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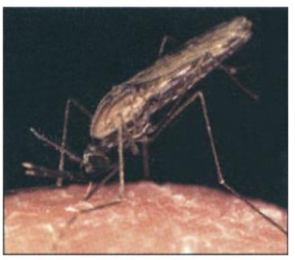
Bloodthirsty Hitchhikers?

NEARLY 100 YEARS AGO, FAHRENHOLZ claimed that parasite phylogeny mirrors host phylogeny (1). The report "Resolution of the early placental mammal radiation using Bayesian phylogenetics" by William J. Murphy and colleagues (14 Dec., p. 2348) brought to our minds Fahrenholz's idea, which, although in many cases not true in the strict sense, served as a hypothesis in numerous studies of coevolution. There are some striking parallels between the proposed placental mammal phylogeny and the proposed phylogeny of the mosquito genus *Anopheles* (*2*).



A female *Anopheles gambiae*.

CREDIT: JIM GATHANY

Anophelines are mammalian ectoparasites whose life cycles depend on a protein-rich blood meal required by females for egg production. Similar to placental mammals, anophelines likely originated on the ancient supercontinent Gondwana, and their basal lineages experienced rapid diversification that might have coincided with the separation of South America and Africa.

Because no sufficiently old mosquito fossil records are available, our understanding of anopheline evolutionary history depends largely on a careful analysis of their geographical distribution. Of the six *Anopheles* subgenera, *Stethomyia*, *Lophopodomyia*, *Kerteszia*, and *Nyssorhynchus* inhabit South America, *Cellia* is found in the Old World, and *Anopheles* is cosmopolitan. Remarkably, like the placental mammal clade Boreoeutheria, subgenera *Anopheles* and *Cellia* not only appear to occupy a derived (as opposed to basal) position in the anopheline phylogeny, but also are the most diverse. Phylogenetic evidence suggests that the subgenus *Anopheles*, after origination in South America and rapid dispersal throughout Laurasia, reentered the Neotropics from the north. Presumably, then, the early radiation of mammals was closely followed by radiation of anophelines, which thrived on the blood of newly emerging taxa. If this was the case, further studies of *Anopheles* phylogeny might shed new light on such issues in mammalian evolution as timing of divergences and routes of dispersal.

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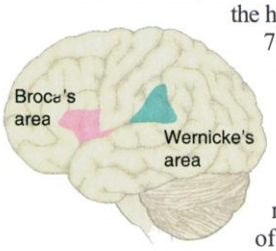
Neuroscience of Stuttering

WE DISAGREE WITH WILLIAM H. PERKINS' comments (Letters, "Stuttering: a matter of bad timing," 26 Oct., p 786) pertaining to the Random Samples item "The stammering brain" (3 Aug., p 795). Perkins takes issue with the discovery by Anne Foundas at Tulane University and her colleagues that anatomical differences between stutterers and nonstutterers in the two brain regions

associated with speech and language, Broca's area and Wernicke's area, might explicate the etiology of stuttering (*1*).

Briefly, Perkins says these areas function at cognitive rates too slow to be considered a source of stuttering. Perkins cites George Miller's studies, decades ago, indicating that the human cognitive rate is 7 ± 2 thoughts per second, the same as the rate of syllable production, which is under voluntary control (*2*). Perkins maintains, "Any cause of stuttering has to account for why it can be involuntary and ... how high-speed speech sounds can be produced with low-speed cognitive and linguistic equipment."



Certainly, Broca's area and Wernicke's area can process data more rapidly than 7 ± 2 items per second. Miller's review related to issues of absolute judgement and immediate memory when humans are in a stimulus-response setting or functioning in a communication system. He discussed how brains overcome the apparent 7 ± 2 limit, for instance, by making stimuli multidimensional, and he emphasized that such recoding powerfully increases the amount of information that can be processed. Our language, he commented, can repackage material into a few chunks rich in information (*2*).

Without demonstration of an equivalency between how we process the stimulus input we memorize and how we process spontaneous thought for speech/language production, the 7 ± 2 limit lacks merit when applied to linguistic encoding of thoughts. Regardless, there is no neuroscientific data indicating that we think at the rate at which we talk, not to mention that there is no denotation of what a "thought" is.

Foundas' discovery that abnormalities in the anatomic substrate of language might differ among stutterers raises the possibility that some have difficulty processing speech-motor control because of aberrancies in the frontal opercular areas (Broca's), others because of aberrancies near Wernicke's area. Their research has far-reaching implications: Perhaps stutters with anterior versus posterior gyral abnormalities differ in severity of their disorder, genetic background, or response to speech or pharmacologic therapy.

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1. A. L. Foundas *et al.*, *Neurology* **57**, 207 (2001).
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CORRECTIONS AND CLARIFICATIONS

REPORTS: "Direct determination of the timing of sea level change during Termination II" by C. D. Gallup, H. Cheng, F. W. Taylor, R. L. Edwards (11 Jan., p. 310). On page 312, in the second column, the initial sea level value was erroneously given as 8 ± 5 m below present sea level. The correct value is 38 ± 5 m below present sea level. And in the legend for Fig. 2C, the SPECMAC record is not benthic but planktonic.

BOOKS *ET AL.*: "The pill in context" by L. Schiebinger (7 Dec., p. 2106). The caption that accompanied this review of Lara V. Marks' book *Sexual Chemistry: A History of the Contraceptive Pill* was mislabeled. The 1979 poster showing women how to use the pill was identified as Malaysian, but is Thai.

CREDIT: PRESTON MORRIGHAN