
RECENT GENETIC CONTRIBUTIONS TO THE STUDY OF LANGUAGE

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ABSTRACT. The notion that humans are endowed with certain innate predispositions for language acquisition was revitalized with the consolidation of the generativist linguist paradigm, introduced by Noam Chomsky in the 1950s. In spite of several criticisms, it still constitutes the most extended perspective within the frame of cognitive science. The suggestion of an innate linguistic knowledge has been accompanied by an interest in identifying specific genes related to the faculty of language. This is the purpose of numerous twin, adoption, and linkage studies. However, it has taken a long time to isolate the first actual gene related with language, *FOXP2*. A mutation of this gene produces a speech and language disorder (Lai *et al.*, 2001). Neuroimaging studies have allowed the identification of the brain areas related with the disorder, while genetic studies have described the gene's evolutionary history. In the present work we review some of these studies and analyze the implications that the discovery of *FOXP2* has for the study of language, its neural correlates, and its evolution. We argue that they favor a picture of language as a set of cognitive processes that have different neural correlates, arise from different processes of genetic expression, and are the results of different evolutionary episodes, but function coordinately, allowing our species to develop and use language and verbal communication.

KEYWORDS. Language, gene, *FOXP2*, brain, cognitive neuroscience, language disorder, development, language evolution, communication, fMRI.

INTRODUCTION

There is a widespread, though usually implicit, belief that language is one of the traits that most clearly sets our own species apart from the rest of animals. Language is viewed as a unique cognitive faculty that defines us as human beings. Not surprisingly, thus, researchers from disciplines such as philosophy, psychology, the neurosciences, artificial intelligence, or anthropology have focused their attention on language. In spite of con-

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siderable efforts, some of the most relevant questions in relation to language have still to be satisfactorily answered: Which are the brain mechanisms that support this faculty? Which are its genetic correlates? How did it evolve? To conceive language as a unique or exceptional trait has led different authors to consider that these three aspects—brain organization, genetic substrate and evolution—are also unique or special. The idea that there are brain areas that exclusively carry out language functions and specific genes that contain the instructions to develop language, and that gene and brain systems related with language are exclusively human, are implicit in many studies.

One of the main obstacles for the clarification of these questions is the disagreement among authors regarding the most adequate way to conceive language itself. There are currently two main approaches to the study of language. From the generativist point of view, language is understood as a true “species property” coded by genes (Chomsky 2000; Hauser, Chomsky & Fitch 2002). Moreover, the faculty of language can be considered as an “organ of language,” in the same sense as biomedical sciences refer to “immune system,” “locomotor apparatus,” or “sensory organs,” that is to say, as organs of the human body (Chomsky 2000). From an alternative point of view, functionalism, it is believed that cognitive processes involved in language are intimately related with its communicative and social interaction functions. Additionally, the existence of strictly linguistic innate principles is rejected, and it is assumed that language’s grammatical and social aspects are acquired by means of certain general learning mechanisms and operations (Newmeyer 1991). Some linguists have expressed interest in the reconciliation of both positions, but most are reluctant to draw these positions closer. They really constitute paradigmatic positions that determine, to a great extent, the aspects of language researchers attend to, the kind of data they consider, and the methodology they use.

The purpose of the present work is to examine the extent to which the results from recent studies on the genetics of language can be useful for the construction of a conception of language which is closer to the data, and less influenced by *a priori* theoretical positions. Reaching an unbiased description of language is difficult, if not impossible, given that the notion of a genetic basis of language is more compatible with generativist than with functional perspectives. However, the main discrepancy does not refer so much as to whether the principles that underlie language and its acquisition are innate or genetically coded, but to whether they are language-specific. In any case, to maintain that human beings come to the world with a certain predisposition (general or specific) to acquire language, this is to say, that they possess, at birth, certain cognitive capacities (such as segmenting speech into syllables or discriminating human lan-

guage phonemes) and attentional biases (preference for language stimulation or for the mother's language prosody) that direct the development of any human language, implies placing the substrates of such capacities and biases in certain neural structures constructed and arranged according to information contained in the human genome. However, we are presently far from identifying the genetic correlates of such cognitive biases and operations. Evidences for a genetic predisposition for language development are, for the moment, mainly indirect and have been studied with greater emphasis from the generativist point of view. Let us very briefly review some of these arguments⁵:

1) Universality and specificity: The language faculty is universal, given that all healthy humans develop it, and specific because it is exclusive of the human species⁶.

2) Existence of a specific neural substrate: The language faculty is related *at least partly* with the activity of certain regions of the brain's left hemisphere (in right-handed subjects). This asymmetry seems to be anatomically and functionally predetermined.

3) Existence of a critical period for the acquisition of language: In some instances, it occurs that organisms that are predetermined to manifest a specific trait—such as language in humans, according to chomskyan theses—require the exposure to certain kinds of pertinent stimulation or experiences during a lapse of time which is also predetermined as critical.

4) The "paradox of language acquisition" (or Plato's problem): The principles underlying the functioning of languages are acquired by children at a very early age, in a spontaneous, fast, progressive and effortless way, going through the same stages and with similar rhythms independently of their intelligence, language, culture, or education. This is true in spite of the fact that stimulation is very limited, heterogeneous, incomplete, sometimes erroneous and contradictory (Chomsky 1957, 1972, 1986; Mendivil Giró 2003: 340-355; Lorenzo & Longa 2003: 30-32).

5) Modularity or cognitive specificity: A number of examples of selective cognitive disorders, that is to say, the affectation of specific mental capacities while the rest are left intact, have been described. For instance, people affected by Specific Language Impairment or genetic dysphasia conserve their cognitive capacities unaltered, but manifest very important linguistic alterations (Newmeyer 1997; van der Lely 1997). Conversely, individuals diagnosed with Williams's syndrome have a mean IQ of 60-70, while their linguistic competence is higher than expected.

6) Studies of adopted twins: A good part of the variance in linguistic abilities of people with and without language disorders is explained by genetic factors (Stromswold 2001).

THE GENETIC SUBSTRATE OF LANGUAGE: THE KE FAMILY

The identification of inherited alterations exclusively affecting language functions represents a different kind of evidence. It would allow the direct characterization of the genetic basis of language by relating the activity of a specific gene with certain development processes of neuronal structures that support the language faculty. Although this task is not simple, a language disorder has recently been identified that could permit to relate genes and language more directly. In 1987 the principal of a school for children with speech and language problems recommended a British family, known as the KE family, to allow a genetic specialist to examine their case, given that several members had attended the same school. The members of the family belong to three generations and approximately half of them, fifteen, presented a severe speech and language disorder. The study of the family's structure revealed that the inheritance of the disorder is monogenetic and dominant autosomal. This inheritance mechanism, as opposed to other more complex patterns that result from the interaction of the effects of several genes, is ideal to carry out a study on the relation between genes and language. Let us review some of the main features of the disorder affecting this family.

Cognitive and behavioral characterization

Hurst *et al.* (1990) considered that the affected members of the KE family suffered a developmental verbal dyspraxia, characterized by language and speech problems. On the one hand, regarding the articulation level, researchers observed the existence of articulation problems related with moderate or severe dyspraxia that caused unintelligibility of speech, such as the simplification of consonant groups, omission of initial and final consonants, omission of syllables, and so on. On the other hand, and regarding strictly syntactic competences, Hurst and collaborators detected difficulties in the construction of grammatical sentences, the comprehension of comparative, passive and reduced-relative sentences, problems with the subjects of sentences and semantic mistakes. Alternatively, Gopnik and Crago (1991) catalogued the family KE's disorder as "developmental dysphasia," characterized by difficulties in the acquisition and use of morphological rules: the affected members acted as if they did not know the rules and treated each word as an exception, as irregular. This was unrelated, according to Gopnik and Crago (1990), with a lower intellectual capacity or the absence of the concept of number.

The thorough studies carried out by Vargha-Khadem and Passingham (1990), Vargha-Khadem *et al.* (1995) and Watkins, Dronkers and Vargha-Khadem (2002) revealed that the disorder did not only affect language, but also motor and articulatory capacities related with speech and, to a certain extent, other cognitive capacities. In addition to the problems cited

by Hurst *et al.* (1990), deficits in the repetition of words and sentences and word comprehension were added. In sum, Watkins and collaborators catalogued the alteration as a "verbal dyspraxia." ⁷ Regarding the morpho-syntactic alteration, they believed it was similar to that observed in Specific Language Impairment and that the difficulties and mistakes made by the affected members involve both regular and irregular morphology, and so it is not related with specific morphosyntactic rules. Lai *et al.* (2001) and Fisher, Lai and Monaco (2003) pointed out severe problems both in the selection and sequencing of fine orofacial movements necessary for articulation (and unrelated to facial musculature anomalies) in language processing (phoneme sequencing), grammatical abilities (linguistic competence) and in the production and comprehension of syntactic structures and flexion. Written language is also altered. Fischer *et al.* (2003) considered that the non-verbal IQ in the KE family might even be lower than the verbal IQ, but this is also present in unaffected members, while only the verbal IQ of the affected members is clearly below the general mean.

Genotypic characterization

Fisher and collaborators (1998) were able to narrow the location of the gene responsible for the disorder to a small interval of chromosome 7, the locus known as SPCH1, in the region 31 of the long arm of such chromosome. Lai *et al.* (2001) explored the affected members' chromosome 7 in search of the SPCH1 allele, candidate to be the cause of the language and speech anomalies. Finally, the gene related with the disorder was isolated. It turned out to code for a member of a family of regulating proteins, or transcription factors, with a common structural feature: the three-dimensional shape "helix–turn–helix motif", with two helices separated by a short turn that allows them to recognize certain DNA sequences. This observation led the gene to be included in the *FOX* (forkhead box) gene family, and according to current nomenclature was named *FOXP2* (Kaestner *et al.* 2000). *FOX* genes code transcription factors with diverse functions in cellular proliferation and differentiation mechanisms and in the transcription and translation signals. Many of the proteins belonging to this family play an important role in the regulation of embryogenesis in species as diverse as mice and human beings.

Lai and collaborators (2001) demonstrated that the *FOXP2* sequence in the KE family presented a substitution of a guanine (G) by an adenine (A) nucleotides, in exon 14. This mutation present in the KE family is translated into the substitution of the aminoacid arginine for histidine, located in helix 3, a critical region involved in binding to the promoters of other genes that facilitate its transcription. The changes in this region alter the protein's function in the regulation of DNA expression.

Neurobiological characterization

A series of neurobiological studies has allowed the structural, functional and ontogenetic description of the brain correlates of the disorder affecting some of the members of the KE family. The most consistent result of structural studies refers to the reduction of the gray matter in the caudate nucleus, although this is not the only region with a different volume of grey matter regarding control participants (Vargha-Khadem *et al.* 1998; Watkins, Vargha-Khadem *et al.* 2002; Benton *et al.* 2003). However, although the group of affected members has an overall reduced volume of gray matter in the caudate nucleus regarding unaffected members, this is not the case in each of the affected members (Watkins, Vargha-Khadem *et al.* 2002). This means that there are affected members whose caudate nucleus has a similar volume to that of their healthy relatives or control subjects. Thus, the reduction in size of the caudate nucleus cannot be taken as a determinant correlate of the disorder.

Functional studies have supported the subcortical focus of the main circuits altered by the mutation, in addition to extending its influence to certain cortical areas. Vargha-Khadem and collaborators (1998) showed, by means of Positron Emission Tomography, that during word repetition there was an overactivation of the left caudate nucleus in the affected members of the family. Later, Liégeois *et al.* (2003) performed an experiment using Functional Magnetic Resonance, and found an underactivation of the left hemisphere Broca's area and the putamen bilaterally—a structure closely related to the caudate nucleus—during linguistic tasks.

Regarding the ontogenetic dimension, Lai *et al.* (2003) and Ferland *et al.* (2003) have described this gene's pattern of expression during embryonic and fetal development in the brain of mice and humans. Both teams found a great conservation in the timing and distribution in tissues during the beginning of the expression of the gene in both species' central nervous system. As development progressed, the expression went from being uniform and diffuse to being restricted to certain locations in the brain. The expression of *FOXP2* has been detected in the cerebellum, the thalamus, the caudate nucleus—among other basal ganglia structures— and the spinal cord until the end of early gestation. The structures in which the transcript was found are intimately connected and are related mainly with motor functions. In general terms, the expression of the gene coincides with the regions containing functional or structural anomalies detected by means of the aforementioned neuroimaging studies in the affected KE family members. Specifically, *FOXP2* seems to be involved in the development of corticostriatal and olivocerebellar circuits, including the caudate nucleus and putamen. This might explain the functional and structural anomalies related with orofacial motor problems and linguistic impairments observed in people affected by this gene's mutation.

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This general characterization is limited by a series of issues. In first place, there seems to be only a partial agreement regarding which of the language impairment's symptoms should be considered nuclear and which may be understood as being a consequence of them. Second, although the caudate nucleus comes across as the strongest possible altered brain site from the neuroimaging studies, there are many inconsistent results among them. Third, still it is not at all clear how the different brain anomalies detected by neuroimaging studies relate to the behavioral and cognitive symptoms. However, in spite of these limitations the findings stemming from the identification of the *FOXP2* gene and its involvement in the development of brain circuits related with language functions are, generally speaking, in conflict with a conception of language that postulates a strictly specific or unique neural, genetic and evolutionary substrate. In this section we will discuss how the discoveries associated with *FOXP2* shed light on these three intimately related questions: (i) the genetic basis of language, (ii) its neural correlates, and (iii) its evolutionary history.

The genetic basis of language

Let us deal first with the question of language's genetic correlates. The faculty of language is often understood, from a generativist point of view, as part of human beings' biological inheritance, a component of the human brain or mind that, when exposed to an adequate linguistic stimulation, produces the knowledge of a particular grammar; a computational system that generates linguistic representations of sound-meaning pairs and whose essential feature is recursivity (Chomsky 1988, 1995). The initial state of this faculty, known as Universal Grammar, is considered as the set of principles which are quite specific and beyond a general learning mechanism, and include a series of parameters fixed by experience. This perspective implies that at birth human beings have a rich linguistic knowledge which must somehow be specified in our genes. Some authors that argue against such nativist positions (such as Elman *et al.* 1996) have caricaturized the efforts to identify genetic correlates of the knowledge of language as the search for the "language gene" or the "grammar gene." However, although some psychologists and linguists occasionally seem to expect to find genes related with the faculty of language, they do not normally refer to "the language gene," but to "several language genes."

In the previous section we pointed out some evidences that suggested that *FOXP2* is involved in the construction of brain circuits that sustain certain aspects of language. Does this mean that therefore it is "one of the language genes"? The answer is negative, and the question meaningless in the light of current genetic knowledge, which rejects for most cases the linear mechanical scheme: one gene to one protein to one phenotypic trait.

A discovery that should illustrate this point, and should help us change the way we approach the question of language's genetic bases, is that *FOXP2*, in addition to being expressed in the brain, it is also expressed in specific regions of other tissues during development—including lungs, some organs in the digestive apparatus and the heart—as in various tissues of the adult organism (Shu *et al.* 2001). This is actually coherent with what is known about other transcription factors, many of which carry out diverse functions. On the other hand, the same gene seems to organize the development of similar brain circuits in humans and the rest of mammals. In this sense, the finding that *FOXP2* is involved in the specification of neural circuits related with the vocalization of certain birds which require learning in order to adequately develop their song (Haesler *et al.* 2004; Teramitsu *et al.* 2004) is very suggestive.

Thus, our initial conclusion must be that the first gene reliably related with linguistic processes is not only expressed in the brain, but also in other organs of the body. Additionally, human beings share this gene with many other animals; that is to say, the gene itself is not specific of our species. In any case, it is as senseless to consider *FOXP2* a "language gene" as it would be to consider it a "circulatory gene" or a "digestion gene". Obviously, this conclusion is not contrary to the notion of a genetic basis for language, or that this basis may be very rich. The case of *FOXP2* illustrates that the genetic mechanisms that construct the language organ in human beings: (i) may also be involved in the construction of other organs of the body, and (ii) may be present in other animals.

The neural correlates of language

Let us turn now to the neural organization of language. The classic model, also the one most authors explicitly or implicitly adhere to, considers that there are two areas related with language located in the cortex of the left hemisphere, an inferior frontal region and a superior temporal region, (Broca and Wernicke's areas respectively) and the *arcuate fasciculus*, a tract of fibers that connects those two areas. This scheme, rearticulated during the sixties by Norman Geschwind (1965), constitutes the standard model that has guided research in and out of the neurosciences for almost a century and a half. However, a series of inconsistencies that cast doubts on the model have surfaced during the last two decades. First, it has become increasingly clear that the different aphasic syndromes, the evidence upon which this standard model is built, are not homogeneous entities defined around unchanging symptoms. Second, the linguistic concepts referred to by the model are very poor, and the complexity of the different components and cognitive operations that are involved in the semantic, syntactic, phonological and other systems is not acknowledged. Third, there are often problems at an anatomical level, such as the non-

correspondence between the kind of aphasia and the injured areas specified by the model (Poeppel & Hickok 2004).

These considerations have led researchers to consider that the classical model must be expanded to include other cortical areas previously not recognized as participating in linguistic processing (Bookheimer 2002; Brown & Hagoort 1999; Nobre & Plunkett 1997; Patterson & Bly 1999; Poeppel & Hickok 2004). Furthermore, many of the elements of the neural net underlying different aspects of language are also involved in the performance of other cognitive tasks (Kaan & Swaab 2002), in interaction with other brain regions (Heim *et al.* 2003). For instance, Broca's area, related in the classical model with syntactic aspects, seems to be involved in the processing of musical sequences (Koelsch *et al.* 2004; Patel 2003), the imagination of movement (Binkofski *et al.* 2000) and the perception of the rhythm of movement (Schubotz & von Cramon 2001). However, the classical model still influences the thinking on the neural bases of language. For instance, Sakai (2005) stated that certain regions of the left inferior frontal *gyrus* (specifically the opercular and triangular parts) can be regarded as the brain's "grammar center." This conclusion is drawn from the observation of brain activity in those regions during syntactic decision tasks when compared to verbal short-term memory tasks. Sakai's conclusion overlooks the fact that the resultant activity is the product of the subtraction of two experimental conditions. Thus, areas active in both tasks, which may be relevant for sentence comprehension and verbal short-term memory, appear as inactive. The use of transcranial magnetic stimulation, as summarized by Sakai (2005), does allow showing that these areas participate in grammatical processing; but this does not mean that these are the only regions that do so, other areas might be necessary. Regarding the specialization of these regions for linguistic processing, the fact that they are active during syntactic decision tasks does not preclude them, in principle, from participating in any other number of different tasks. Thus, the appearance of a grammar center could be nothing more than the product of the methods available to study the neural correlates of language, and the *a priori* notion that a certain cognitive operation is related to a certain delimited brain region, and that this region is only related with one cognitive operation.

Moreover, the studies on *FOXP2*, which consistently identify the neurological alteration in the caudate nucleus, lend support to the work of those authors that defend a deeper revision of the classical model of language and propose that certain subcortical structures are also involved in this faculty. From this viewpoint, it is understood that the neural correlates of language consist of two main systems that are, to a certain point, dissociable (Lieberman 2000, 2002; Ullman *et al.* 1997; Ullman 2004). One of those systems, related with verbal working memory, declarative

memory, or lexical storage, is believed to be supported by a cortically distributed net, not a single localized region. This first system includes Broca and Wernicke's areas, but also their right hemisphere homologues and others in premotor and prefrontal cortices. The second system, related with procedural memory, syntactic rules and other sequencing cognitive operations, involves fronto-striatal circuits, with the basal ganglia, especially the caudate nucleus (Ullman 2004: 246), constituting a fundamental piece of this net.

This renewed view of the biological bases of language fits well with the conception of language as a system that allows an unlimited expressive capacity based mainly on two mechanisms: the arbitrary pairing of sounds and meanings, that is to say, words, and the combination of into larger meaningful units, sentences (Pinker 1998, 1999). These could easily correspond with the two aforementioned brain circuits⁸. In sum, the studies related with *FOXP2* suggest a conception of language's brain bases as a widely distributed net of cortical and subcortical regions, many of which participate in the performance of cognitive or motor operations in addition to language.

The evolution of language

One of the greatest traditional problems hampering the study of language evolution has been the lack of data that can be used to ground and contrast hypotheses. In fact this is the motive that led, in 1866, the Linguistic Society of Paris to discourage any effort aimed at clarifying the origin and evolution of language. Although there have been great advances in neurosciences, linguistics and palaeoanthropology, in the century and a half that has gone by since then, we still lack an objective and reliable source of data regarding the evolution of language. The three physical evidences that have most often been used to ground hypotheses regarding this question are: the size of the hypoglossal canal, the position of the larynx and the surface of cranial endocasts. However, it has recently become clear that none of these three kinds of evidence constitutes a reliable indicator of the linguistic capacities of our ancestors (Cantalupo & Hopkins 2001; DeGusta *et al.* 1999; Fitch & Giedd 1999; Fitch & Reby 2001; Gannon *et al.* 1998, 2001; D. Lieberman & McCarthy 1999; D. Lieberman *et al.* 2001; Sherwood *et al.* 2003). Thus, the possibility that the findings related with the gene *FOXP2* may offer useful data to constrain hypotheses concerning language evolution merits attention.

An interesting discovery from the two genetic expression studies that have been carried out (Ferland *et al.* 2003; Lai *et al.* 2003) is the great similarity between this gene's expression patterns in mice and humans. Neither study found specific human locations where this gene is expressed. This homologue expression in humans and mice supports the

idea that this gene has a relevant role in the development of motor circuits in some current mammal species and, at least, in the last common ancestor of humans and mice. The fact that this gene has been involved for millions of years in the development of neural circuits devoted to motor control, together with evidences that it has been subjected to positive natural selection (Zhang *et al.* 2002), suggests that at least part of the brain systems related with language evolved by means of the modification of developmental sequences and patterns of some of our very early ancestors' nervous systems. Thus, language must be understood as the result of natural selection taking advantage of old systems in a new way, obtaining a novel function from pre-existing neural structures.

Additionally, Svante Pääbo's team (Enard *et al.* 2002) carried out a detailed study of *FOXP2*'s evolutionary history that uncovered some clues regarding the moment at which the human form appeared. The fact that the coded protein's versions in chimpanzees, gorillas and macaques are identical, and show a single difference regarding the mouse version, is indicative of the gene's high conservation within the mammal lineage. However, the human version presents two differences in relation to most species of apes. This accelerated evolution in the human lineage reflects the adaptive importance awarded by language. Enard *et al.* (2002) arrived at the conclusion that the human version of the gene got fixed no more than 200 000 years ago, approximately coinciding with the appearance of our own species. This could be interpreted as direct support for the language evolution models that locate its appearance in recent stages of human evolution. However, from the fact that the establishment of neural circuits related with language is altered by the KE family's mutation, it cannot be concluded that more archaic human versions of the gene would have prevented the development of this faculty.

The conclusions rendered by the studies on the evolutionary history of the gene *FOXP2* point in the same line as comparative studies that suggest that some cognitive operations that sustain human language acquisition are shared with other primates (Ramus *et al.* 2000; Hauser *et al.* 2001; Hauser *et al.* 2002). These two independent lines of evidence suggest that some of the cognitive operations and genetic structures that make language possible were present millions of years before the appearance of human beings, while some seem to be specifically human (Fitch & Hauser 2004; Enard *et al.* 2004). Not all cognitive operations, neural structures and genes related with language appeared along the human lineage during the last seven million years as adaptations that increase linguistic efficiency. Thus, before proposing models of language evolution we should aim to clarify which components of modern human language appeared specifically along the human lineage, after hominids separated from panids, and which appeared at earlier stages of primate evolution.

CONCLUDING REMARKS

The evidences uncovered by recent research on *FOXP2*, reviewed in the present work, favor an image of language in which unique features are only a part of an overall system. In fact, they suggest that one of language's main attributes is its integration in larger-scale systems. In the first place, we believe that it cannot longer be held, as Caplan (1988) did, that nuclear linguistic functions, those related strictly with the comprehension of words and sentences (the lexicon, syntax and semantics) depend on a relatively small area of the human neocortex (the association cortex situated around the Sylvian fissure), or that a certain brain region, separable from other systems, can be considered the "grammar center" (Sakai 2005). Rather, language appears, at a neuroanatomical level, as a system richly interconnected with other systems. But language is just a specific case of the wider problem of locating cognitive functions in the human brain. We believe with Maestú *et al.* (2005) that trying to study isolated processes or to isolate processes constitutes a very limited strategy. Cognitive faculties result from the addition of activity in different brain regions and, thus, of different cognitive processes performed in temporal coincidence and by means of a recognition code based on the firing frequency. When studying the neural correlates of cognitive processes three questions can be asked: (i) where, or in which areas, does the brain activity appear?; (ii) when, or at which moment, does the activity begin and end?, and (iii) how is the brain's activity organized during the performance of a given cognitive process? Researchers have spent the last decades focusing mainly on the first question. However, it has become increasingly clear that the neural foundation of cognitive activity is constituted by the singular qualities of different active brain areas, involving a specific spatial and temporal distribution for that net and person. It is very probable that the moment and frequency of the activation of each area provides relevant information for the final result (Maestú *et al.* 2005).

Thus, language must be seen as a cognitive faculty whose neural correlates are integrated in the brain. These correlates do not necessarily refer to discrete brain areas, but to activity distributed in time and space. In a similar way, the genetic correlates of the language faculty are integrated in the human's genetic inheritance, they are not separable or isolatable. It does not seem probable, in view of the studies related with *FOXP2* and the general genetic knowledge, that we will find a fraction of the human genome devoted exclusively or predominantly to language. Language's most plausible evolutionary history, as suggested by the reviewed studies, is not special or mysterious. It is another example of how natural selection has taken advantage of available cognitive and neural resources. It is not necessary to postulate catastrophic mutations to explain the sudden appearance of language as a whole (Bickerton 1995), nor

selective pressures that can account for the appearance of each of language's mechanisms or traits (Pinker & Bloom 1990). It is very possible that several of language's ingredients were present in some of our primate ancestors, though involved in non-linguistic, even non-communicative, functions. This is not to say that language is equivalent to other animals' cognitive capacities, or that language does not grant our species new possibilities. What we mean is that whatever seems new or unique in language is built on, or results from, a combination of very primitive and recent cognitive operations. It must be emphasized here that small, gradual and cumulative genetic changes might be enough to produce what, at a phenotypic level, might seem as a profound discontinuity or an evolutionary saltation.

We believe the conception of language that best fits the data reviewed in the present work is that of a collection of cognitive operations whose integrated functioning allows our species to develop and use language and verbal communication. Such cognitive operations may be related with different patterns of neural activity, be the result of different processes of genetic expression and have a very different evolutionary origin.

NOTES

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- 5 For a more detailed presentation see for instance: Lorenzo & Longa (2003: 21-65); Jenkins (2000: chapter 4); Stromswold (2001).
- 6 The classic work by Hockett (1960), among many others, provides a characterization of animal communication systems.
- 7 "This impairment is so pronounced as to render the speech of many of the affected members unintelligible to the naive listener, and it is so disabling, particularly during the childhood, that they have been taught a sign system to augment their speech" (Vargha-Khadem *et al.* 1995: 930). Also: "The nature of the behavioral phenotype shared by 15 affected members of the KE family [...] is best characterized as a deficit in the sequencing of articulation patterns rendering speech sometimes agrammatical and often unintelligible" (Watkins, Dronkers & Vargha-Khadem 2002: 466).
- 8 Longworth *et al.* (2005) prefer to reserve the involvement of striatal structures in language to integration processes, especially of an inhibitory kind, excluding their participation in morphological and syntactic processes. These conclusions derive of a study with patients with a neurodegenerative disease of the basal ganglia that took into account only the production and comprehension of past regular forms. However, the declarative/procedural model refers to a greater amount of linguistic functions and brain structures. What is suggested is that the basal ganglia are a part of a circuit related with the sequential and hierarchical combination of representations to form complex structures. These combinatorial operations are involved, in addition to morphology, in syntactic, phonological and, possibly, semantic aspects (Ullman, 2004). This perspective has been supported by a study by Teichman *et al.* (2005) in which they showed that Huntington patients with damage to the striatum present deficiencies in the application of rules in linguistic (morphological and syntactical) and non-linguistic (substraction) domains.

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