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Emerging Plant Science

frontiers in biotechnology





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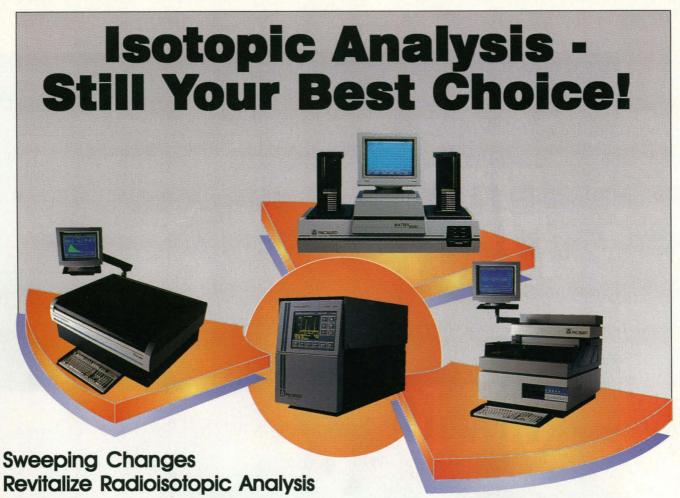
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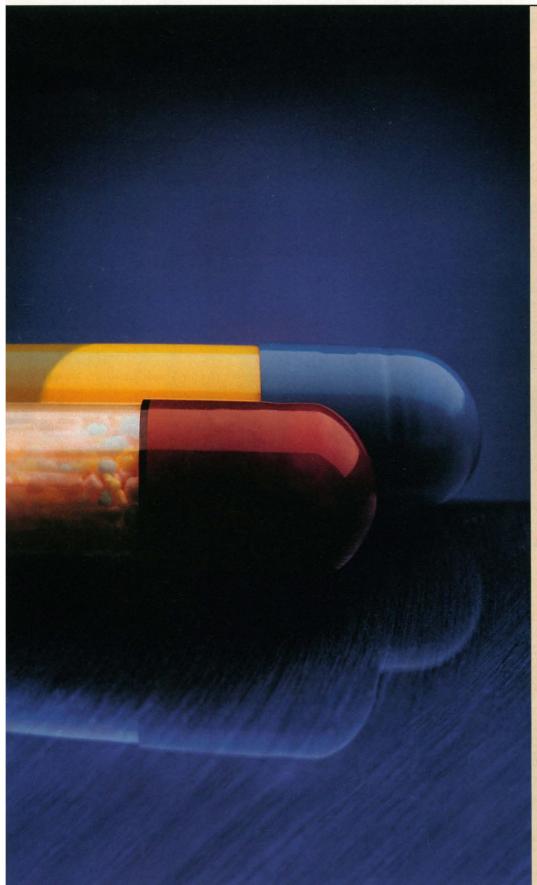
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COVER

The plant stress hormone ethylene inhibits cell elongation in the hypocotyl and apical hook of seedlings grown in the dark. The scanning electron micrograph shows this ethylene-mediated phenotype in an Arabidopsis seedling (diameter ~0.3 millimeter at base of image). Mutational analysis of the response has led to the identification of genes in the ethylene signal transduction pathway. See page 667. In this special issue are collected News stories, Articles, and Reports on various topics in emerging plant science. [Image: J. R. Ecker, G. Ahlstrand, and J. Hayden]



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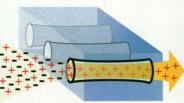
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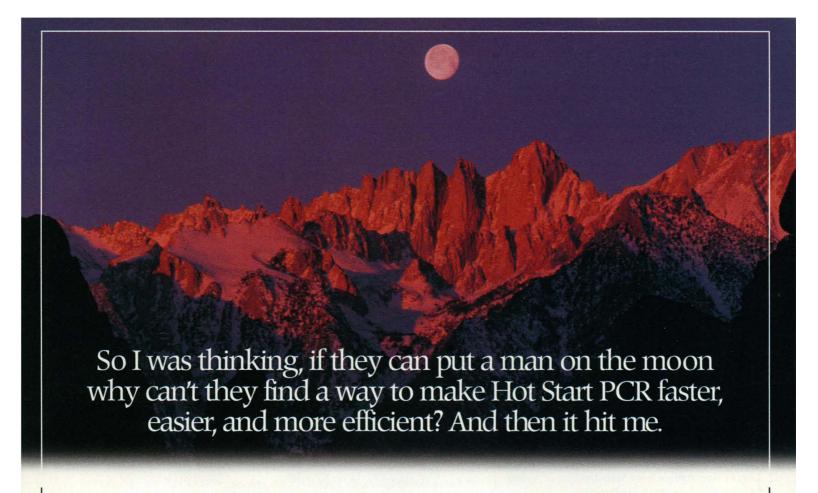
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THIS WEEK IN SCIENCE

edited by DAVID LINDLEY

Lost in space

The surface of Mars betrays signs that it once had running water, but now the atmosphere is far too thin to allow water to exist as a liquid. Earlier models had indicated that the solar wind might have eroded about onesixth of a bar of early martian atmosphere, but a more detailed analysis by Kass and Yung (p. 697) indicates that as much as 3 bars could have been sputtered away. An atmosphere dense enough to allow surface water may thus have been lost to space over the last 3.5 billion years.

Tube's gold

Membranes with pores of molecular size are finding increasing numbers of applications. By depositing gold for varying times onto polycarbonate filters with pores 25 nanometers in diameter, Nishizawa et al. (p. 700; see also news by Pool, p. 639) made porous metallic membranes with uniform pore size down to less than a nanometer; the longer the gold deposition, the narrower the channels become. Placing the metallic membranes in a suitable ionic solution causes excess charge to build up on the channel surfaces, and either cations or anions can be selected to pass through according to the sign of the surface charge.

Deficit dispelled

Stratospheric models meant to reproduce upper atmosphere chemistry generally produce too little ozone to compensate for calculated ozone destruction pathways, creating a strictly theoretical ozone deficit. Combining abundances measured from the Upper Atmosphere Research Satellite with an atmo-

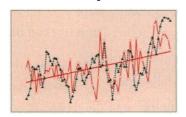
Repetition causes stumbling

In fragile X syndrome, a CGG triplet in an untranslated region of the gene FMR1 can be repeated hundreds of times, as against about 30 times in normal individuals. FMR1 is abnormally methylated, and the absence of the protein it normally expresses, FMRP, leads to the mental retardation symptomatic of the syndrome. Feng et al. (p. 731) cloned several fibroblasts from an individual with a mild form of fragile X, and found triplet repeats numbering from 57 to 285. In all cells, FMR1 was unmethylated and its messenger RNA was present, but levels of FMRP varied dramatically, dropping off rapidly in cells with more than 200 repeats. Absence of FMRP is thus due to translational failure, apparently a result of the translational apparatus stalling as it moves along the RNA. The result implies that therapies aimed at reversing FMR1 methylation may not solve the problem.

spheric chemistry model, Crutzen et al. (p. 705) find no ozone deficit; if anything, the model now has insufficient ozone destruction. The difference between this and earlier results is due to downward revision of the measured ozone abundance, a more complete chemical model, and a full treatment of diurnal abundance variations.

That sinking feeling

A main goal of the TOPEX/POSEIDON satellite was accurate measurement of sea level changes from global altimetry data of the sea surface. Errors associated with previous satel-



lite measurements or data from coastal stations obscured short-term trends. Nerem (p. 708) reports that the first 2 years of TOPEX/POSEIDON data show that sea level rose by about 4 millimeters per year, but it is still not clear whether this sig-

nal is related to global warming or is a short-term response to some other process.

Taking the B road

High-affinity B cells from the germinal center (GC) may develop either into memory B cells or plasma cells. Arpin et al. (p. 720) were able to grow undifferentiated GC B cells in culture, in the presence of interleukin (IL)-2, IL-10, and T cells expressing the ligand for CD40. With the CD40 ligand present, memory B cells developed; when it was removed, plasma cells were generated. Re-addition of CD40 ligand had no effect, indicating that its absence is a switch causing terminal differentiation of GC B cells into plasma cells.

Receptor's role

The endogenous role of the aryl hydrocarbon receptor (AHR), which binds to dioxin and other toxic substances, has been mysterious. To investigate its endogenous function, Fernandez-Salguero *et al.* (p. 722; see also news by Stone, p. 638) generated a mouse line lacking AHRs. Half of these mice died shortly

after birth. The survivors had abnormally developed livers and fewer lymphocytes in their peripheries, indicating that the AHR is required for normal development of two unrelated systems. These mice may prove useful in chemical risk assessment and the study of how toxins contribute to carcinogenesis.

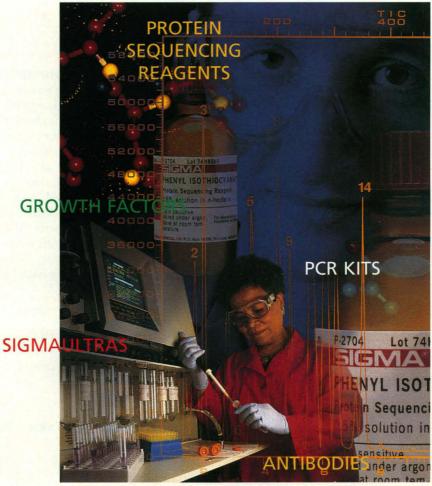
Plant-animal signaling

Cellular signaling mechanisms using Ca²⁺ occur in both plants and animals, and the regulator inositol 1,4,5-trisphosphate (InsP₃) exists in both. Allen et al. (p. 735) show that Ca²⁺ release in vacuolar membranes of the storage root of the red beet is regulated not only by InsP₃ but also by cyclic adenosine 5'diphosphoribose (cADPR), which had previously been identified only in animals. The occurrence of both pathways in the same membranes suggests mechanisms by which the Ca2+ response to a variety of stimuli could be individually tailored. See also the special section in this issue on plants (p. 653).

No repair anywhere

Patients with hereditary nonpolyposis colorectal cancer (HNPCC) have germline mutations in genes encoding DNA repair proteins. DNA repair defects are readily apparent in the patients' tumors, which show a high rate of mutation. Contrary to expectation, Parsons et al. (p. 738) found that at least in a subset of HNPCC patients, the repair defects occurred in normal cells as well. These results suggest that an elevated mutation rate is compatible with normal human development and may not in itself be a sufficient driving force for tumorigenesis.

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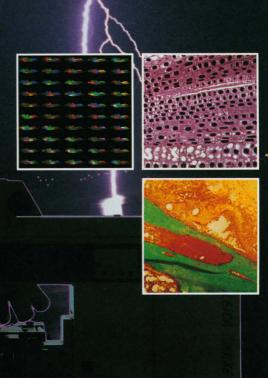


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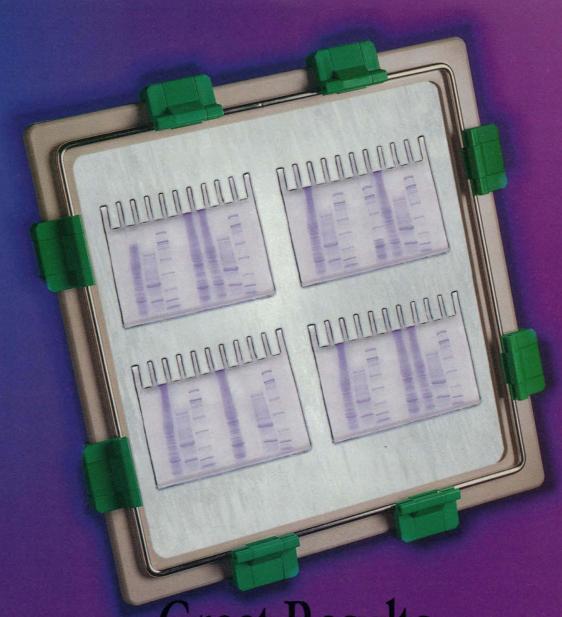
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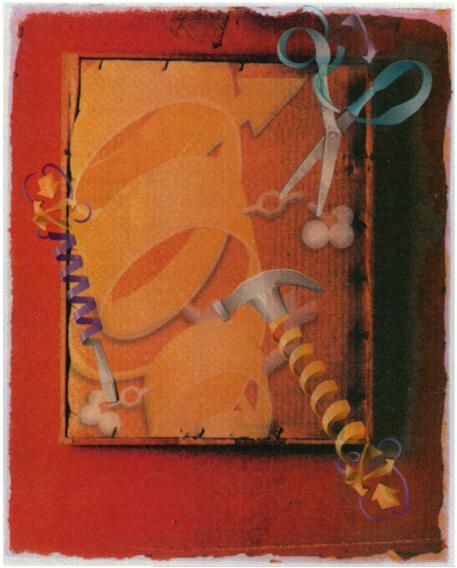
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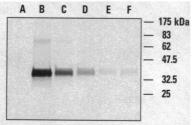
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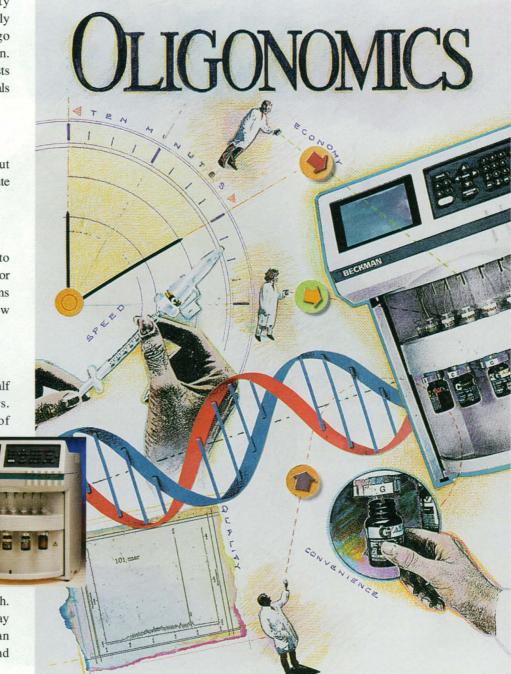
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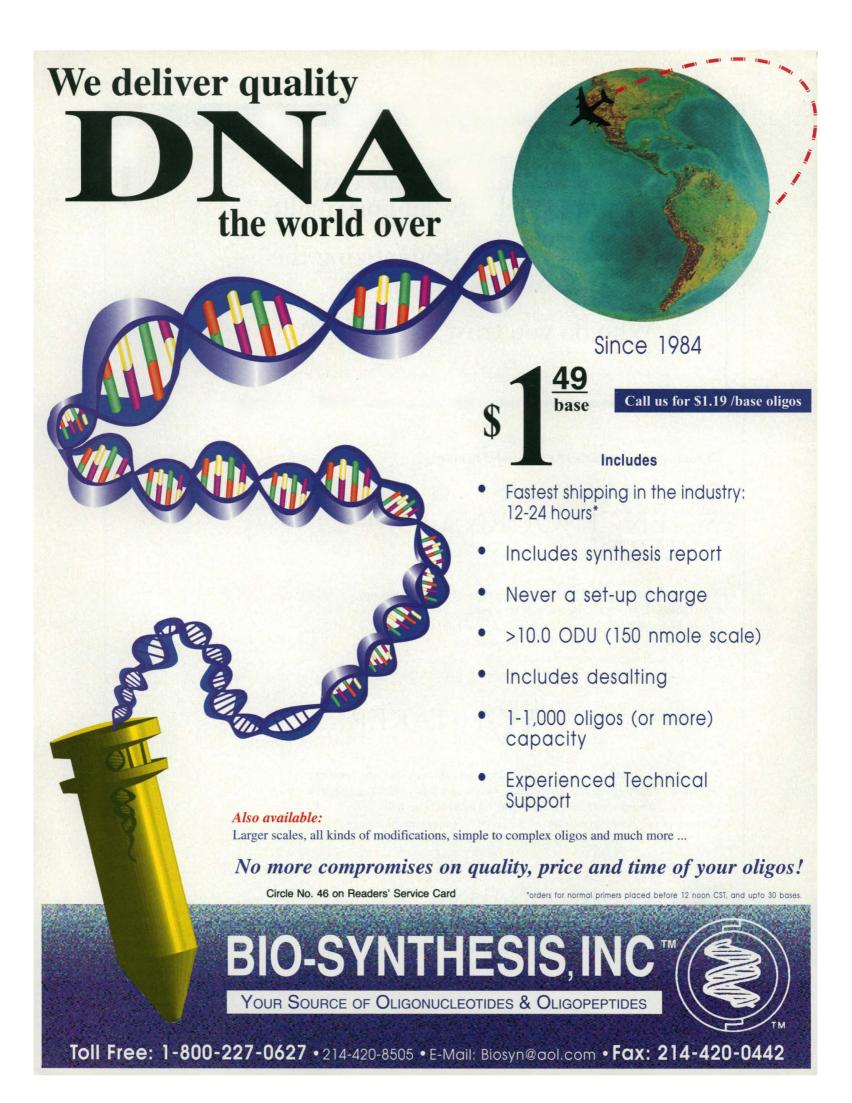
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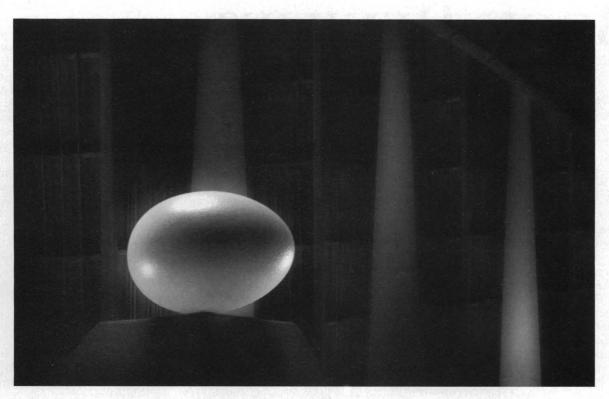
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