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References
1. N. G. Jablonski and G. Chaplin, *J. Hum. Evol.* **24**, 259 (1993).
2. ———, in *Human Evolution Through Developmental Change*, N. Minugh-Purvis and K. McNamara, Eds. (Johns Hopkins Univ. Press, Baltimore, MD, in press).

DNA Discovery

In a News section of the "Evolution" special issue (25 June, p. 2107), Virginia Morell writes, "he [Charles Darwin] wrote 100 years before DNA was discovered." She was in all likelihood referring to Watson and Crick's classic 1953 paper suggesting a structure for DNA, not its discovery. Any student of biology will be aware of the delightful irony that Mendel's seminal results were first reported in 1865, just 6 years after *On the Origin of Species* was published, although it was not until the modern synthesis some 70 years later that the two fields became integrated. What is not so well known is that Frederick Miescher first isolated DNA from pus-laden bandages in Tubingen Castle in Germany in 1869, although once again, the hereditary function of DNA was not conclusively demonstrated until the 1940s. We are now seeing a synthesis of evolutionary genetics and molecular biology, ultimately deriving from these three results, remarkably published within a decade of each other in the 19th century.

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Mitochondrial Recombination? (Continued)

In her article "Can mitochondrial clocks keep time?" (News of the Week, 5 Mar., p. 1435), Evelyn Strauss references E. Hagelberg *et al.* (*1*) as providing evidence for recombination in human mitochondrial DNA (mtDNA). Those authors suggest that a genetic mutation (at 16076 in HVS I) found in all three haplogroups among a study population of Nguna islanders is best explained "by paternal leakage of mtDNA and subsequent recombination" (*1*, p. 490). They also suggest that previously identified "hypervariable" mtDNA sites are actually ancient substitutions present in multiple haplogroups that result from recombination with paternal mtDNA.

We agree with Peter Arctander (Letters, 25 June, p. 2090) that these are improbable suggestions. Paternal mtDNA transmission in humans has not, to our knowledge, been confirmed. Paternal mtDNA in interspecific crosses of mice is apparently eliminated in early embryogenesis (*2*); "leakage [is] restricted to the first interspecific cross, and it did not spill over to subsequent backcrossing" (*3*, p. 885).

Recombination should disrupt the linkage between mutations within haplogroups. We estimated linkage disequilibrium (*4*) between all pairs of variable sites in HVS I for the 41 Nguna (*1*), 376 Native Americans, and 695 European individuals from a mtDNA database (*5*). Ninety-nine percent, 96%, and 93%, respectively, of all D^1 values indicated complete linkage (or nonlinkage) of variable sites. The incompletely linked sites were compared to sites previously identified as hypervariable (*6*). Within the region surveyed by Wakeley (*6*), four of six in the Nguna, five of six in the Amerind, and nine of 13 in the European samples were identified as hypervariable. Thus, most variable sites are completely linked or unlinked, and the great majority of the incompletely linked sites are "traditional" hypervariable sites.

There is little evidence for recombination outside of the D-loop (*7*) or between it and the coding regions (*8*). Because migration and recombination are presumably rare, the combination of these events is extremely unlikely. Alternative explanations for the 16076 polymorphism include that it is a hypervariable site specific to the Nguna or the result of systematic sequencing errors. Either way, better evidence would be required before recombination could be considered as a viable explanation for this polymorphism.

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References
1. E. Hagelberg *et al.*, *Proc. R. Soc. Lond. Ser. B* **266**, 485 (1999).
2. H. Kaneda *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **92**, 4542 (1995).
3. H. Shitara, J-I. Hayashi, S. Takahama, H. Kaneda, H. Yonekawa, *Genetics* **148**, 851 (1998)
4. S. Schneider, J-M. Kueffer, D. Roessli, L. Excofier, *Arlequin: A Software for Population Genetic Data Analysis, Version 1* (Genetics and Biometry Lab, Department of Anthropology, Univ. of Geneva, Geneva, 1997).
5. O. Handt, S. Meyer, A. von Haeseler, *Nucl. Acids Res.* **26**, 126 (1998).
6. J. Wakeley, *J. Mol. Evol.* **37**, 613 (1993).
7. D. A. Merriwether *et al.*, *ibid.* **33**, 543 (1991).
8. L. Graven *et al.*, *Mol. Biol. Evol.* **12**, 334 (1995).

Protein Crystallization at NASA: Well Grounded

The article "Negative review galls space crystallographers" by Jennifer Couzin (News of the Week, 24 July 1998, p. 497) summarized a previous report by the Amer-