

LETTERS

Ecosystems of Deep-Sea Vents

H. W. Jannasch and M. J. Mottl conclude their interesting article on the geomicrobiology of deep-sea hydrothermal vents (23 Aug., p. 717) with a misleading statement. They state that vent ecosystems depend "on geothermal (terrestrial) rather than solar energy." Nine of the ten chemical reactions they discuss require oxygen or oxidants that exist on the earth only because of the presence of atmospheric oxygen. Atmospheric oxygen derives entirely from solar energy. Because it is impossible to specify whether the oxidant or the reductant is the source of the energy from a chemical reaction, these communities *do* depend on solar energy, and they might not persist in the permanent absence of light.

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Although Jannasch and Mottl state clearly that the growth of microorganisms near hydrothermal vents depends on the energy released by reactions between reducing substances in the vent plume and oxidizing agents such as O₂, nitrate, and sulfate in ambient sea water, they refer several times to the plume as the energy source. Is it not more correct to say that the *reaction* is the source of the energy? If so, the reducing and oxidizing agents are equally important. Because the latter are products of photosynthesis in surface water, the suggestion at the end of their extremely interesting article that this ecosystem could survive a "nuclear winter" becomes doubtful.

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Both comments focus on the same point: the actual source of energy is the reaction between the reductant and the oxidant, not the reductant per se. Therefore, both are equally important in chemosynthesis. This is certainly true from a strictly physico-chemical point of view. We emphasized in our article the geothermal origin of the inorganic reductant (in a biochemical context often described as the electron donor or energy source), and we certainly do not want to imply that the existence of the primarily aerobic deep-sea ecosystems could be independent of the photosynthetically

produced free oxygen during a "permanent" absence of light from the surface of the globe. We suggested that the immense store of free oxygen in the oceans for survival of the chemosynthetically supported deep-sea ecosystems might outlast a conceivable catastrophic darkening of the globe's surface ("nuclear winter" could be one of them) given the relatively minute oxygen consumption involved. Anaerobic chemosynthesis at deep-sea vents, on the other hand, is independent of free oxygen, using H₂ as the reductant and CO₂ as the oxidant, both produced geothermally. The existence of anaerobic ecosystems in a permanent absence of light is, of course, highly speculative, but not "misleading."

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Colon Cancer Screening

Gina Kolata's article "Debate over colon cancer screening" (Research News, 16 Aug., p. 636), although comprehensive, contains no mention of biologic markers valued by epidemiologists seeking to prevent colon cancer on a national scale (1).

I refer specifically to an assay of the enzyme ornithine decarboxylase (ODC) in the colon wall applied to screening of family members who are at risk for, but do not (yet) show any telltale signs of, polyp formation. Developed by Gordon Luk and Stephen Baylin at Johns Hopkins University Medical Center (2), the ODC assay reliably predicts progression from polyposis to colon cancer. ODC catalyzes production of polyamines, which earlier studies (3) implicated in overgrowth of the colonic lining. Hence, reasoning that heightened ODC activity signifies malignant transformation, Luk and Baylin measured activity of the enzyme in biopsy specimens of colonic mucosa from 60 adult subjects divided into three groups: normal, at risk for polyposis, and overt bearers of colonic polyps (2). ODC activity rose, commensurate with the cellular condition of the mucous membrane, ranging from flat through disorganized (dysplastic) to malignant (neoplastic).

If implemented nationwide, the promising ODC assay would not only overcome administrative, economic, and technical problems but also would enable epidemiologists to target well-defined high-risk populations for nutrition-

al and drug interventions. These strides could lead to definitive identification and elimination of causative agents, thus achieving primary prevention of colon cancer.

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References and Notes

1. B. C. Morson, in *Precancerous Lesions of the Gastrointestinal Tract*, P. Sherlock, B. C. Morson, L. Barbara, U. Veronesi, Eds. (Raven, New York, 1983), pp. 255-59.
2. G. D. Luk and S. B. Baylin, *New Engl. J. Med.* 311, 80 (1984).
3. J. M. Gaugas, Ed., *Polyamines in Biomedical Research* (Chichester (Wiley), 1980).
4. H. G. Williams-Ashman and Z. N. Canellakis, *Perspect. Biol. Med.* 445 (spring 1979).

Erratum: Figure 10 on page 351 of the Research Article "Constitutive and conditional suppression of exogenous and endogenous genes by anti-sense RNA" by J. G. Izant and H. Weintraub (26 July, p. 345) was reproduced erroneously, so that the green stain (NBD-phalloidin) of the actin filaments was not chromatically resolved. The micrographs are intended to document the specific disruption of the actin microfilament distribution, while the RNA and DNA staining pattern (orange-red) was unaffected. The correct figure and legend appear below.

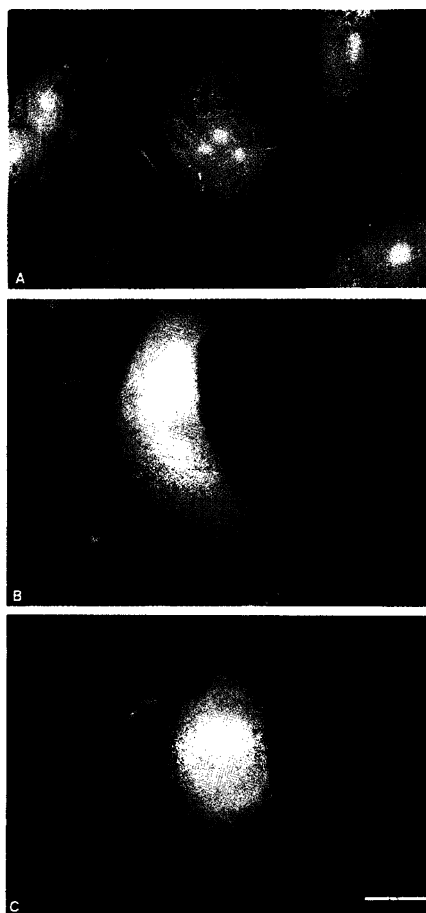


Fig. 10. Inhibition of actin cable formation by anti-sense actin RNA. Capped RNA was synthesized in vitro with SP6 RNA polymerase and plasmids that direct the synthesis of either (A) sense or (B and C) anti-sense actin RNA. In vitro capping was achieved by including 5 mM diguanosine triphosphate G(5')p₃(5')G in the reaction (14). 0.1 pl of RNA at 0.5 µg/µl was injected into BSC-1 cells, and after 30 hours the cells were fixed and stained with fluorescent phalloidin (green) (9) to stain filamentous actin and propidium iodide (red-orange) to stain DNA and RNA.