پژوهشنـامه علوم انسانی: شماره 55، پاییز 1386 «102 ـ 95»
Human Sciences: No. 55, Autumn 2007 «71 - 78»

Of Mice and Chimps (and Finches too)

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Abstract

Perhaps the most challenging as well as the most interesting question raised in linguistic studies is the Innateness Hypothesis of Human Language. There have been various types of evidence proposed to corroborate such a hypothesis. One type is based on different linguistic disorders resulting from brain damages. In the present paper, I shall focus on Specific Language Impairment (SLI), a linguistic syndrome, which does not correlate with general intelligence, but is known to be involved in linguistic deficiencies concerned with morphosyntax or the understanding of embedded (e.g., relative) clauses. This syndrome is related to the FOXP2 gene, which is active in the development of language skills, including grammatical competence. However, brain regions where the gene is expressed (caudate nucleus, cerebellum) are often characterized as being dedicated to the "motor control" phenomenon. Now the question is whether this gene is responsible for motor control or the linguistic behavior of the human kind. Investigating the role of this gene in chimps, mice and birds, we shall demonstrate that the findings corroborate the latter as well, in turn confirming the innateness hypothesis-or in today's terminology, the genetic basis of language-adopted by Chomsky.

Keywords: ***Specific Language Impairment (SLI), FOXP2 Gene, Brain Regions, Language Skills, Motor Control***

از موش و شامپانزه تا سهره

اوان یوری اگه‌رکا

استاد گروه زبان‌شناسی، دانشگاه مریلند

چکیده

*شاید از چالش برانگیزترین و در عین حال جالب‌ترین پرسش‌های مطرح در مطالعات زبان‌شناختی فرضیه فطری بودن زبان است همان‌گونه که در دستور همگانی چامسکی مفروض می‌باشد. به منظور تأیید این فرضیه، بسیاری از اختلالات زبانی، از جمله آسیب‌های مربوط به درک و تولید گفتار، مورد مطالعه و بررسی قرار گرفته‌اند. مقاله حاضر به بررسی آسیب خاص زبانی می‌پردازد. این سندروم تأثیری بر هوش عمومی ندارد اما حوزه واژ ـ نحوی و نحوه ادراک جمله واره‌های درونه‌ای ـ مانند جمله واره‌های موصولی ـ را تحت‌تأثیر قرار می‌دهد. سندروم مورد نظر بر اثر آسیب‌دیدگی ژن **FOXP2** پدید می‌آید که در شکل‌گیری توانش دستوری نقش بسزایی ایفا می‌نماید. لیکن نشان داده شده است که این ژن عامل شکل‌دهی به نواحی از مغز است که به نواحی کنترل حرکتی در مغز مشهوراند. بنابراین پرسشی که مطرح می‌شود آن است که آیا ژن **FOXP2** مربوط به رفتار زبانی انسان است و یا فعالیت‌های حرکتی وی؟ با بررسی نقش این ژن در موش‌ها، شامپانزه‌ها، و پرندگان (سهره‌ها) نشان داده خواهد شد که یافته‌های پژوهش حاضر مؤید فرضییه فطری بودن زبان ـ یا وجود خاستگاه ژنتیکی برای زبان ـ است.*

*کلیدواژه‌ها: آسیب خاص زبانی، ژن **FOXP2**، نواحی مغز، مهارت‌های زبانی، کنترل حرکتی*

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1 - Introduction

Few genes have captured our imagination like FOXP2. ***Google*** the FOXP family and you'll get half a million hits, including a Wikipedia entry describing the gene as "implicated in the development of language skills, including grammatical competence." If, however, you're thinking that clinches it - Chomsky is right: Human language is innate - remember Albert Einstein's quip: "Rafiniert ist der Herr Gott". FOXP2 is playing its role in language structuring in more sophisticated ways than anyone had anticipated.

2 - The FOX-sites Saga

It all started with Myrna Gopnik's (1990) description of Specific Language Impairment (SLI) in the KE family. This is a linguistic syndrome (today understood as an entire class thereof) that crucially does not correlate with general intelligence, and which involves deficiencies in such central language areas as morphosyntactic rules or the understanding of embedded (e.g. relative) clauses. But two qualifications are in order.

One, the relevant data ought to be analyzed by syntacticians. This is not to disparage the work already done, but to indicate that syntax is a mature, nuanced, science. For instance, deficiencies in "morphosyntax" vary depending on whether they occur on tensed verbal expressions (***walk*** vs. ***walked***) or morphemes yielding ***destruc-tion*** from ***destruct***. The syndrome could be profound, affecting the very fabric of syntax, if systematic ungrammaticalities of the first kind are discovered; if only morphemes of the second sort are affected, it may be more superficial.

Second qualification: what may be ***specific*** to language is not a pre-theoretical issue, just as it is not obvious whether, say, a virus is a living creature. Surely many notions are linguistic, a priori. But whether behaviors with a less directly observable linguistic base happen to depend on the linguistic system also is rather more delicate. Is anybody prepared to decide, for instance, whether the ability to (un)tie knots - which is unique to humans and occasionally co-morbid with linguistic syndromes - is (or is not) parasitic on the linguistic system?

That said, controversy was served from the moment Gopnik and collaborators argued for the linguistic nature of SLI in the KE family, particularly since oral praxis is also affected in these individuals, as well as other abilities that may be seen as broadly rhythmical. These were taken by Faraneh Vargha-Khadem as direct indications that the syndrome is non-linguistic, for what seems less linguistic than failing to trace a circle, or biting your lip after you blow your cheeks? (For recent reviews of this perspective see Watkins, Dronkers & Vargha-Khadem 2002.) Then again: Can anyone get non-linguistic creatures to perform ***any*** of those tasks? Has anybody trained chimps to tie something?

Whatever its ultimate nature is, in 1996 Anthony Monaco and Simon Fischer tracked, at Oxford, the genetic and brain correlates of the KE deficit (see Lai et al. 2001). Since it segregates in a classical Mendelian autosomal

dominant pattern, by using standard methods the team singled out chromosome 7, eventually the q31 region. And the "smoking gene" turned out to be quite different from a mere speech controller. In fact, it is a member of the regulating FOX (forkhead/winged-helix) family!

Here the story gets humbling: FOX sites are present all the way "down" to yeast, and of course all cordates. The Oxford group, lead by Cecilia Lai, even determined the locus of the KE mutation: Two "spelling mistakes" in exon 14, one of them crucial for the syndrome; these among 600,000 bases, of which 14,000-plus code the protein. To make life interesting, Svante Pääbo and Wolfgang Enard reconstructed the phylogenesis of FOXP2 across the mammalian world (see Enard et al. 2002). Their research finds merely 3 point mutations between us and mice (75 million years of evolution), 2 of which occur between us and chimps (less than 6 million). So something drastic happened in our lineage, involving the FOXP genes and specifically FOXP2; they calculate that within the last 200,000 years...

3 - Singing in the Brain

The story first told could be summarized in a broadcast sound-bite. Brain regions where the gene is expressed (caudate nucleus, cerebellum) are often characterized as dedicated to "motor control". So a gene regulating that must affect "grammatical skills": Tinker a bit (***threonine*** to ***asparagine*** at position 303, ***asparagine*** to ***serine*** at position 325), and bingo! The ability for "vocal learning" is in place, and evolution marches on.

Of course, if it's all so easy, why haven't scores of species hit on such a wonderful system - scores of times over the evolutionary eons, the way locomotion or breathing independently (re)appear. Moreover, if FOXP2 is playing such a motor control role for us, what is it doing in mice, birds, fish... all the way down to creatures where the whole concept of "information process" seems harder to ascertain?

As the regulating gene it is, FOXP2 does much more than "be involved" in motor control. This much is uncontroversial, and clear even in the KE family, where minor deficiencies in the maternal copy, though affecting language, have no discernible consequences on other processes associated to the gene, which regulates aspects of embryogenesis related to the heart, lung, guts and various brain regions. But is this supposed to make things easier?

In addition, while the gene is involved in such apparently "non-information" tasks even in fish, things do change as the action gets closer to us. In the mouse, for instance, Joseph Buxbaum's lab showed in 2005 that knocking out the gene results in pups failing to communicate with their mother (see Shu et al. 2005). This has lead to research into the nature of relevant information systems in mice, which because of their ultrasonic nature had never been studied - mice "sing" in a high key... Which holds an important lesson: perhaps the behavioral role of genes like these partially lies beyond our initial expectations.

The most interesting case arises in song-birds, which have two curious brain circuits. Young males rely on a tutor to acquire their song. Interconnected regions of the striatum, thalamus and nidopallium create a circuit which, if damaged, prevents the song from being learnt. Once acquired, it is somehow produced by activating interconnected regions of the arcopalium, an area called the "high vocal center", and again the striatum –a link between both circuits in the basal ganglia, called Area X. Not only is FoxP2 (lower case denotes the non-human allele) expressed in Area X and thalamic region; as Constance Scharff has shown, expression in Area X increases during the critical age (post-hatch days 35 - 50) at which the bird acquires the song (see Scharff and White 2004 for a review).

It gets better. Stephanie White and Ikuko Teramitsu have just shown how the mRNA of FoxP2 ***sub***-regulates in Area X as males sing to themselves, effectively practicing small variants of the song, while when the action is directed to a female it slightly ***up***-regulates (Teramitsu and White 2006). This is an extraordinary result, as in both instances the same motor control is at stake. So FoxP2 cannot just mean "motor control".

What structure could be crucial both at the acquisition and production stages? If asking about humans, "parsing" would be the first answer to try (Piattelli-Palmarini and Uriagereka, forthcoming). After all, a baby has to parse the structure she's experiencing, and linguistic production is "parsing in reverse". The issue arises for any complex structure that has to be "squeezed out of the brain", into some unidimensional channel like chirping - or, of course, speech.

The chaffinch song is divided in two halves ("trill" and "flourish"), the first composed of several sub-constituents, each containing complex syllables; moreover the length ratio of trill to flourish is inversely correlated. These matters are being analyzed by Robert Berwick at MIT, among others, to uncover their computational conditions; all indications are that we are talking about robust constructions that require serious computational memory to represent them, and whose fine-tuning and effective communication determines success in mating.

FoxP2 might even regulate a "memory window". If it narrows, "variations" are expected, since what is held constant is, well, narrow. If the window widens, more structural components will be held in the "memory buffer". Musicians know how to "jazz it up" or "square it", sticking, or not, to the rhythmico-melodic structure. Regulating memory is key to this. Moreover, Sébastien Derégnaucourt and colleagues have shown that sleep affects how birds acquire their song, and speculate that they may dream it, singing it in their brain (see Derégnaucourt et al. 2005). If so, the issue is how that rich internal structure can be pushed out into the air-waves, one note at a time, á la Bobby McFerrin.

4 - Remember: One-step-at-a-time

FoxP2 differs in song-birds and mice at only five amino-acid positions, and from our own FOXP2 at 8. This is an extraordinarily conserved protein.

Moreover, the pattern of FoxP2 expression in the avian brain is virtually identical to the mammalian counterpart (see Jarvis 2006 for perspective). FOXP2 in human fetuses is expressed in the analogue of the bird's Area X, the caudate nucleus. Could this be happening with ***no*** connection to whatever role FoxP2 has in song-birds - especially if the closest we have to a human knock-out version of the gene yields SLI?

Again, it gets better. Michael Ullman observes that SLI-style impairments are associated with dysfunctions of the caudate nucleus and the frontal cortex (Broca's area), and that crucially such frontal/basal-ganglia circuits play a core role in "procedural" memory (see Ullman and Pierpont 2005). Rule-governed (vs. idiosyncratic) linguistic mappings can be captured by distinguishing procedural vs. declarative memory. While idiosyncratic mappings stored in a "mental lexicon" depend on declarative memory, rule-governed computations involve, instead, grammatical workings involving procedural memory. Individuals afflicted with procedural system brain abnormalities result in characteristic SLI impairments, including "sequential" behaviors that depend on this system.

If Ullman is right, his observation about human procedural memory can perhaps be connected to the above speculations regarding avian "parsing" memory, involving an area analogous to the human caudate nucleus. This is a coincidence not just on anatomical circuits, their presumed protein regulation, or even the hormonally-regulated physiological factors concerning activation in "critical periods" related to acquisition of a relevant competence; it is an abstract structural and functional coincidence, remarkably.

The idea is testable. For birds, m-RNA expressions should be roughly analogous for listening females parsing their crooners' ballads. Bird knock-out technology is not too far into the future, so we may soon know what FoxP2 is doing in their Area X - in fact linguists should study bird songs to fully uncover corresponding "grammatical" nuances. For humans, it would be good to observe the in-vitro effects on human cells of the FoxP2 protein expressed in chimps, and moreover the precise differences in the intron sequences of chimp and human alleles. And of course we have to figure out what the target genes of FoxP2 are, and whether they work together in different species in any given way - or whether there was any selective sweep for them associated to the mutations found in FOXP2 in humans. Staying within chimps, mice and finches might do it for a first approximation, as there probably is meaning to the idea that our lineage may well be some function of combining the chimp's "semantic" capacities with the finch's "musical" abilities, to squeeze them into a melodic line –the mouse then used for contrast. But of course we should also be prepared to see the gene playing a role in other species capable of "vocal learning" or with rich communication systems, like bats or dolphins.

That is all speculation, no doubt –but not unreasonable. The chimp's cortex, and their corresponding "thought world", looks frighteningly similar to ours, as

Marc Hauser has shown even for more distant tamarins (see Uller et al 2001). In contrast, the finch has no neo-cortex to seriously speak of, but presents instead an inner brain circuit that seems great for linearizing complex, abstract structures. This is perhaps achieved through operational memory regulation via FoxP2, possibly aided by the rest of the FoxP genes, among other functions these factors have that go beyond brain activity. Neither of those (sets of) properties is necessary: species reach stable evolutionary niches without singing (Erich Jarvis shows in his (2006) how only 3/24 major avian groups have anything of the sort, though they use FoxP2 for other functions) or the thought systems higher apes have achieved (which are surely exotic for mammals). But they are, nonetheless, building blocks that nature has at its disposal, whose very recent combination has had drastic effects on our lineage.

5 - Conclusion

So do these findings prove or disprove Chomsky? The answer is "Yes!" The evidence suggests the human language faculty is innate, and whether it is also specific depends on whether we accept the combination just alluded to as uniquely linguistic. It is certainly unique to the species. The fact that it may also be involved in other "sequential" behaviors doesn't invalidate that claim –it may only show that the language faculty is even more central to human cognition than it may have seemed at first. We may be using it even when remembering to take various daily tasks one-step-at-a-time... So stay tuned.

References

Derégnaucourt S., P. Mitra, O. Feher, C. Pytte and O. Tchernichovski. 2005. "How sleep affects the developmental learning of bird song." ***Nature,*** 433: 710-716

Enard, W., M. Przeworski, S. Fisher, C. Lai and V. Wiebe. 2002. "Molecular evolution of FOXP2, a gene involved in speech and language." ***Nature,*** 418: 869-872.

Gopnik, M. 1990. "Feature-blind grammar and dysphasia." ***Nature,*** 344: 715-722.

Jarvis, E. 2006. "Evolution of structures for song-learning in Birds: A synopsis." ***Acta Zoologica Sinica,*** 52 (Supplement): 85-89.

Lai, C., S. Fisher, J. Hurst, F. Vargha-Khadem and A. Monaco. 2001. "A forkhead-domain gene is mutated in a severe speech and language disorder." ***Nature,*** 413: 519-523.

Piattelli-Palmarini, M. and J. Uriagereka. Forthcoming. "The case of FOXP2 (the so-called language gene) revisited," in ***Biolinguistics: Language Evolution and Variation***, ed. Anna Maria di Sciullo. Oxford: OUP.

Scharff, C., and S. White. 2004. “Genetic Components of Vocal Learning”, pp 325–347, in ***Behavioral Neurobiology of Birdsong***, eds. H. Philip Zeigler and Peter Marler, New York: New York Academy of Sciences.

Shu W., J. Cho, Y. Jiang, M. Zhang, D. Weisz, G. Elder, J. Schmeidler, R. De Gasperi, M. Sosa, D. Rabidou, A. Santucci, D. Perl, E. Morrisey and J. Buxbaum. 2005. “Altered ultrasonic vocalization in mice with a disruption in the Foxp2 gene”. ***Proc Natl Acad Sci U S A,*** 102: 9643-9648.

Teramitsu, I. and S. White. 2006. “FoxP2 Regulation during Undirected Singing in Adult Songbirds.” ***The Journal of Neuroscience,*** 26: 7390-7394.

Uller, C., M. D. Hauser and S. Carey. 2001. “Spontaneous representation of number in cotton-top tamarins.” ***Journal of Comparative Psychology,*** 115: 248-257.

Ullman, M., and E. Pierpont. 2005. **“**Specific Language Impairment is not specific to language: The Procedural Deficit Hypothesis.” ***Cortex,*** 41.3: 399-433.

Watkins, K., N. Dronkers and F. Vargha-Khadem. 2002. “Behavioral analysis of an inherited speech and language disorder: Comparison with acquired aphasia.” ***Brain,*** 125: 452-464.