"Language impairment gene" does not necessarily equate to "language gene"

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Abstract: The finding of the same language deficit in half the members of the KE family is taken as suggesting that a specific allele (FOXP2) is normally involved in the development of language. Recent studies, however, question the exclusivity of FOXP2, and it is argued that the finding of a gene that disrupts language should not be taken as strong evidence for the existence of genes that underlie it.

Locke & Bogin's (L&B's) argument that life history theory provides a fruitful way of re-examining the relationship between evolution and language development is, for the most part, a compelling if somewhat speculative argument. Both the unique life history pattern and the uniqueness of language in our species require explanation. I agree that such patterns should be examined in the light of selective advantages. L&B provide evidence of selective advantages for a wide range of aspects of language development. Where I would urge caution, however, lies in the use of studies of language deficits in the "KE" family to imply that these provide evidence of the existence of genes directly related to the neurological substrate supporting language production and grammar.

In the early 1990s the KE family caused a stir among linguists when it was discovered that more than half of the 31 members of three generations of the family had impaired language abilities (sometimes referred to as specific language impairment, or SLI). It was found that individuals with SLI have a number of problems, including a deficit in the ability to use inflection (e.g., producing the past tense of a verb) and problems of articulation, comprehension, and orofacial dyspraxia (i.e., putting together orofacial movements; Bishop 2002; Gopnik 1990; 1997; Pinker & Jackendoff 2005).

The finding of the same language problem in such a high proportion of family members has been taken by a number of researchers as suggestive of a specific underlying genetic problem. If a specific allele leads to such problems then it has been argued that an alternative allele might normally be involved in the development of appropriate neural hardware to support language (Gopnik 1990; Jackendoff 1994; Pinker 1999). L&B are clearly sympathetic to such an argument.

In support of L&B's stance there is now strong evidence of a defective dominant allele of a single gene that is found on the long arm of chromosome 7 in the impaired members of the family – FOXP2 (Lai et al. 2000; 2001). Moreover, as L&B point out, scanning techniques have demonstrated associated neurological anomalies of structure and function in sufferers, such as a bilateral deficit in activation of Broca's area (Liégeois et al. 2003) and abnormal activation of the caudate nucleus (Vargha-Khadem et al. 1998). Although L&B do not explicitly label FOXP2 as a "grammar gene," their use of the KE family to back up their argument that "selection acted on [language] performance" (sect. 11 of the target article) implies that this is an allele directly related to language and hence has arisen via selection pressures for that purpose.

It is entirely possible that the KE family's SLI does provide evidence of an allele related to the development of language, but currently there are at least two problems with the argument as presented by L&B. First, the evidence is presented as if there is general consensus that an exclusive language-related problem is found in the KE family and that this provides support for the existence of a specific defective gene related to language production. In fact, Gopnik (1997) herself has documented how controversial this conclusion is, with some experts suggesting that the language impairments uncovered might be part of a more general cognitive deficit, which might include non-linguistic problems (Leonard 1994; Vargha-Khadem et al. 1995). It is certainly possible that the problem is a part of a more general cognitive deficit since afflicted members of the family have IQ scores 18 to 19 points lower than those of unimpaired members (including nonverbal aspects of IQ; Vargha-Khadem et al. 1995; 1998).

Second, even if an allele has been discovered that disrupts language production via the development of neurological abnormalities, this is not strong evidence that an alternate allele exists that is directly involved in the development of neural circuitry underlying language and speech (Karanth 2005). As Lai and colleagues caution with regard to the FOXP2 allele, the biological deficit may well be in the form of a trigger that disrupts a key stage of embryogenesis rather than the replacement of an allele coding for normal language development (Lai et al. 2001; Karanth 2005). Removing an air-filter from an internal combustion engine will stop a car from running but that does not mean that the air-filter is responsible for running the car (Workman & Reader 2004). Finally, Newbury et al. (2002), through association and mutation screening analysis, have thrown doubt on the argument that variants in FOXP2 underlie such forms of language impairment.

None of these misgivings constitutes a deathblow to L&B's argument; they may well be correct that language development and production are directly related to specific alleles. But, currently, the evidence from families that show language deficits such as the KE is equivocal and needs to be examined with a degree of caution. L&B have done a service to our understanding of the relationship between evolution and language by weaving together a number of sources of evidence using the framework of life history theory. They should be careful not to leave any loose threads.

Authors' Response

Life history and language: Selection in development

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Abstract: Language, like other human traits, could only have evolved during one or more stages of development. We enlist the theoretical framework of human life history to account for certain aspects of linguistic evolution, with special reference to initial phases in the process. It is hypothesized that selection operated at several developmental stages, the earlier ones producing new behaviors that were reinforced by additional, and possibly more powerful, forms of selection during later stages, especially adolescence and early adulthood. Peer commentaries have provided opportunities to explain human life history more comprehensively, and to add details to our account of spoken language. We made no attempt to explain syntax in the target article, but we propose here that selection for "vocal plumage" may have increased our species' capacity for utterance complexity, a development that would have benefited all levels of language.

It is a rare pleasure to have comments on our target article from so many thoughtful colleagues, representing such a wide range of academic disciplines and cultures. We will begin our response by addressing comments that relate

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