Letters

Hot Little Pond?

M. Mitchell Waldrop's article (Research News, 23 Nov., p. 1078) reviews the opinions of the planetary scientists who dismiss Darwin's Warm Little Pond in favor of deep-sea hot springs as the locales where life may have originated. Even if it were necessary to accept the underlying assumption of the "ocean blasters" that life actually existed on Earth before the end of the late-stage bombardment about 3.8 billion years ago, there are several problems associated with this viewpoint.

Stanley Miller has already commented on both the necessity for a protracted origin of life and the difficulties involved with the formation and stability of organic molecules under intensely hot, deep-sea conditions. But Günter Wächtershäuser's novel ideas regarding pyrite are offered by Waldrop as a model consistent with deep-sea hot springs. In this connection it is important to note that Wächtershäuser's postulates require that the environment be rich in hydrogen sulfides that are in contact with metal sulfides and pyrite. This requirement places another constraint on the use of deep-sea hot springs. This is so because the deep oceans of the early earth are said to have been saturated with the dissolved ferrous iron necessary for banded iron-formation deposition. Clearly, the insolubility of ferrous sulfides means that the *deep* sea cannot have been a place where both hydrogen sulfide and ferrous iron were present in excess. The geochemists cannot have their reduced iron and the prebiotic chemists their reduced sulfur in the deep ocean at the same time.

If hot springs were necessary, it is more likely that they were located at the surface of the earth where the "food stuffs" hydrogen, hydrogen sulfides, elemental sulfur, and sulfates can all have been available, as they are for the various thermofile bacteria today. In surface hot springs the important heating of the deep sea would still have been available, but the equally important cooling and the wetting and drying necessary for oligopeptide-nucleotide concentration and growth were also there. Of course, in this scenario life would have to have originated after the early "impact frustration" tapered off, a viewpoint that Preston Cloud (*1*) felt the very evidence for asteroid pounding itself supported. The ocean blasters need to specify why it is necessary to have life originate so early that it must have been subjected to the indignities of bombardment, how dissolved iron and sulfide could have been available at the same time in the same place, and how prebiotic nucleotide bonds grew and survived in the deep sea. Darwin may have been wrong only in his assessment of the temperatures required. Goodbye Warm Little Pond, Hello Hot Little Pond?

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REFERENCES

1. P. Cloud, *Sci. Am.* **249**, 181 (September 1983).

Royal Shorthand

One hesitates to contradict one of the world's most celebrated philosophers, Karl R. Popper (with Günter Wächtershäuser, Letters, 23 Nov., p. 1070), but the issue is a question of fact, and Sir Karl is well known for his commitment to a criterion of falsification.

Popper and Wächtershäuser quote the famous motto of the Royal Society of London—"*Nullius in verba*"—and then, unfortunately, also give the canonical mistranslation ("there is nothing in words," in their version).

Although I am sure we all agree with their central contention that facts are more important than words, the motto of the oldest and most venerated of English scientific societies deserves its proper citation, especially since the full quote is such a lovely statement embodying such an important principle for all of us.

The motto is so often mistranslated because "*Nullius in verba*" is shorthand for a longer statement, a famous line from Horace's *Epistulae*:

> *Nullius addictus iurare in verba magistri,*
> *quo me cumque rapit tempestas, deferor hospes*
> (I am not bound to swear allegiance
> to the word of any master,
> Where the storm carries me,
> I put into port and make myself at home).

Thus, the motto advocates freedom of thought and action, not the insignificance of words. (We go astray by misreading the genitive singular "*nullius*" as the nominative "*nullus*" and by not recognizing the abbreviated citation.)

A valuable and general point does emerge from the correction. Words mean nothing in themselves, but we communicate by them and must be clear. We so often fail (not only in speaking to the general public, but even to our scientific colleagues in other subdisciplines) because we use the shorthands of our contemporary jargon and don't even recognize our elisions. Any well-educated, 17th century gentleman (a group including nearly all English scientists of the time) knew that "*Nullius in verba*" would convey a common message to all club members, just as we feel no need to explicate the terms in $E = mc^2$. We should remember that our modern shorthands can confuse others, just as we misconstrue the motto of our predecessors because numbers have superseded Latin as our imprimatur.

But enough of this. While we strive to keep words clear, we shall also remember Horace's more important advice—"*inter silvas Academi quaerere verum*" (to seek for truth in the garden of Academus).

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Carcinogenesis Models

The Perspective "Too many rodent carcinogens: Mitogenesis increases mutagenesis" by Bruce N. Ames and Lois Swirskey Gold (31 Aug., p. 970) and the article "Cell proliferation in carcinogenesis" by S. M. Cohen and L. B. Ellwein (31 Aug., p. 1001) raise significant, but often overlooked, questions about the process of carcinogenesis and the manner in which the carcinogenicity of new compounds is tested.

Mitogenesis, induced by physiological growth factors in platelets, promotes the induction of the neoplastic phenotype in carcinogen-treated cells. I have recently reported that the platelet-derived growth factor (PDGF), which is mitogenetic, is also a potent promoter of neoplastic transformation in the C3H/10T1/2 fibroblast model of carcinogenesis (*1*). I have also reported that vitamin A, an effective antipromoter in vivo and in vitro, inhibits the mitogenic response of preneoplastic cells to PDGF and EGF. (*2*).

Several noteworthy investigators have reported that wound-healing is as effective as the phorbol esters in promoting tumor formation in the mouse skin model of carcinogenesis (*3*). Apparently, this tumor promotion results from the exposure of preneoplastic cells to growth factors as a consequence of microvascular leakiness and platelet degranulation in response to 12-*O*-tetradecanoyl phorbol-13-acetate treatment. Thus, the apparent carcinogenicity of new compounds may be the result of oxidative damage and the cellular proliferation associ-