Although this result is not new, is not universally appreciated. It seems to have been stated first by Abrams [8], and cited by Charlesworth [9] and Williams and Day [10]. Curiously, Abrams' paper [8] is mentioned in the text of Ref. [1] only for conclusions about density dependence. His important result for the density-independent case appears only as an undiscussed entry in Table 1.

The importance of age-specificity is not restricted to continuous models, age-classification, deterministic models or density-independent models. If demography is described by a discrete matrix population model (age- or stage-classified,

$$\mathbf{n}(t+1) = \mathbf{A}\mathbf{n}(t), \qquad \text{[Eqn II]}$$

then the selection gradient on a trait θ is $d \log \lambda / d\theta$, where λ is the dominant eigenvalue of $\mathbf{A}$ (e.g. Ref. [11]). Imposing an age-independent (or stage-independent) mortality h multiplies $\mathbf{A}$ by the scalar e^{-h}. Because the derivatives of λ depend on the eigenvectors of $\mathbf{A}$, which are unchanged by such multiplication, the selection gradients are unaffected by the additional mortality.

If the additional mortality $h(t)$ varies stochastically, the selection gradient is the derivative of the stochastic growth rate $\log \lambda_s$ [12,13], and this selection gradient is equally unaffected by the additional age-independent mortality. If the extra mortality is imposed on a density-dependent model,

$$\mathbf{n}(t+1) = \mathbf{A}[\mathbf{n}(t)]\mathbf{n}(t), \qquad \text{[Eqn III]}$$

then there are two possible sources of age dependence: the direct effect of the additional mortality and its indirect effect through the equilibrium density. The invasion exponent for Equation III is the log of the dominant eigenvalue of $\mathbf{A}[\hat{\mathbf{n}}]$; the selection gradient is the derivative of this exponent, $d \log \lambda(\mathbf{A}[\hat{\mathbf{n}}])/d\theta$ If neither the extrinsic mortality nor the density dependence are age specific, then the extra mortality has no effect on the pattern of selection gradients.

In conclusion, for several ecologically interesting cases, age-independent mortality does not change the age pattern of selection and, hence, does not change the tendency to evolve senescence. Extrinsic mortality affects senescence only if it affects different age classes differently. It is an interesting problem to figure out how the age specificity of mortality interacts with that of the selection gradients. Some preliminary results suggest that extrinsic mortality that increases with age also increases the tendency to senescence, and vice versa (H. Caswell, unpublished).

The paper by Williams *et al.* [1] is valuable for its exploration of the issues involved in measuring aging and its relation to environmental hazards. However, one of the reasons for the 'overall inconclusive findings of the existing body of empirical work' that they report is that the prediction on which some of that work is based is not, in fact, a prediction.

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0169-5347/$ – see front matter
doi:10.1016/j.tree.2007.01.006

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 evol-

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Available online 2 February 2007.

utionary-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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