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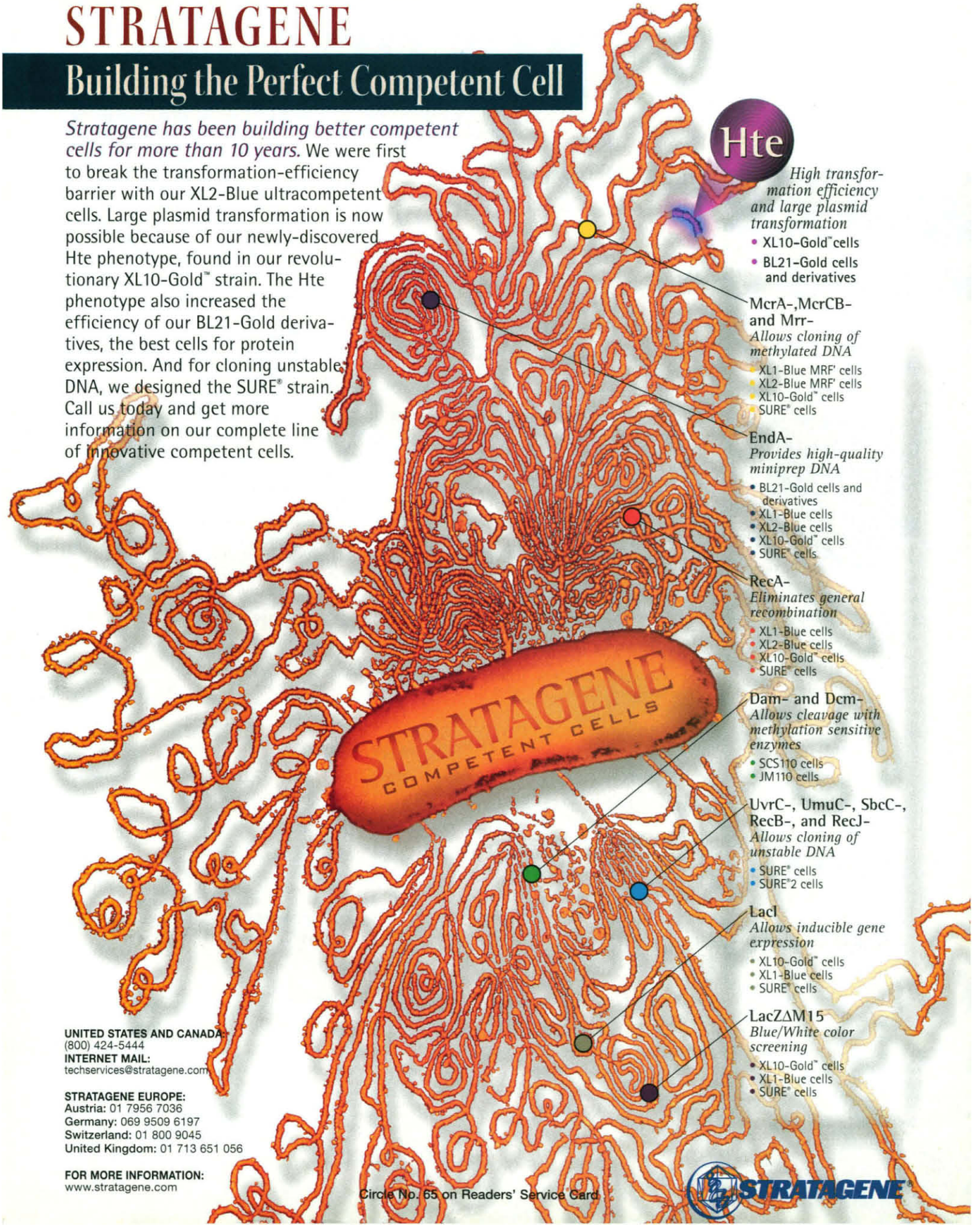

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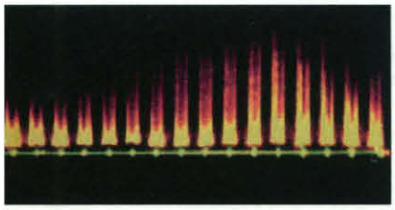

994
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1007 & 1039
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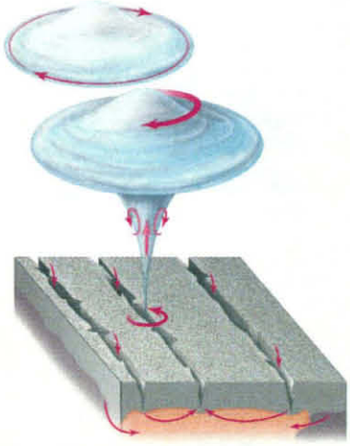

1034 & 1052
Taking a spin in the ocean

NEWS & COMMENT

Hubris and the Human Genome 994
Picking Up the Pace of Sequencing 995

The Power of the Front Page of *The New York Times* 996
The Roadblocks to Angiogenesis Blockers 997

Mars 2001 Mission Hits the Wall 998

State Department Sees S&T Weaknesses 998

German Biotechs Form Gene Venture 999

Earthquake Prediction: Japan Urged to Drop Short-Term Goal 1000

Allen Roses: From 'Street Fighter' to Corporate Insider 1001
The Alzheimer's Gene Puzzle 1002
A Clash Over Testing for Alzheimer's Disease 1004

RESEARCH NEWS

Probing the Biology of Emotion 1005
Unmasking the Emotional Unconscious 1006

Biggest Extinction Looks Catastrophic 1007

Exploding Stars Flash New Bulletins From Distant Universe 1008

On the Trail of Supercharged Hydrogen 1009

New Probes Open Windows on Gene Expression, and More 1010

SCIENCE'S COMPASS

Policy

Interpretation of International Test Score Comparisons 1030
I. C. Rotberg

Books and New Media

Early Neuro Networks 1032
M. Constantine-Paton
Vignette 1033
Browsings 1033

Research

A New Spin on Hydrothermal Plumes 1034
K. G. Speer
The Expanding Role of Cell Cycle Regulators 1035
T. Jacks and R. A. Weinberg
Landscaping the Cancer Terrain 1036
K. W. Kinzler and B. Vogelstein

RESEARCH ARTICLE

U/Pb Zircon Geochronology and Tempo of the End-Permian Mass Extinction 1039
S. A. Bowring, D. H. Erwin, Y. G. Jin, M. W. Martin, K. Davidek, W. Wang

DEPARTMENTS

NETWATCH 975

THIS WEEK IN *SCIENCE* 977

EDITORIAL 983
Toward a Transparent Federal S&T Budget
J. Bingaman and J. Lieberman

LETTERS 983
Haeckel, Embryos, and Evolution: M. K. Richardson, J. Hanken, L. Selwood, G. M. Wright, R. J. Richards, C. Pieau, A. Raynaud • Demographic Thinking: J. W. Vaupel • Bird, Dinosaur Link: K. Padian • Science in Vietnam: T. V. Nguyen • A Good Estrogen: V. S. Pribluda and S. J. Green • Causal Systems in Ecology: J. H. Reynolds • Corrections and Clarifications

SCIENCESCOPE 993

RANDOM SAMPLES 1013
Neuron Growth and ApoE • Moscow Dino Egg Scramble • Authentic *Deep Impact*

ESSAYS ON SCIENCE AND SOCIETY 1014
Science as a Craft Industry
F. J. Dyson

TECH.SIGHT 1099

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COVER

Skull and head reconstruction of *Majungatholus atopus*, a large (7- to 9-meter) predatory dinosaur from the Late Cretaceous of Madagascar. The well-preserved skull (~57 centimeters long) is very similar to those of contemporaneous forms from Argentina and India, suggesting that land connections among the host landmasses (via Antartica) were retained much longer than previously thought. See page 1048. [Photograph: J. Weinstein, ©The Field Museum, GEO86106.2.9c. Artwork: B. Parsons]

REPORTS

Chemical Amplification: Continuous-Flow PCR on a Chip 1046
M. U. Kopp, A. J. de Mello, A. Manz

Predatory Dinosaur Remains from Madagascar: Implications for the Cretaceous Biogeography of Gondwana 1048
S. D. Sampson, L. M. Witmer, C. A. Forster, D. W. Krause, P. M. O'Connor, P. Dodson, F. Ravoavy

Tracking the Evolution of a Hydrothermal Event Plume with a RAFOS Neutrally Buoyant Drifter 1052
J. E. Lupton, E. T. Baker, N. Garfield, G. J. Massoth, R. A. Feely, J. P. Cowen, R. R. Greene, T. A. Rago

Earthquakes on Dipping Faults: The Effects of Broken Symmetry 1055
D. D. Oglesby, R. J. Archuleta, S. B. Nielsen

Percolation of Core Melts at Lower Mantle Conditions 1059
M. C. Shannon and C. B. Agee

Localized Reconnection in the Near Jovian Magnetotail 1061
C. T. Russell, K. K. Khurana, D. E. Huddleston, M. G. Kivelson

Ferromagnetism in $LaFeO_3$-$LaCrO_3$ Superlattices 1064
K. Ueda, H. Tabata, T. Kawai

Inducible Repair of Thymine Glycol Detected by an Ultrasensitive Assay for DNA Damage 1066
X. C. Le, J. Z. Xing, J. Lee, S. A. Leadon, M. Weinfeld

Inhibitory Function of $p21^{Cip1/WAF1}$ in Differentiation of Primary Mouse Keratinocytes Independent of Cell Cycle Control 1069
F. Di Cunto, G. Topley, E. Calautti, J. Hsiao, L. Ong, P. K. Seth, G. Paolo Dotto

Genetic Evaluation of Suspected Cases of Transient HIV-1 Infection of Infants 1073
L. M. Frenkel, J. I. Mullins, G. H. Learn, L. Manns-Arcuino, B. L. Herring, M. L. Kalish, R. W. Steketee, D. M. Thea, J. E. Nichols, S.-L. Liu, A. Harmache, X. He, D. Muthui, A. Madan, L. Hood, A. T. Haase, M. Zupancic, K. Staskus, S. Wolinsky, P. Krogstad, J. Zhao, I. Chen, R. Koup, D. Ho, B. Korber, R. J. Apple, R. W. Coombs, S. Pahwa, N. J. Roberts Jr.

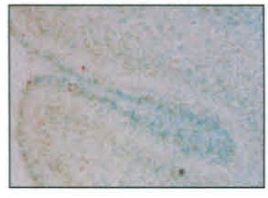
Large-Scale Identification, Mapping, and Genotyping of Single-Nucleotide Polymorphisms in the Human Genome 1077
D. G. Wang, J.-B. Fan, C.-J. Siao, A. Berno, P. Young, R. Sapolsky, G. Ghandour, N. Perkins, E. Winchester, J. Spencer, L. Kruglyak, L. Stein, L. Hsie, T. Topaloglou, E. Hubbell, E. Robinson, M. Mittmann, M. S. Morris, N. Shen, D. Kilburn, J. Rioux, C. Nusbaum, S. Rozen, T. J. Hudson, R. Lipshutz, M. Chee, E. S. Lander

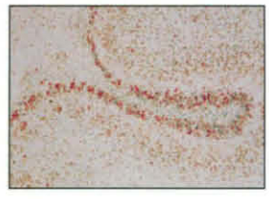
RasGRP, a Ras Guanyl Nucleotide–Releasing Protein with Calcium- and Diacylglycerol-Binding Motifs 1082
J. O. Ebinu, D. A. Bottorff, E. Y. W. Chan, S. L. Stang, R. J. Dunn, J. C. Stone

Mutations in the *SMAD4/DPC4* Gene in Juvenile Polyposis 1086
J. R. Howe, S. Roth, J. C. Ringold, R. W. Summers, H. J. Järvinen, P. Sistonen, I. P. M. Tomlinson, R. S. Houlston, S. Bevan, F. A. Mitros, E. M. Stone, L. A. Aaltonen

Requirement for Atm in Ionizing Radiation–Induced Cell Death in the Developing Central Nervous System 1089
K.-H. Herzog, M. J. Chong, M. Kapsetaki, J. I. Morgan, P. J. McKinnon

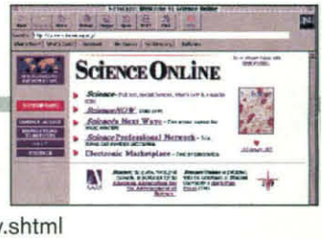
***COI1*: An *Arabidopsis* Gene Required for Jasmonate-Regulated Defense and Fertility** 1091
D.-X. Xie, B. F. Feys, S. James, M. Nieto-Rostro, J. G. Turner





1089

Atm and control of cell death in the central nervous system



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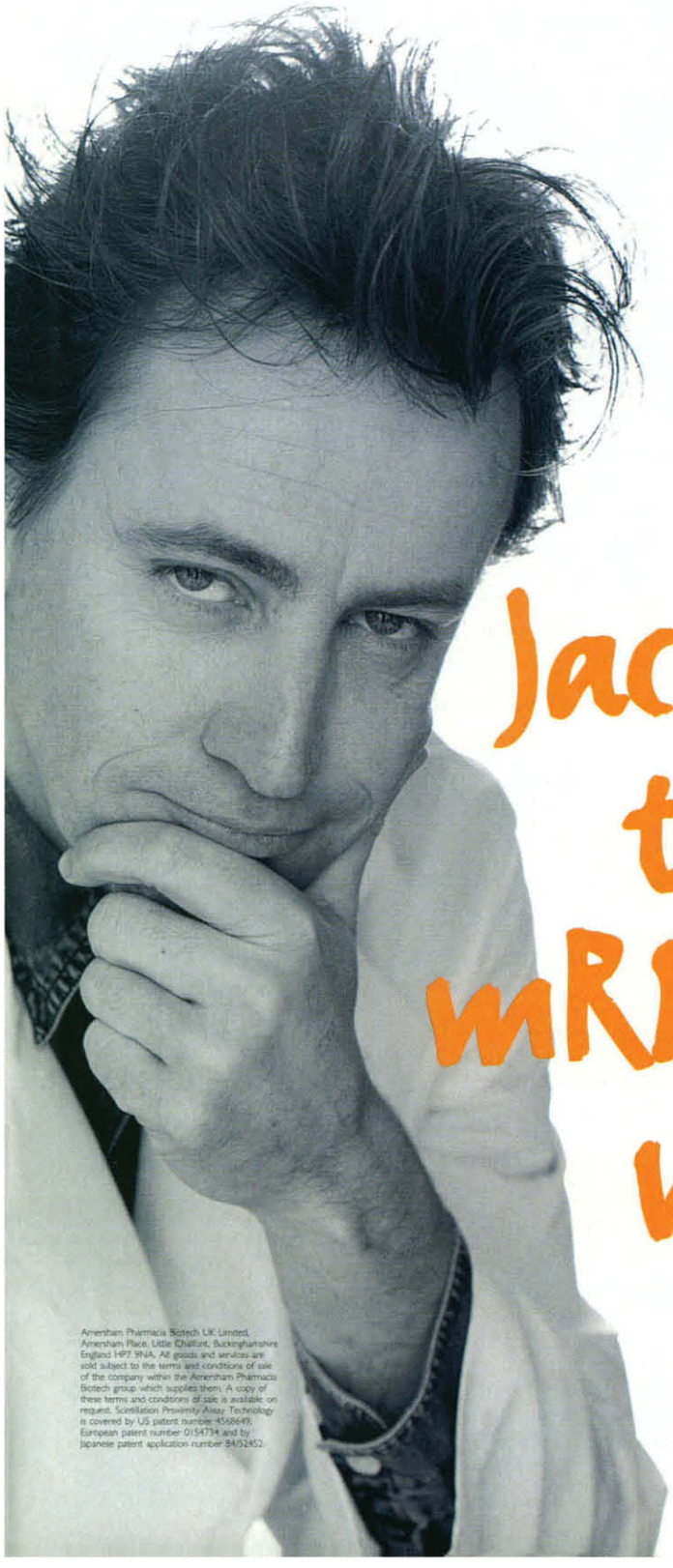

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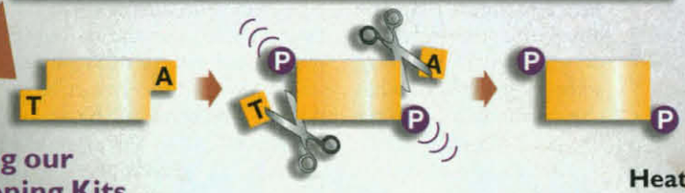

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*The Polymerase Chain Reaction (PCR) process is covered by patents owned by Hoffmann-La Roche.
†Conditions that use DNA polymerases lacking 3'→5' exo-activity (*e.g., Taq, Tth*)

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edited by PHIL SZUROMI

Ocean twister

The steady venting of fluids from mid-ocean ridge hydrothermal systems at black smokers is dwarfed by event plumes or megaplumes that mark the rapid release of hot water associated with intrusion of new magma. These plumes can extend for tens of kilometers above a ridge and persist for months; they were first recognized from their anomalous helium isotopic composition and have been used to trace ocean circulation. Although there have been several theoretical studies of how they persist, in situ observations have been lacking. Lupton *et al.* (p. 1052; see the commentary by Speer, p. 1034) were able to insert a neutrally buoyant tracking float into an event plume that formed over the Gorda Ridge in spring 1996. During a 60-day period, the float traced an anticyclonic path marking the circulation of water in the plume. Chemical and physical data collected at the beginning and end of this period imply that the plume remained coherent and maintained concentrations of iron particles and dissolved manganese. The data imply that such plumes could persist for periods greater than 1 year.

■

Performing PCR on a chip

The three steps of the polymerase chain reaction (PCR), melting the double-stranded DNA, binding specific primers, and enzymatically extending the primers, have been automated on a chip. Kopp *et al.* (p. 1046) developed a continuous flow system that temperature-cycles the sample and the necessary reactants through 20 rounds of amplification. At the highest flow rates, the process is complete in 90 s, and successive samples appear to be amplified without cross-contamination.

■

End of an era

The end-Permian extinction is the largest in Earth's history. Its age, 251 million years ago, corresponds to the eruption of the Siberian Traps, the largest flood basalt province on Earth. However, the cause of the extinction remains unclear, in part because the duration of the extinction and of the terminal part of the Permian has not been well resolved. Bowring *et al.* (p. 1039; see the news story by Kerr, p. 1006) provide uranium-lead dates on several volcanic tuff horizons straddling the Permian-Triassic boundary in China. The data imply that the final extinction pulse there, corresponding to loss of most of the marine species, lasted less than 1 million years and perhaps occurred within only a few hundred thousand years.

Fossil record of land bridges

Several fossil remains of vertebrates from the Upper Cretaceous (about 80 million years ago) from Madagascar, including a nearly complete skull of a predatory dinosaur, are described by Sampson *et al.* (p. 1048; see the cover). Similar fossils have been so far found only in South America and in India, which was still connected to Madagascar in the Late Cretaceous, but not yet in Africa. This distribution suggests that land bridges remained between South America and India through Antarctica into the Latest Cretaceous.

■

Fault asymmetries

The ground motion caused by a thrust (reverse) fault is greater than the ground motion caused by a normal fault for earthquakes of the same magnitude. Oglesby *et al.* (p. 1055) performed dynamic simulations of earthquakes on different fault geometries and found that the difference in the intensity of the ground motions is caused by time-dependent stress produced by the interaction of the earthquake stress field with Earth's free surface. These simulations will be helpful for characterizing previous earthquakes and modeling ground motion for future events.

■

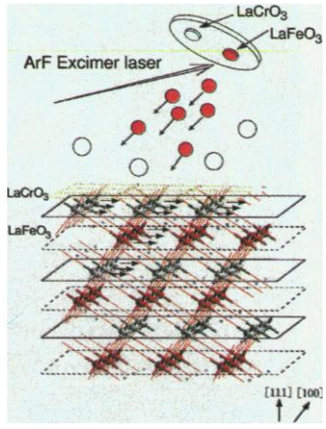
Ironing out the lower mantle

The formation of Earth's core required separation of iron melt from Earth's silicate mantle. Whether this separation involved percolation of melt through a solid silicate matrix or the settling of iron melt through a molten matrix may have depended on the nature of the pore network through $MgSiO_3$ perovskite, the dominant silicate phase in the lower mantle. Shannon and Agee (p. 1059) measured experimentally the wetting angle between iron melts and perovskite. The data imply that percolation of iron through the Earth's lower mantle might have been possible, unlike the case for Earth's upper mantle, where the mineralogy is different.

■

Forcing ferromagnetism

Interactions between magnetic ions in oxides can be estimated theoretically to predict whether



materials will exhibit ferromagnetism. Although theory predicts that Fe^{3+} and Cr^{3+} should be able to form ferromagnets in a perovskite structure, phase separation normally occurs in the bulk oxides and antiferromagnetic materials are formed. Ueda *et al.* (p. 1064) grew superlattices of alternating layers of $LaCrO_3$ and $LaFeO_3$ on the (111) face of $SrTiO_3$. They show that this material, which forces the proximity of Fe^{3+} and Cr^{3+} ions, is ferromagnetic.

■

Reconnected magnetotails

The magnetosphere of Jupiter is controlled by currents in the jovian interior, but the volcanically active moon, Io, is expected to upset the magnetosphere by spewing out tons of ions in a magnetized plasma. Theories suggest that one way to release this plasma sheet is to reconnect the jovian magnetotails and allow the plasma to flow out of the system. Russell *et al.* (p. 1061) have been sampling the magnetospheric field from the Galileo spacecraft since its insertion into orbit around Jupiter in December 1995. They have found evidence for transient reconnections of the magnetotail

(Continued on page 979)

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(Continued from page 977)

that supports this theory and helps explain how Jupiter can maintain a balanced magnetic flux in the presence of Io.

Alternative function for cell cycle inhibitor

The protein p21 inhibits cell division by inhibiting the activity of cyclin-dependent kinases and binding to proliferating cell nuclear antigen (PCNA). Such effects of p21 may contribute to terminal differentiation of cells by promoting withdrawal from the cell division cycle. However, Di Cunto *et al.* (p. 1069; see the commentary by Jacks and Weinberg, p. 1035) report that the opposite is true in the later stages of differentiation of mouse keratinocytes. In this situation, p21 has an inhibitory effect on expression of markers of terminal differentiation. These results are consistent with observations that the amounts of p21 first increase as the cells begin to differentiate and then diminish. Furthermore, the inhibitory effects of p21 on differentiation appear to be produced independently of its effects on cell division.

Lab error and transient HIV infection

Numerous cases have been reported in which infants of HIV-infected mothers appear to have been transiently infected. If validated, these findings would have significant implications for thinking about the ability of the host immune response to successfully fight HIV. However, when Frenkel *et al.* (p. 1073) reanalyzed 42 cases in which a transient infection was suspected, the results were more likely due to mislabeling or laboratory contamination. The authors suggest that criteria for the conclusive demonstration of a transient infection should include phylogenetic analyses on previously unmanipulated portions of the specimens in a manner that can minimize cross-contamination.

SNPing away at the human genome

New tools are becoming available for the identification of disease genes, characterization of human traits, forensic identification, and studies of human evolution and diversity. Wang *et al.* (p. 1077) present a proof-of-principle study to show that large-scale analysis of single-nucleotide polymorphisms (SNPs) in human DNA is feasible. They identified more than 3200 candidate SNPs across the entire human genome (with about 40 percent confirmed so far), constructed a map more than 2000 of the loci, and used 500 of these to create a genotyping chip.

Linking to Ras

The small guanine nucleotide–binding protein Ras participates in control of cell signaling pathways that regulate diverse cellular functions from cell division to neuronal activity in the brain. Ebinu *et al.* (p. 1082) describe a new guanine nucleotide releasing factor, RasGRP. Ras is active when GTP (guanosine triphosphate) is bound to it, and exchange factors like RasGRP activate Ras by facilitating release of bound guanosine diphosphate and binding of GTP. RasGRP is found primarily in the brain and contains protein domains that bind the intracellular signaling molecules calcium and diacylglycerol. RasGRP appears to link signals that alter intracellular concentrations of calcium and diacylglycerol to changes in Ras activity.

SMADs and cancer predisposition

Familial juvenile polyposis (JP) is an autosomal dominant disease characterized by a predisposition to hamartomatous polyps and gastrointestinal cancer. Howe *et al.* (p. 1086; see the commentary by Kinzler and Vogelstein, p. 1036) show that a subset of JP families carry germline mutations in the SMAD4/DPC4 gene on chromosome 18q21.1. SMAD4 is a key player in the transforming growth factor–β (TGF-β)–signaling pathway, which mediates cell growth inhibitory signals from the cell surface to the nucleus.

Jasmonic acid and auxin signaling

Jasmonic acid and its derivatives affect developmental and defensive responses in a great

variety of plants. Xie *et al.* (p. 1091) have cloned and mapped a gene from *Arabidopsis* that is required for responses to jasmonates. The *coi1* gene encodes a protein that has intriguing similarities the *tir1* gene, required for auxin signaling, suggesting that jasmonates and auxin may share some aspects of their signaling pathways. Both proteins include F-box motifs, which are found in proteins that form part of the ubiquitin-dependent protein degradation process.

Surveying the damage

DNA damage and its repair are key determinants in the early stages of carcinogenesis, cancer therapy, and aging. Le *et al.* (p. 1066) have developed an ultrasensitive assay for DNA damage that couples immunochemistry with capillary electrophoresis and laser-induced fluorescence detection. This assay, which is specific for radiation-induced thymine glycols, requires only nanogram amounts of DNA and can detect a single base modification among 10^9 normal bases. With appropriate affinity probes for other types of DNA damage and with further automation, the assay could be adapted for use in risk assessment.

Clues to *ATM* function

A key component in understanding how an abnormal gene can act to cause disease can come from insights into its normal function. The neurodegenerative disease ataxia telangiectasia is associated with mutations in the gene *ATM*. When Herzog *et al.* (p. 1089) have knocked out the *Atm* gene in mice, cells of the developing central nervous system were no longer as sensitive to ionizing radiation and showed a dramatic lack of programmed cell death. The authors speculate that *Atm* may be involved in a developmental checkpoint, and that mutations in this gene may allow damaged cells to survive during development, leading to problems later in life.

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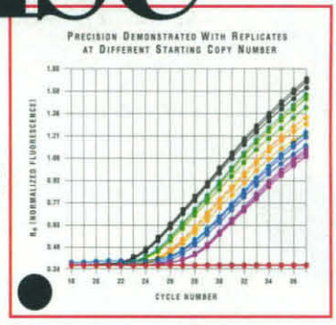

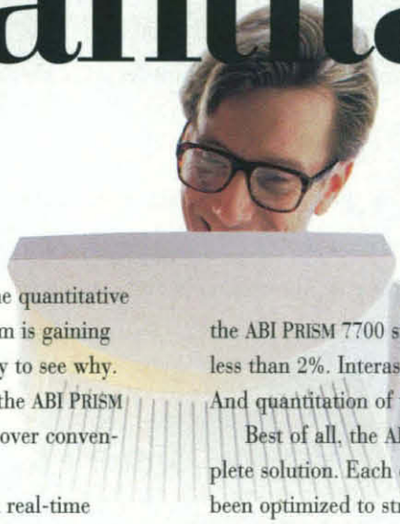

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1. Heid, Christian A., et al. 1996. Real Time Quantitative PCR. Genome Research 6: 986-994. from Molecular Endocrinology
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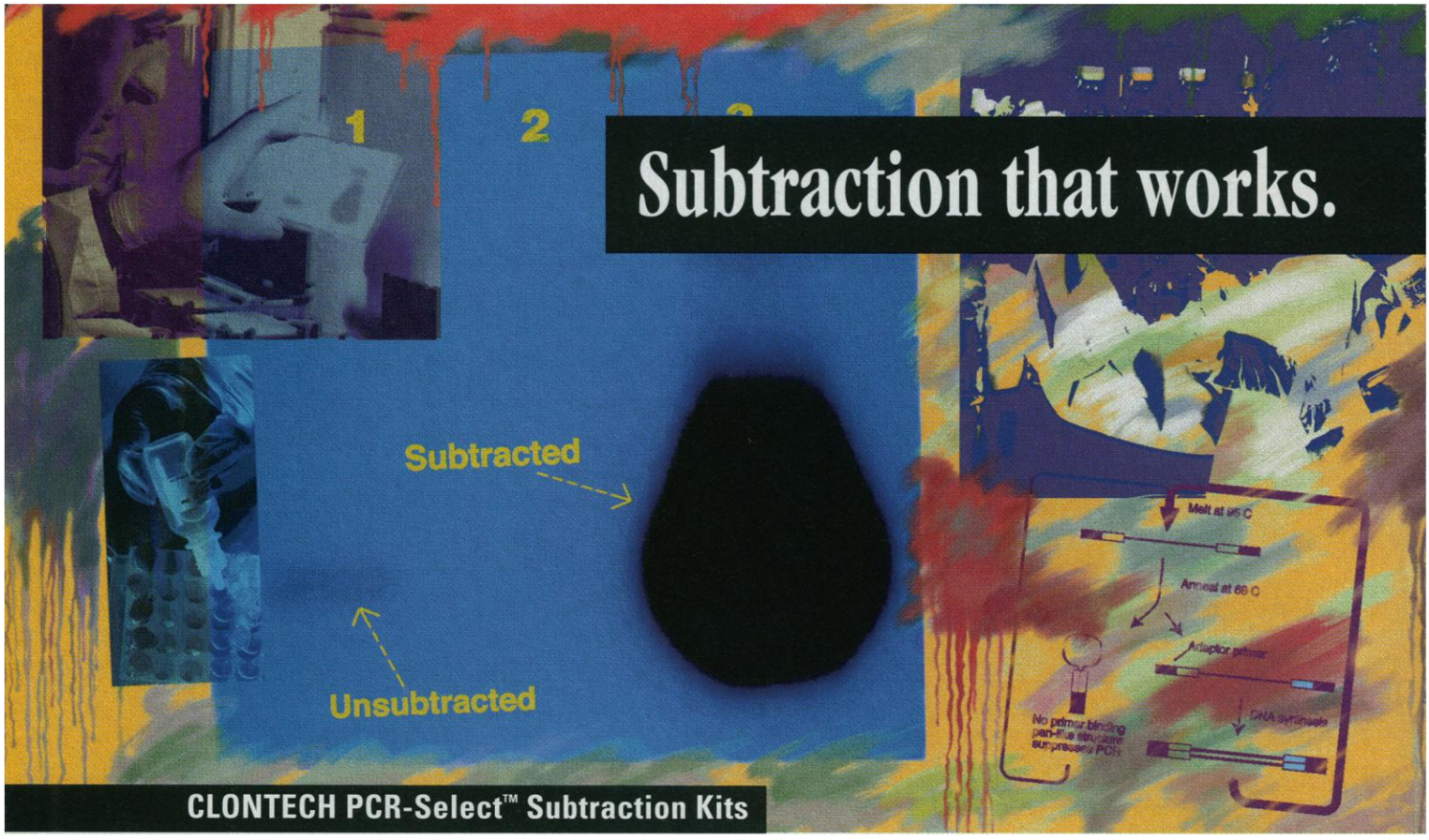

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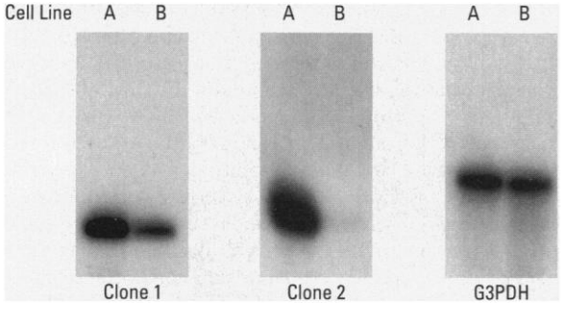

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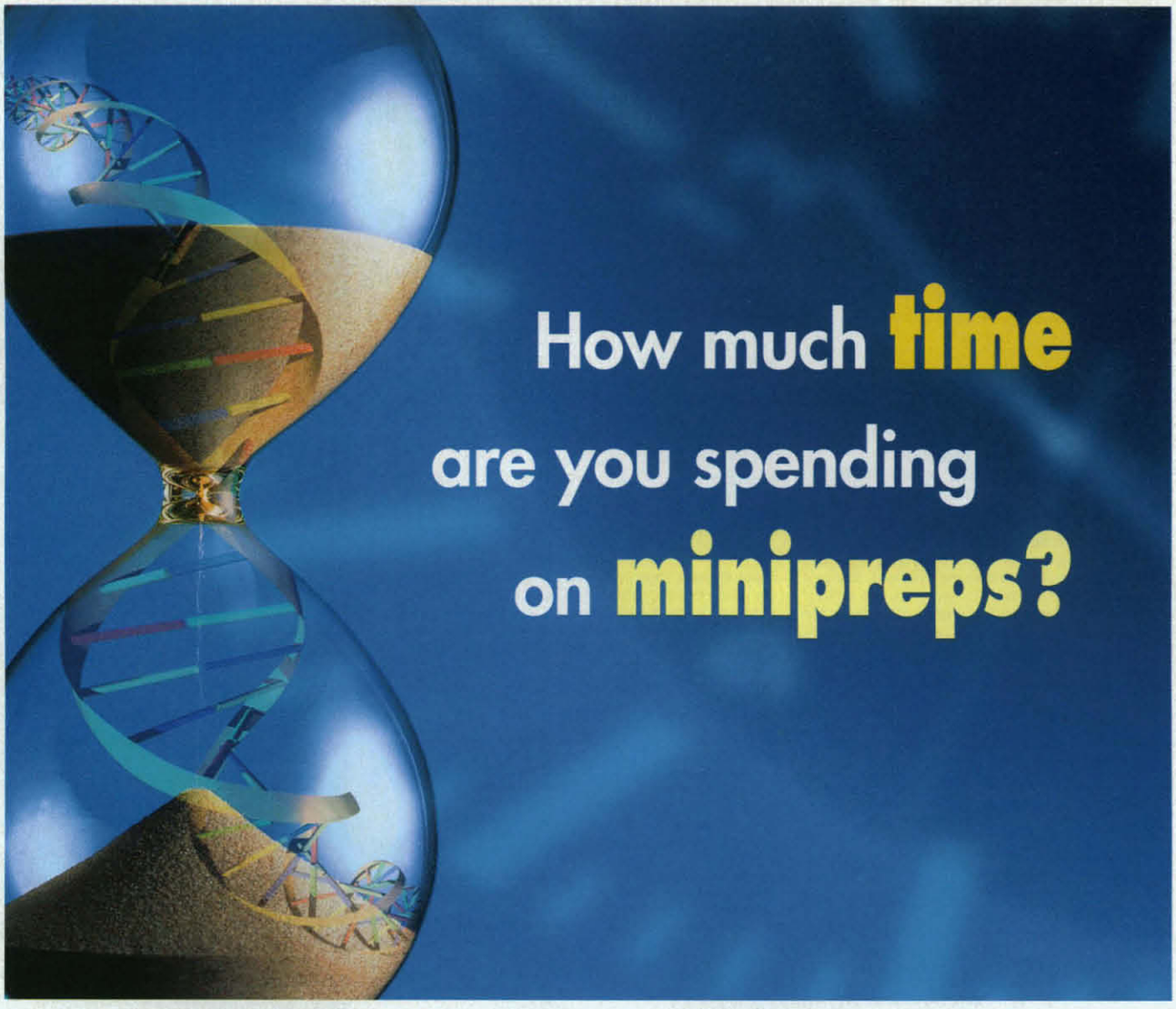

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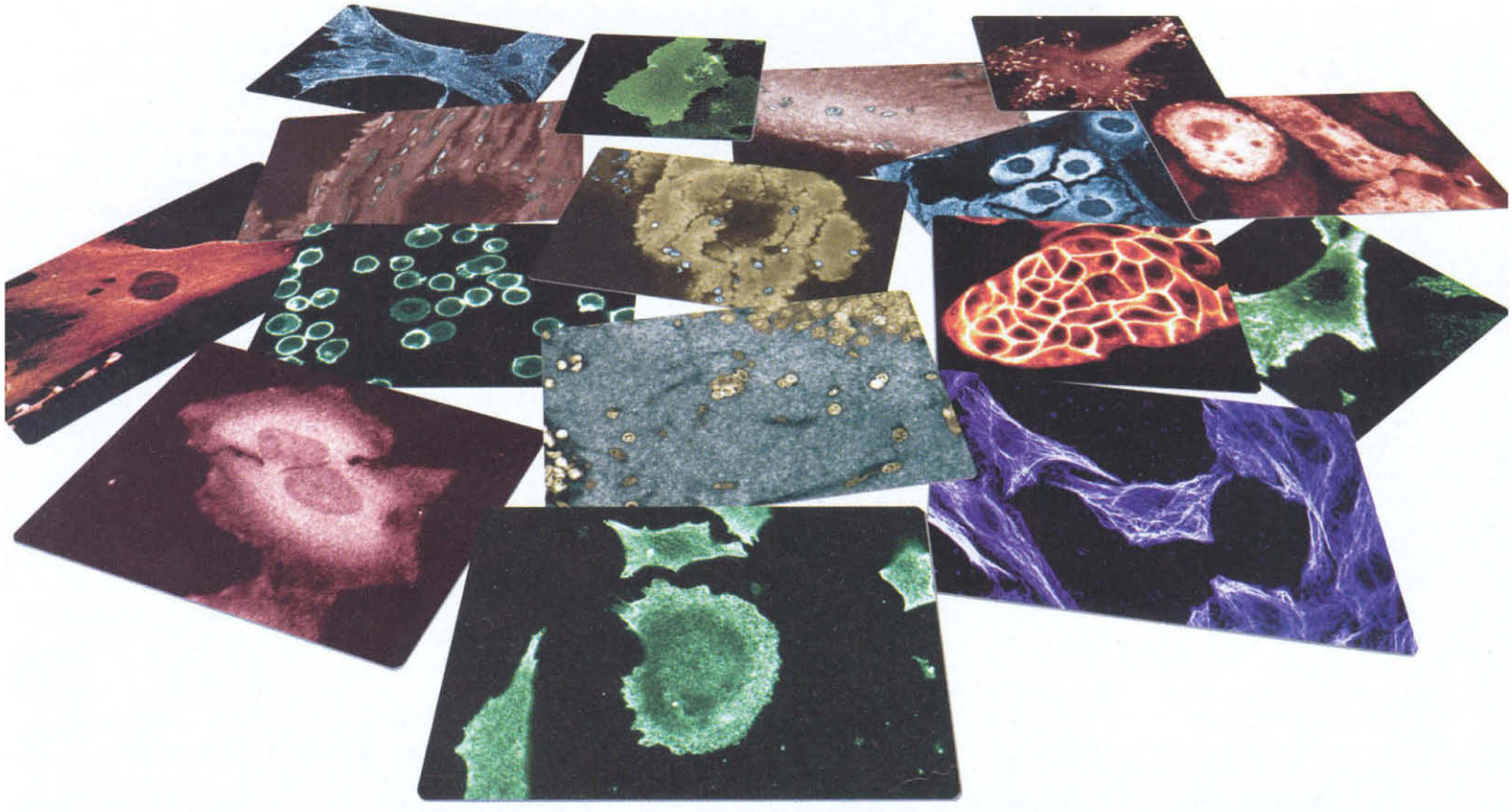

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The grand prize is US$20,000 with an additional seven runners-up winning US$5,000 and being announced in *Science*. The winning essay will be published in full. The award ceremony will be held in Sweden in early December. The Grand Prize winner will feature in next year's Amersham Pharmacia Biotech & *Science* Prize for Young Scientists advertisement. As an additional bonus, all winners and finalists receive a free subscription to *Science*.

Call for entries.

To be eligible, you must have received your Ph.D. between January 1 and December 31, 1997. Your thesis has to be in the field of molecular biology and submitted to us in the form of a 1,000-word essay which describes your work and places it in perspective with regard to the field of molecular biology. The essay can be written in English, French, German, Spanish, Japanese or Chinese (Mandarin).

The closing date is May 31, 1998. All prizes will be presented in Sweden in December 1998. Full details, and the required entry form can be collected from:

* the administrator of the award committee at the address below
* from the *Science* homepage at http://www.aaas.org/science/prize.htm
* from the Amersham Pharmacia Biotech homepage at http://www.apbiotech.com

amersham pharmacia biotech & SCIENCE PRIZE FOR YOUNG SCIENTIST in molecular biology 1998

Amersham Pharmacia Biotech and *Science* Young Scientist Prize Selection Committee

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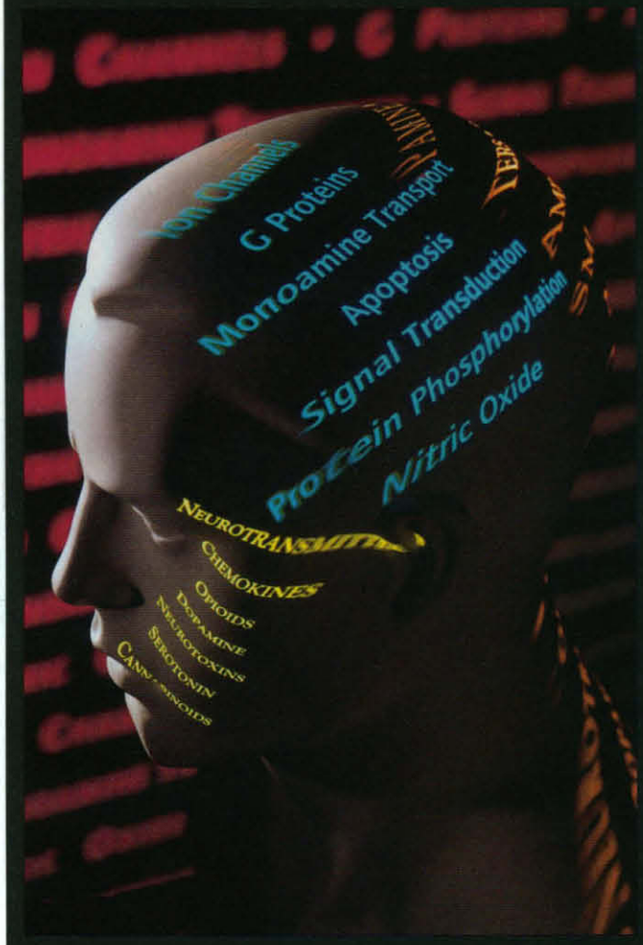

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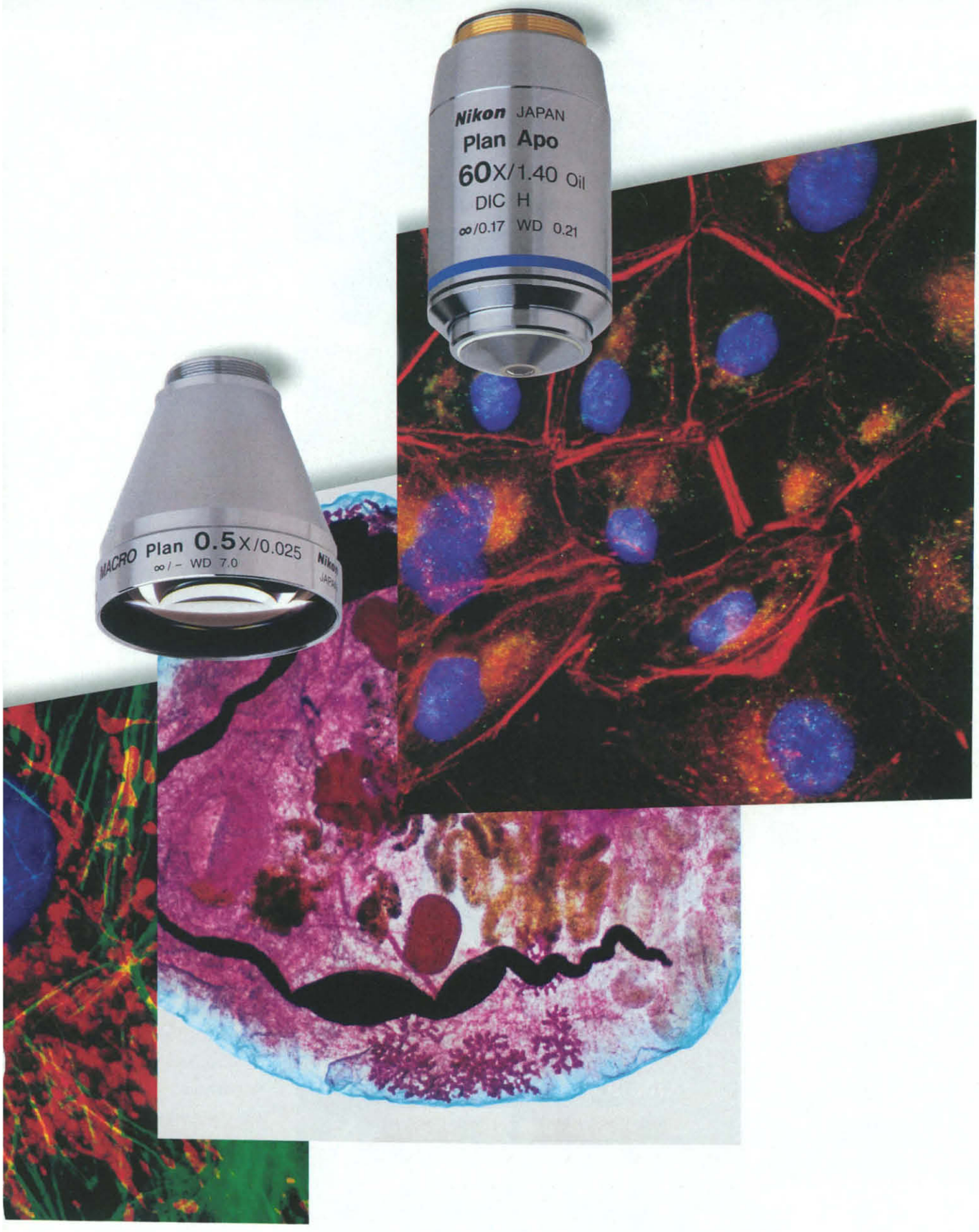
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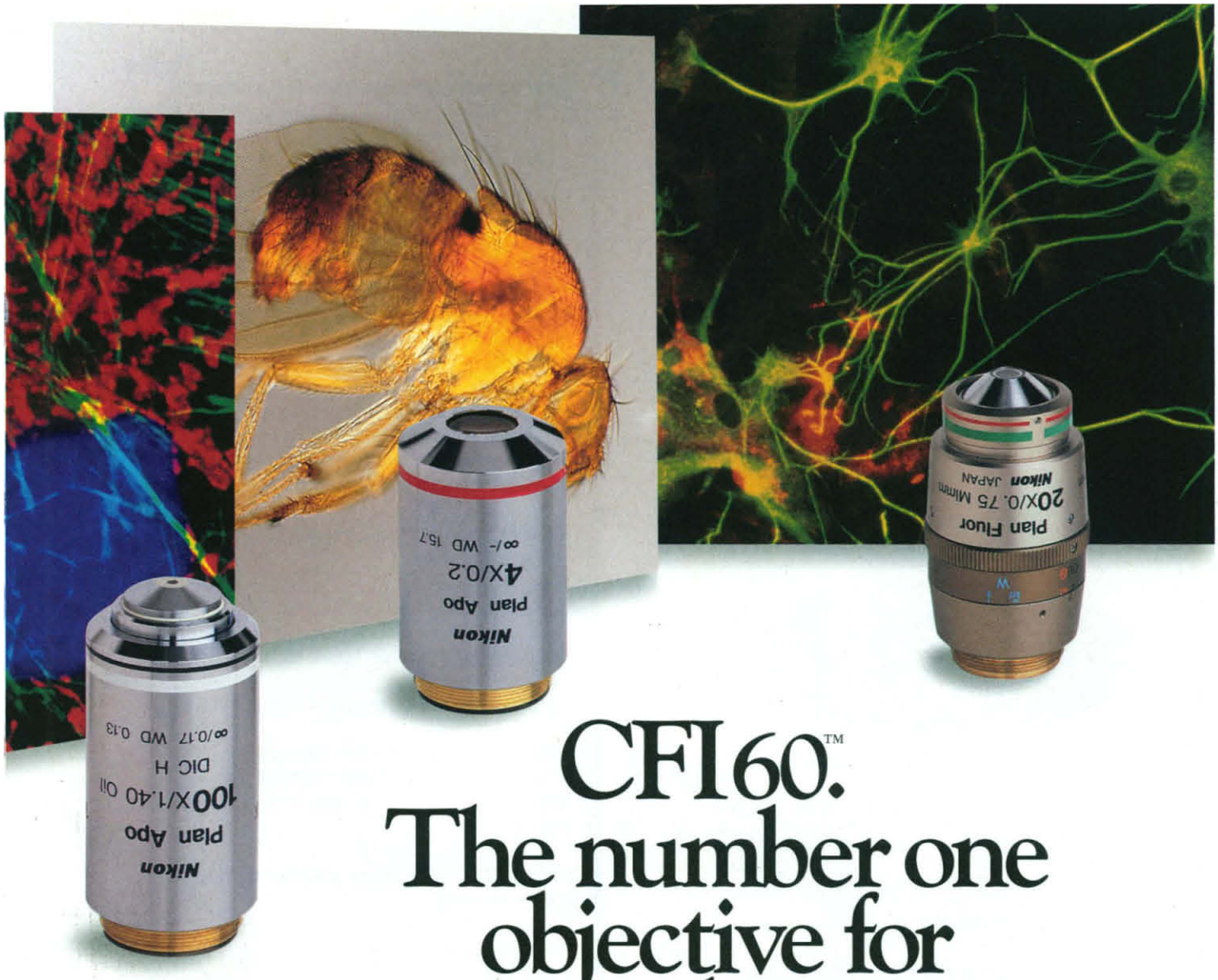

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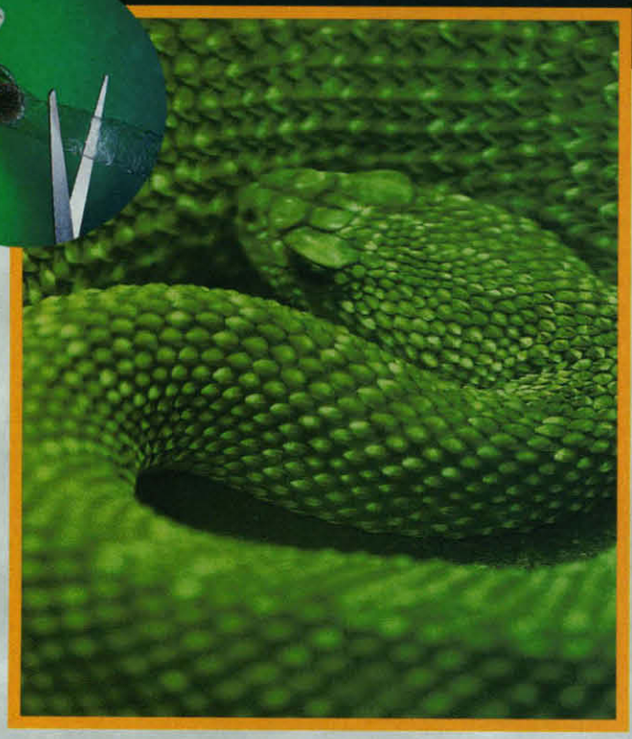

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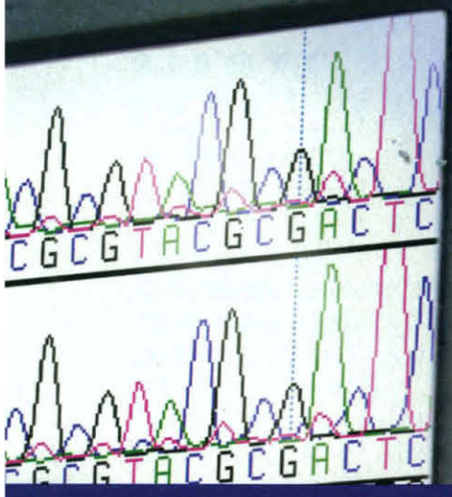

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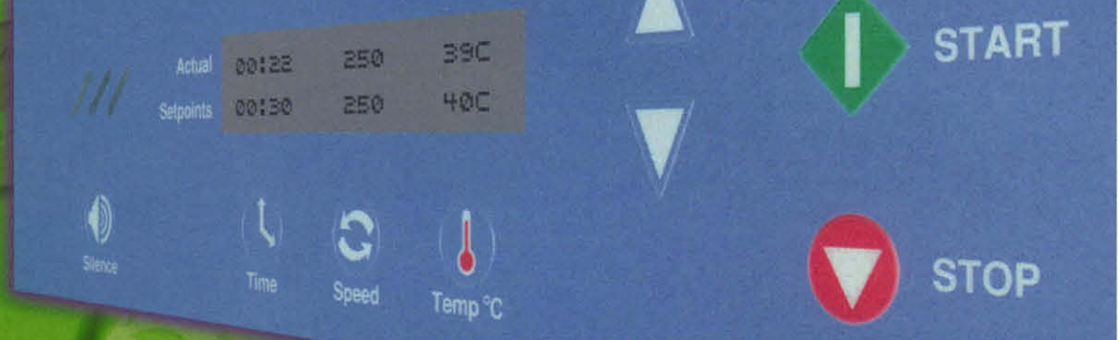

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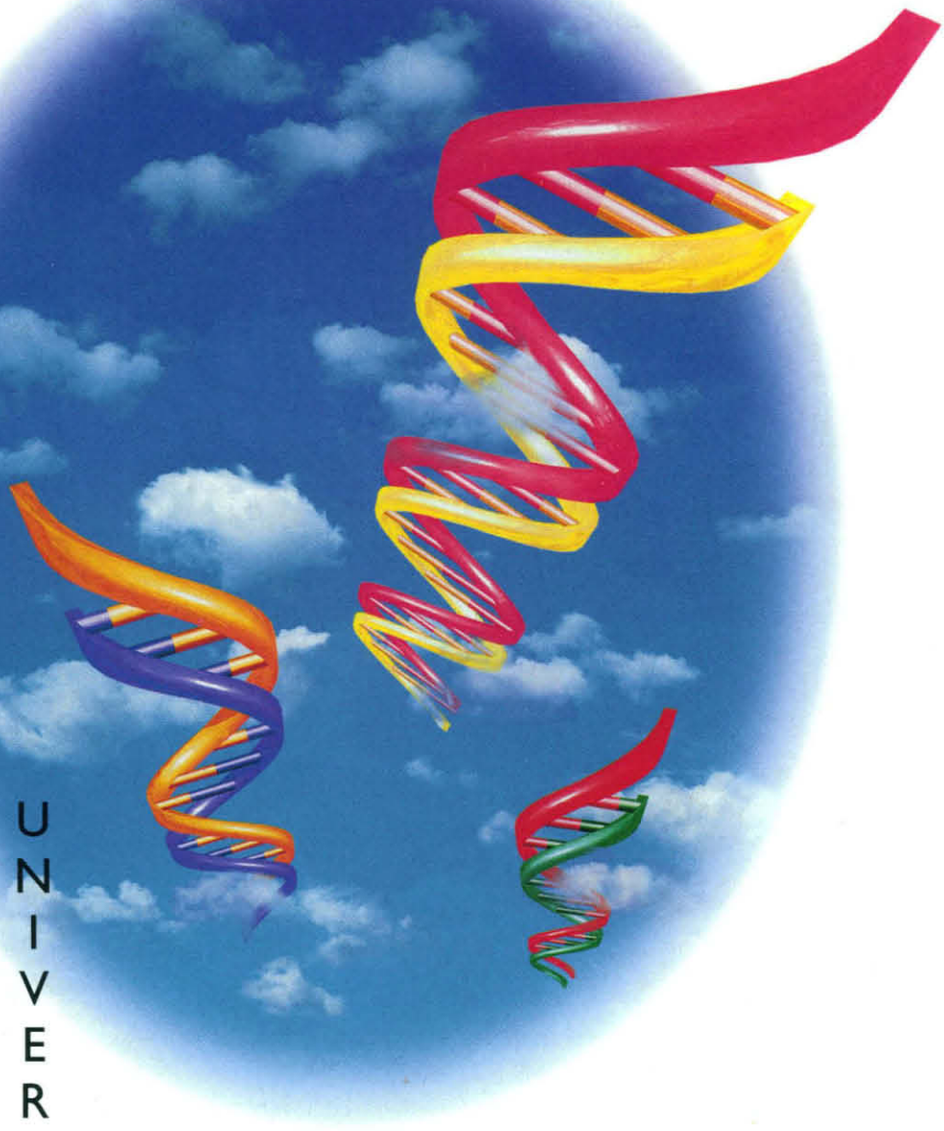

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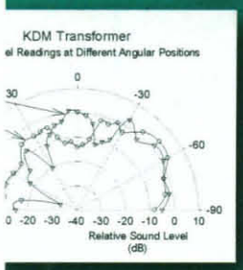

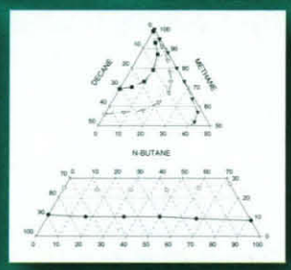

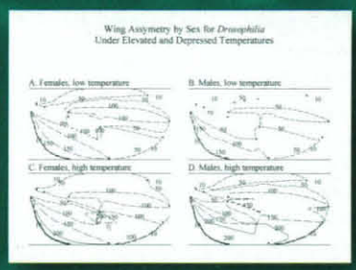

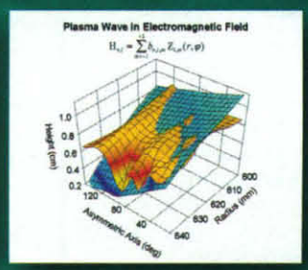

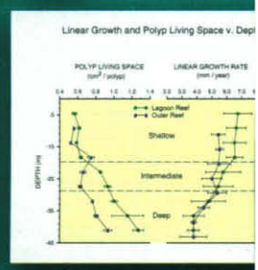

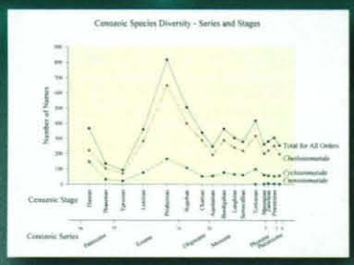

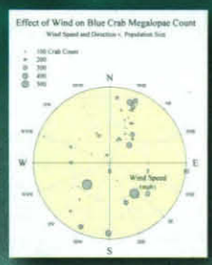

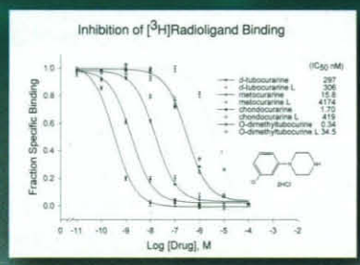

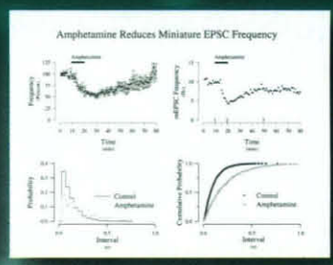

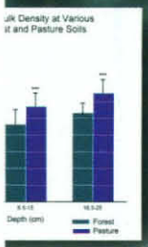

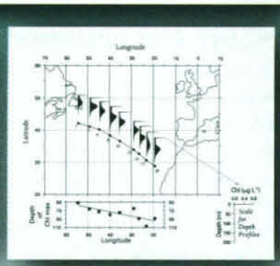

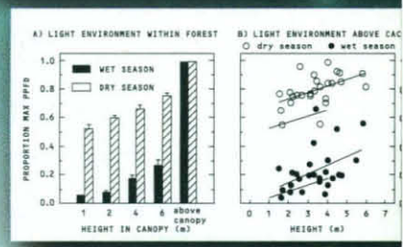

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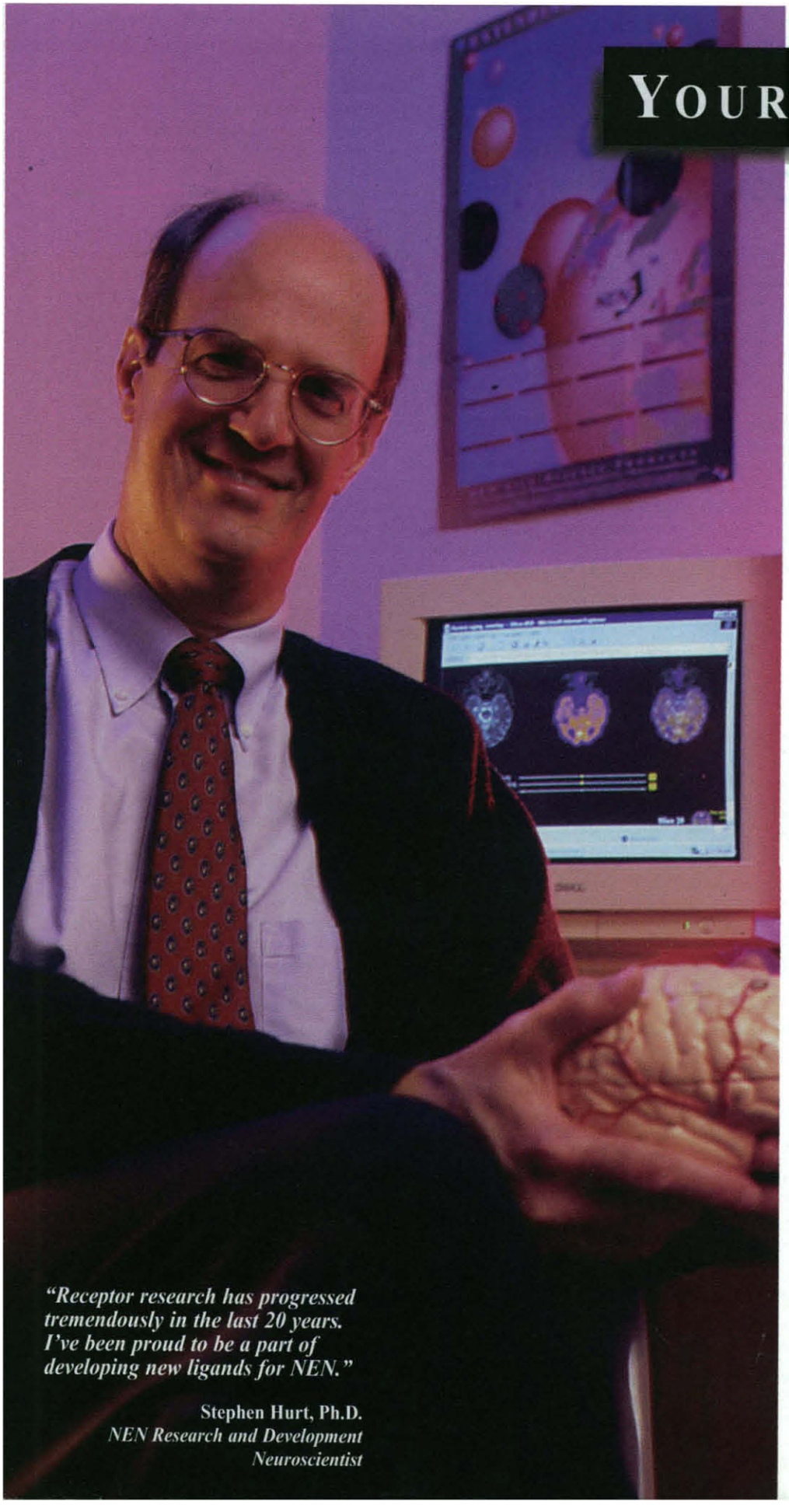

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Mail Nominations to:

McGovern Award Lecture, AAAS Meetings Office, 1200 New York Avenue, NW, Washington, DC 20005

Complete nominations, as described above, must be received by Monday, June 15, 1998.

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The deadline for receipt of abstracts is July 1, 1998. E-mail submissions are strongly encouraged. Please submit 6 copies of mailed abstracts. Direct abstracts or inquiries to: Deborah Runkle, AAAS, 1200 New York Ave NW, Washington, DC 20005. E-mail: safety@aaas.org; telephone: (202) 326-6794; fax: (202) 289-4950.