Letters

New World Epidemics

As anthropologists working with archeological data on the historic Seneca and Onondaga, we would like to add our voices to the controversy discussed by Leslie Roberts in her 8 December article "Disease and death in the New World" (Research News, p. 1245). We dispute the validity of historian Henry Dobyns' use of Seneca archeology data to document the occurrence of 16th-century epidemics of European diseases among the Seneca, which is critical to his extrapolations for the pre-1492 population of the Northeast. Most fundamentally faulted is his unqualified use of the Seneca sequence of village movements (*1, 2*) to prove the occurrence in western New York of specific epidemics that are dateable in the Southeast. The dates referred to in the Wray and Schoff chronology (*1*), as in the archeological chronologies of other areas, were not intended as more than working approximations of occupation intervals, and certainly not as absolute dates with which to document specific events. At times, Dobyns appears to recognize the inherent imprecision of a hypothetical archeological chronology, as he adjusts the dates of Seneca village movements to match the timing of known epidemics in the Southeast. However, he then uses the "concurrences" to demonstrate that the epidemics in question occurred among the Seneca and compelled them out of fear to abandon their villages and move on.

A dubious assumption of Dobyns is that epidemic diseases were the only reason for the periodic village relocations characteristic of Iroquoian peoples in the Northeast—a settlement pattern widely attributed to any number of other possible factors. Furthermore, he seems to assume that any disease process that occurred in the Northeast at roughly the same time as a known disease anywhere along the eastern seaboard must necessarily be the same disease. Yet, according to his own reconstruction of the synchronization between specific disease episodes and Seneca village relocations (*3*, table 29), even contemporary populations located less than a mile from one another were apparently not always affected by the same epidemics. In fact, we have evidence that the Seneca suffered at least two periods of unusual population loss late in the 16th century (*4*). However, in addition to European diseases, many other explanations must be considered, including famine (supported by osteological evidence), diseases of native origin, and warfare.

Finally, and perhaps most compelling, is the observation that the archeological record relating to the period from A.D. 1500 to 1650, for those areas of Iroquoia for which appropriate data exist (the Seneca, Onondaga, and Mohawk regions), does not support the theory of a dramatic drop in population. If anything, an increase in population appears to be indicated for the New York Iroquois, presumably due largely to the well-established in-migration of refugees, adoptees, and captives from other regions of the Northeast.

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REFERENCES

1. C. F. Wray and H. L. Schoff, *Pa. Archaeol.* **23**, 1550 (1953).
2. C. F. Wray, *Manual for Seneca Iroquois Archeology* (Cultures Primitive, Honeoye Falls, NY, 1973).
3. H. F. Dobyns, *Their Number Became Thinned* (Univ. of Tennessee Press, Knoxville, TN, 1983).
4. C. F. Wray, M. L. Sempowski, L. P. Saunders, G. C. Cervone, *The Adams and Culbert Sites* (Charles F. Wray Series in Seneca Archaeology, vol. 1, Research Records 19, Rochester Museum and Science Center, Rochester, NY, 1987); C. F. Wray, M. L. Sempkowski, L. P. Saunders, *Two Early Contact Era Seneca Sites: Tram and Cameron* (Charles F. Wray Series in Seneca Archaeology, vol. 2, Research Records 21, Rochester Museum and Science Center, Rochester, NY, in press).

Evolutionary Questions: The "Progenote"

M. Mitchell Waldrop, in his Research News article "How do you read from the palimpsest of life?" (3 Nov., p. 578), states that Steven A. Benner *et al.* (*1*) "try to reconstruct the 'progenote,' which is their name for the last common ancestor of modern forms of life."

Although the name "progenote" was introduced about 10 years ago (*2*), the underlying concept goes back another decade, to "the recognition that at sufficiently early stages in evolution the fundamental information-transferring processes . . . must have been error-ridden . . ." (*3*). This necessarily follows from the fact that the translation apparatus is very complex, far too complex to have evolved in one step. Therefore, in its rudimentary stages it almost certainly translated genetic information in an imprecise, perhaps even ambiguous, fashion (*2–4*).

As a result of their having rudimentary translation mechanisms, the ancestors of modern cells (which have existed for the last three or so billion years) were by comparison limited in almost every way (*2–5*): Their proteins had to be smaller or less precisely defined than modern proteins, or both, which meant that primitive enzymes were typically quite different from modern enzymes. As nucleic acid replication was a less accurate process than it now is, the number of different genes the ancestral cell could carry was severely limited. And the states of the cell were simpler and less precisely defined than they are in modern cells. To designate entities that were in various stages of evolving a translation apparatus, whose linkage between their genotype and phenotype was not yet as precise as that seen in modern cells, we coined the term "progenote" (the modern cell being considered a "genote").

The article by Benner *et al.*, and consequently Waldrop's article, appear to use "progenote" incorrectly, to mean "the most recent common ancestor of all modern forms of life" (*1*). Whether or not this most recent common ancestor was a progenote (as opposed to being a full-fledged genote) is not a fact, but one of the key unanswered evolutionary questions (*5*). One hopes that it will some day be answered through the sequencing of the appropriate prokaryotic genomes.

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REFERENCES

1. S. A. Benner, A. D. Ellington, A. Tauer, *Proc. Natl. Acad. Sci. U.S.A.* **86**, 7054 (1989).
2. C. R. Woese and G. E. Fox, *J. Mol. Evol.* **10**, 1 (1977).
3. C. R. Woese, *Proc. Natl. Acad. Sci. U.S.A.* **54**, 1546 (1965).
4. ———, *The Genetic Code* (Harper & Row, New York, 1967); in *Evolution from Molecules to Men*, D. S. Bendall, Ed. (Cambridge Univ. Press, Cambridge, England, 1983), pp. 209–233.
5. ———, *Microbiol. Rev.* **51**, 221 (1987).