when speaking to other blind known as such.
Fluency	Speech and gesture are complex or simple in tandem.
Information exchange	Information seen in gesture recalled as speech, and vice versa,

The *gesture-first* theory of language origin holds that the first form of language consisted largely of gestures, to be later supplanted by speech – an idea going back to Étienne de Condillac in the eighteenth century. Gesture-first has attracted much interest in recent years. A difficulty, however, is that it "predicts" the wrong gestures. The initial gestures would have been speechless pantomimes, nonverbal actions with narrative potential, but not the gesticulations that pose dialectic oppositions to language at the far end of Kendon's Continuum. Pantomime may indeed have been present but, if so, did not lead to the **EVOLUTION** of *speech and gesture units* (growth points). Such units would likely have had their own adaptive value. An implication is that different *evolutionary* trajectories landed at different points along the continuum, reflected today in different forms and timing patterns with speech.