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Letters

Sly *FOXP2*: genomic conflict in the evolution of language

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The origin of speech and language is arguably the most important transition in the evolution of modern humans. In a recent issue of *TREE*, Számadó and Szathmáry [1] review hypotheses for the potential selective pressures involved in the origin of language, with a 'top-down' conceptual approach focused on the compatibility of the hypotheses with game theory models, and the development of useful criteria for judging among alternative historical narratives.

However, there is an alternative framework for analyzing the origin of language, based on the analysis of the evolutionary-genetic and neurological changes that were concomitant to modern human origins. This framework is grounded in the mirror-neuron system of humans and related primates, which provides a well characterized neural substrate (i.e. the same sets of premotor neurons fire when one observes or hears a movement or sound made by another individual as fire when making the movement or sound one's self) for an apparent evolutionary transition in the human lineage from gestures, to gestures with articulations, to articulations that are free of gestures [2]. Evidence from functional imaging, gene-expression studies, phenotype–genotype associations, and the molecular evolution of *FOXP2* implicates this gene in the adaptive evolution of the mirror neuron system in humans, and in the origin of articulate speech [3,4].

What can the functional design of *FOXP2* and the mirror-neuron system tell us about the selective pressures involved in the origin of human language? A recent study by Feuk *et al.* [5] provides preliminary evidence that *FOXP2* is subject to effects of genomic imprinting, with relatively high expression from the paternal chromosome. The conflict theory for the evolution of genomic imprinting, which is well supported by evidence from diverse studies of placental development, molecular physiology and behavior (e.g. Ref. [6]) predicts that such a pattern of gene expression evolves in the context of constrained conflict between asymmetrically related kin (especially mothers and offspring), with genes that are paternally expressed in offspring exerting effects that are more 'selfish'. In the case of human language, a simple behavioral mechanism for such conflict would involve the benefits of earlier-developing, more-articulate speech to children in interactions with their mother; indeed, any parent of young children knows that the main function of *their* articulations is to get more of this or that parental resource. By this hypothesis, articulate human speech evolved as it develops, predominantly in the context of mother–offspring interactions, which are permeated by a complex mix of cooperation and conflict. The evolutionary dynamics of language evolution in humans are much more complicated than this and must involve the evolution of many interacting genes; however, the apparent imprinted status of *FOXP2* directly connects molecular genomics with behavior and language, in the context of evolutionary theory.

The hypothesis that articulate human speech and language evolved at least partially in the context of genomic conflict is also supported by: (i) evidence for imprinting of *FOXP1* [7], which interacts with *FOXP2* in early brain development; (ii) the role of *FOXP2* in ultrasonic vocalizations by young mouse pups, which exhibit complex, interactive characteristics that are indicative of mother–offspring communication [8]; and (iii) linkages of *FOXP2* allelic variants to autism and schizophrenia [9], two disorders of the social and linguistic brain whose development is mediated by the mirror-neuron system [10,11] and by imprinting effects (e.g. Ref. [12]). The hypothesis provides a novel selective context for this key transition in the origin of modern humans, one that can, most importantly, move beyond game theory models and historical narratives in being subject to strong empirical tests.

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