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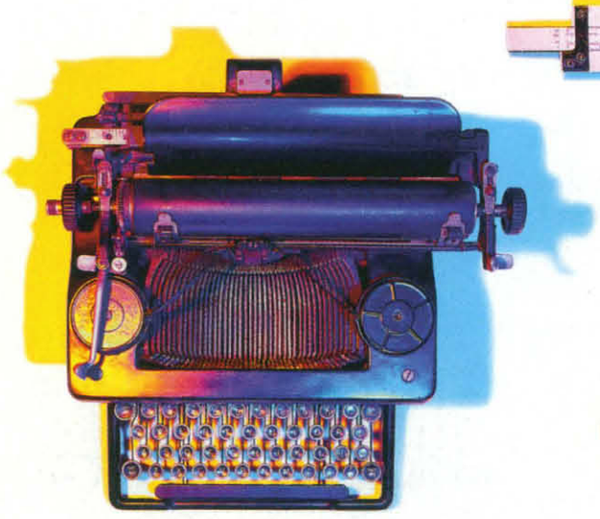

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COVER

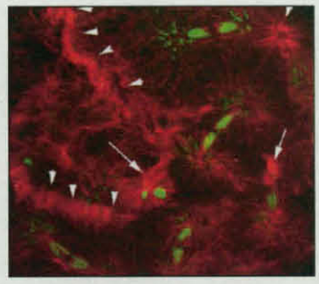
An immune system given free rein is lethal. The roadblocks built into the system or introduced by pathogens are the topic of several Articles starting on p. 237 and the Editorial on p. 179. Mutations of the immune system reveal developmental checkpoints and define mechanisms to turn lymphocytes off, such as the use of death receptors (red). Viruses (yellow) excel in putting up roadblocks to avoid detection, for example, by inhibiting major histocompatibility molecules (green) from moving to the cell surface. [Illustration: Katharine Sutliff]

ARTICLES

TURNING THE IMMUNE SYSTEM OFF

REPORTS

TECHNICAL COMMENTS



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Stalled motor

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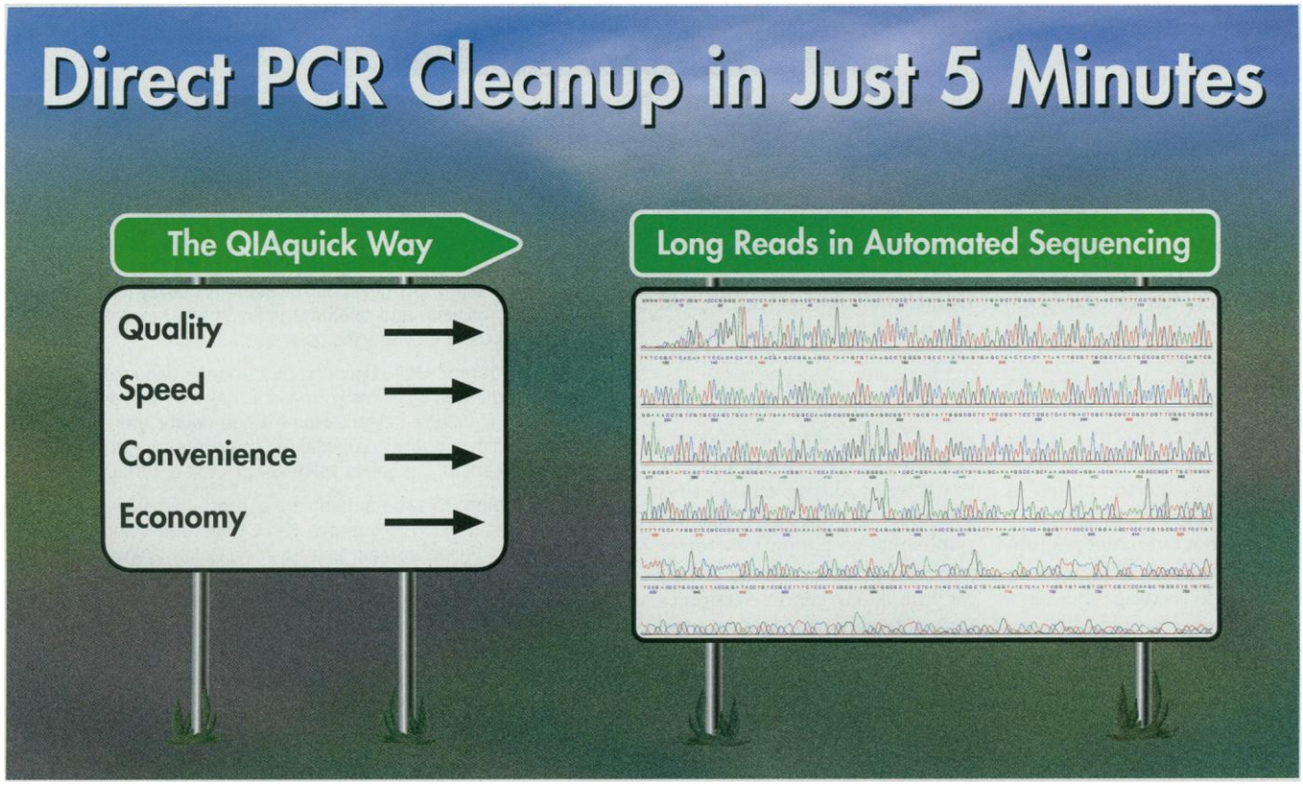

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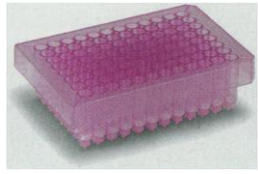

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edited by PHIL SZUROMI

Intradot interaction

Semiconductor quantum dots, such as islands of one semiconductor embedded in another, confine charge carriers in three dimensions. This confinement resembles that of electrons in atoms and results in a discrete energy spectrum. Landin *et al.* (p. 262; see the commentary by Gammon, p. 225) have studied optical emissions from single indium arsenide dots in gallium arsenide and show that the electrons and holes in the quantum dots interact with each other through Coulomb interactions to produce fine structure in the spectra. Such interactions between charge carriers in quantum dots have been predicted theoretically.

■

When a gel breaks

The failure of materials through cracking is a familiar phenomenon. Crystalline materials generally break instantaneously once a well-defined specific force is applied. Bonn *et al.* (p. 265) show that in polymer gels, delayed fracture can occur, with delays of up to 15 minutes for the materials and the applied forces they studied. They find that the behavior can be modeled when the connectivity of the gel network is taken into account, allowing calculation of the activation energy of crack formation.

■

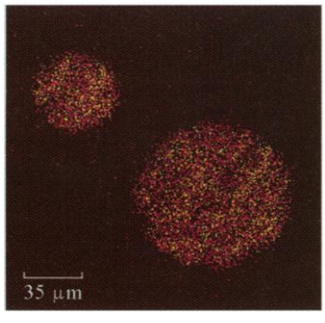
Hot on the trail of catalysts

Many chemical reactions are exothermic, and thus one way to screen catalysts is to measure the relative amount of heat liberated. Taylor and Morken (p. 267) show that thermal imaging can be used to screen active catalysts for reactions run in solution. They demonstrate the method for an acylation reaction; both known catalysts and those created from a library (approximately 3000 different potential catalysts) were supported on resin beads and imaged with an infrared camera (chloroform was used in the solvent so that the beads just float, thus reducing solvent interference with transmission). They show that the most active catalysts are the ones most strongly selected with this assay.

■

Soft support

Although materials such as silica have long been used as supports for catalysts, their inherent polarity can make them less than ideal for many highly reactive transition metal catalysts. Roscoe *et al.* (p. 270) report that noninteracting polymer supports can be used with metallocene polymerization catalysts. Reaction occurs inside catalyst-loaded polystyrene beads in the 50- to 100-micrometer size range to form polyolefin beads roughly 1 millimeter in diameter.



■

Change in momentum

In the BCS (Bardeen-Cooper-Schrieffer) theory of superconductivity, the formation of electron pairs lowers the energy of the system and open up a gap energy that is typically only a few millielectron volts (twice the thermal energy kT_c at the superconducting transition temperature T_c). Because typical energies of electrons near the top of the conduction band (Fermi energies) are a few electron volts, the mixing of electrons with similar energies is further constrained by a momentum requirement—an electron with momentum **k** mixes strongly only with electrons of momentum **k** or **−k** (moving parallel or antiparallel) because of the low gap energy. Shen *et al.* (p. 259) obtained angle-resolved photoemission spectra from optimally doped samples of the high-T_c superconductor $Bi_2Sr_2CaCu_2O_{8+\delta}$ and found changes in energy at certain momentum of up to 300 millielectron volts, or 40 times kT_c, which occurred with a substantial transfer in momentum [on the order of $(0.45\pi, 0)$]. This value is very near that required by the theory of Emery and Kivelson that connects microscopic antiphase domains or spin and charge ordering with superconductivity.

Being rubbed the wrong way

Atomic force microscopy (AFM) allows the determination of the forces between sample surface and tip when the tip is scanned across the sample. However, the molecular organization of the sample cannot yet be directly determined by this technique and requires the use of additional experimental techniques. Liley *et al.* (p. 273) have combined electron diffraction and Brewster angle microscopy with AFM to study the friction anisotropy of a lipid monolayer on a mica surface and show that a small molecular tilt results in a measurable friction anisotropy. This anisotropy is counterintuitive: The friction force is smaller when scanning against the "cat fur" direction than stroking with it.

■

Getting the message across

In messenger RNA translation, transfer RNAs (tRNAs) with attached amino acids dock into the P (peptidyl) and A (aminoacyl) sites of the ribosome for peptide bond formation. Previous work has identified the 23S ribosomal RNA (rRNA) as the ribosomal component that participates in peptidyl transferase function at the P site. Green *et al.* (p. 286) now show that a different domain of the 23S rRNA participates at the A site. Cross-linking analysis showed that in order for the A site to form, the P site has to be bound by tRNA, indicating a cooperative interaction between the P and A sites of the ribosome.

■

Biofilm formation

Certain bacteria can come together and differentiate to form a complex, multicellular structure called a biofilm. Davies *et al.* (p. 295; see the commentary by Kolter and Losick, p. 227) have shown that *Pseudomonas aeruginosa* uses a diffusible, density-dependent signal that is a product of *lasI* to induce differentiation of the biofilm. Biofilm formation by *P. aeruginosa* is a medical problem when it occurs in catheters or in the lungs of patients with cystic fibrosis. A mutation that disrupts the cell-to-cell signal made the biofilm sensitive to the detergent SDS. Agents that inhibit this signal could be helpful in preventing biofilms.

(Continued on page 175)

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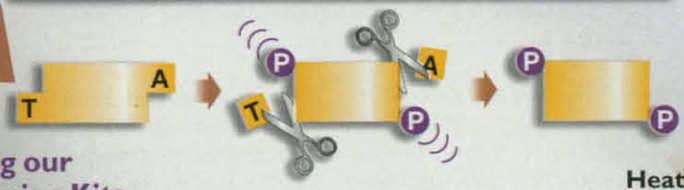

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References

1. Brownstein, J.M., *et al.* (1996) *BioTechniques* **20**, 1004–1010.
2. Magnuson, V.L., *et al.* (1996) *BioTechniques* **21**, 700–709.
3. Novy, R.E., Yaeger, K.W., and Kolb, K.M. (1996) *In**Nova**tions* **6**, 7–11.

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(Continued from page 173)

Stopping molecular motors

The molecular motor kinesin is responsible for many aspects of intracellular motility, including the movement of chromosomes along the mitotic spindle. The motor protein moves along intracellular tracks, the microtubules carrying its cargo through the cell. Studies of kinesin function have been hampered, however, because of the lack of specific agents to block its motor activity. So far only nucleotide analogs are known to act as potent inhibitors of motor function, but they lack specificity, which limits their usefulness in complex assay systems. Sakowicz *et al.* (p. 292) have now discovered a specific kinesin inhibitor in extracts from a marine sponge. The inhibitor appears to act by mimicking the microtubule and blocking motor-microtubule interactions.

■

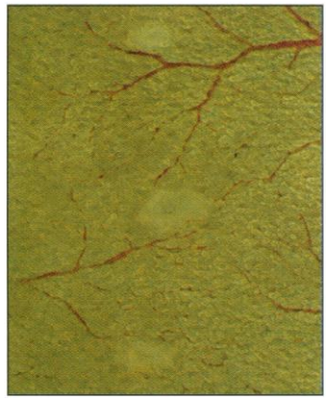
Kingdoms united in splicing

Splicing of transfer RNA precursors (pre-tRNAs) is essential for the production of mature tRNA. In Eucarya and Archaea, this process requires an endonuclease that recognizes the splice sites and releases the intron. Li *et al.* (p. 279) determined the crystal structure of the tRNA splicing endonuclease from the archaeabacterium *Methanococcus jannaschii*. Although the eucaryal and archaeal endonucleases are known to recognize their RNA substrates by very different means, the new structural data indicate that the two groups of enzymes share a common cleavage mechanism resembling that of ribonuclease A. Accompanying results from Fabbri *et al.* (p. 284), who examined endonuclease cleavage of artificial pre-tRNA substrates, support the evolutionary relatedness of the eucaryl and archaeal enzymes.

■

Locating intestinal T cells

The T cells of the intestines are thought to develop outside of the thymus, unlike other T cells. Saito *et al.* (p. 275; see the news story by Williams, p. 198)



have found a new primary lymphoid organ in the mouse small intestine, the recently identified "cryptopatches," and show that this is where the local precursors for intestinal T cells reside, not in the Peyer's patches or the mesenteric lymph nodes.

■

Stopping transcription

Increasing evidence has shown that the transcription apparatus is intimately linked with the messenger RNA (mRNA) processing machinery. Although mRNA polyadenylation signals are necessary for transcription termination, the molecular components of the mRNA cleavage–polyadenylation complexes involved in transcription termination remain unknown. By using temperature-sensitive mutants of polypeptides in the mRNA processing complexes, Birse *et al.* (p. 298) show that in yeast, polypeptides that function in endonucleolytic cleavage of nascent transcripts, but not polyadenylation, are required for efficient polymerase II termination.

■

Flight control

The halteres of dipterous flies evolved from hind wings and are believed to provide direct input to the forewing motor circuitry. Although the halteres do not function aerodynamically, their integrity and input is required for stable flight. In an integrated anatomical and physiological study, Chan *et al.* (p. 289; see the news story by Pennisi, p. 202) demonstrate that visual input directly influences motor control of the halteres and thereby indirectly alters the sensory output of the halteres and how that output regulates the flight musculature.

■

Hairpin reverse

The RAG proteins are the core enzymes responsible for the DNA rearrangements that comprise the making of an active antigen receptor gene. RAGs can cleave the DNA at recombination signal sequences to make blunt or "hairpin" ends. Melek *et al.* (p. 301) now show that the RAGs can also reverse the hairpin reaction with a mechanism reminiscent of the "dis-integration" reaction of retroviral integrases, to rejoin the DNA. This information explains how certain DNA byproducts of reaction are formed and may provide a basis for understanding the organization of the antigen receptor loci.

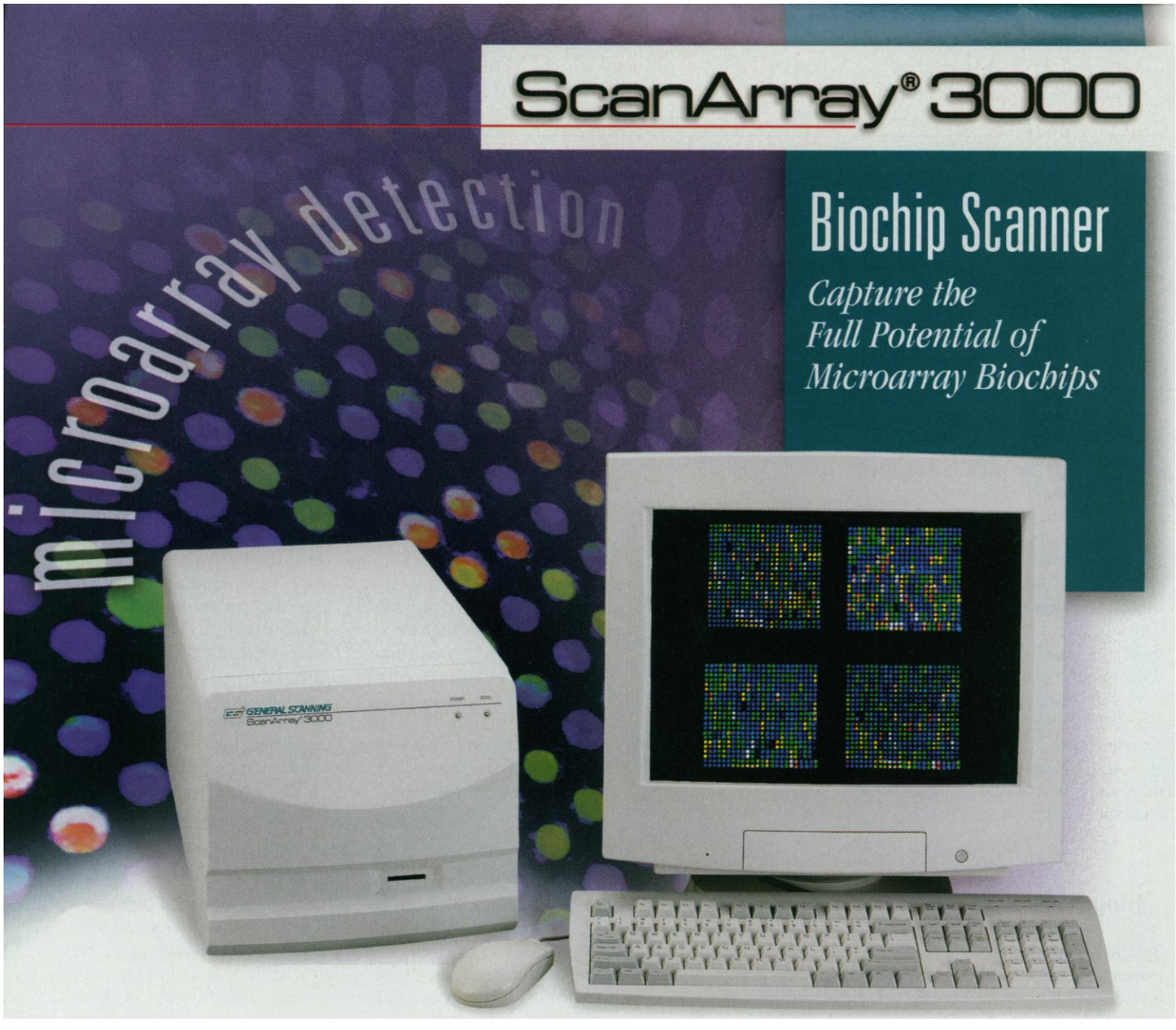
Technical Comment Summaries

Linkage Disequilibrium Mapping and Parkinson's Disease

M. H. Polymeropoulos *et al.* (Reports, 27 June 1997, p. 20) identified a mutation "in the α-synuclein gene, which codes for a presynaptic protein thought to be involved in neuronal plasticity, in an Italian kindred and in three unrelated families of Greek origin" with inherited Parkinson's disease. Other investigators, however, have not found evidence of this mutation in their Parkinson's disease study populations (Letters: The French Parkinson's Disease Genetics Study Group, 20 Feb. 1998, p. 1116; T. Lynch *et al.*, 14 Nov., p. 1212; and 5 Dec. 1997, p. 1696; Technical Comments: W. K. Scott *et al.*, 18 July 1997, p. 387 and T. Gasser *et al.*, p. 388).

B. Rannala and M. Slatkin show that "the recently developed theory of LD [linkage disequilibrium] mapping can be used to quantitatively assess" whether the α-synuclein mutation is indeed causative or if it is "a neutral variant in linkage disequilibrium with some other, causative, mutation." Rannala and Slatkin apply a method that "accounts for several demographic factors that may influence levels of disequilibrium, including population growth, genealogical associations, and sampling effects." They conclude from their analysis that, if the mutation identified by Polymeropoulos *et al.* is not the causative one, then it is probably very close to it on the genome.

The full text and figure of this comment can be seen at www.sciencemag.org/cgi/content/full/280/5361/175a



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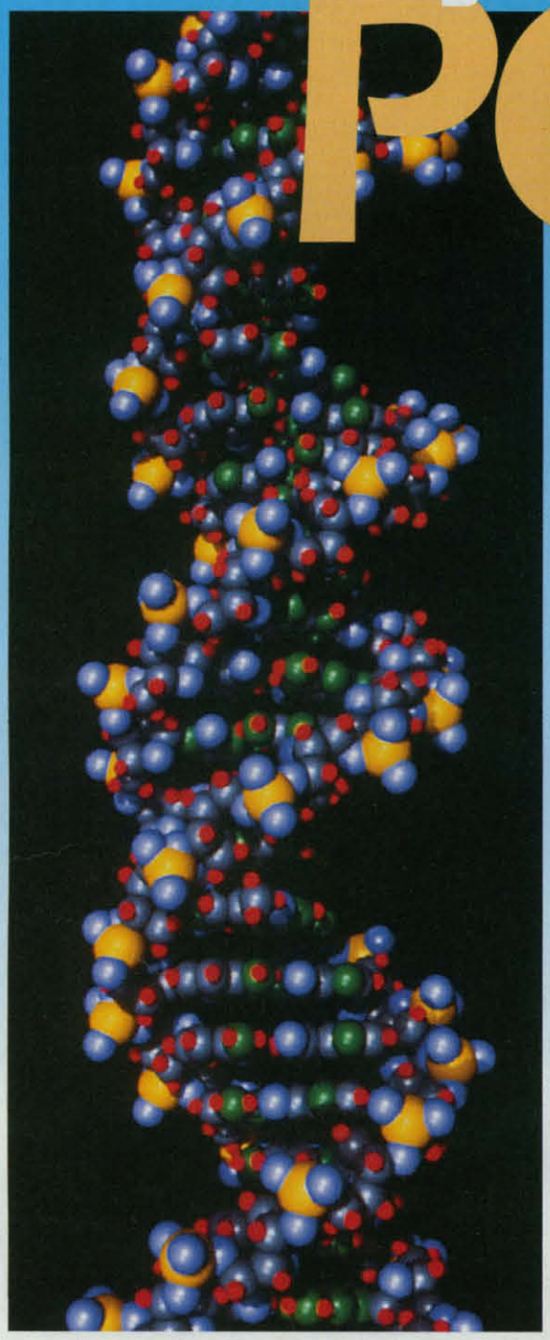

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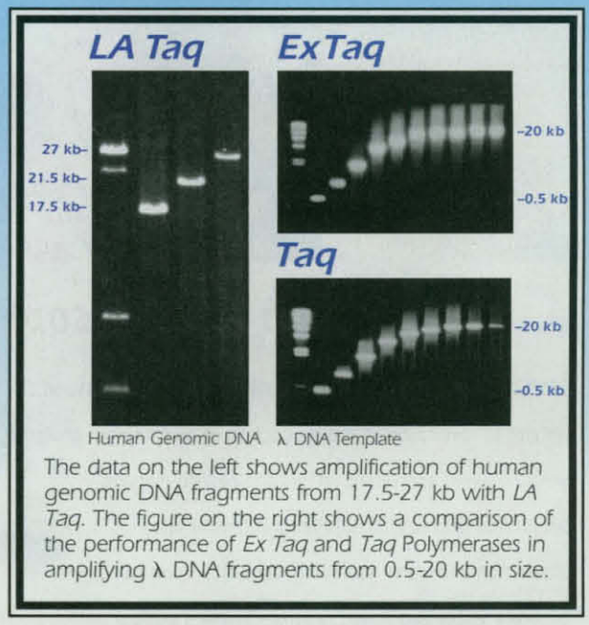

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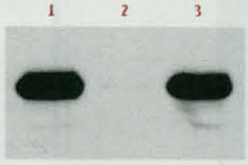

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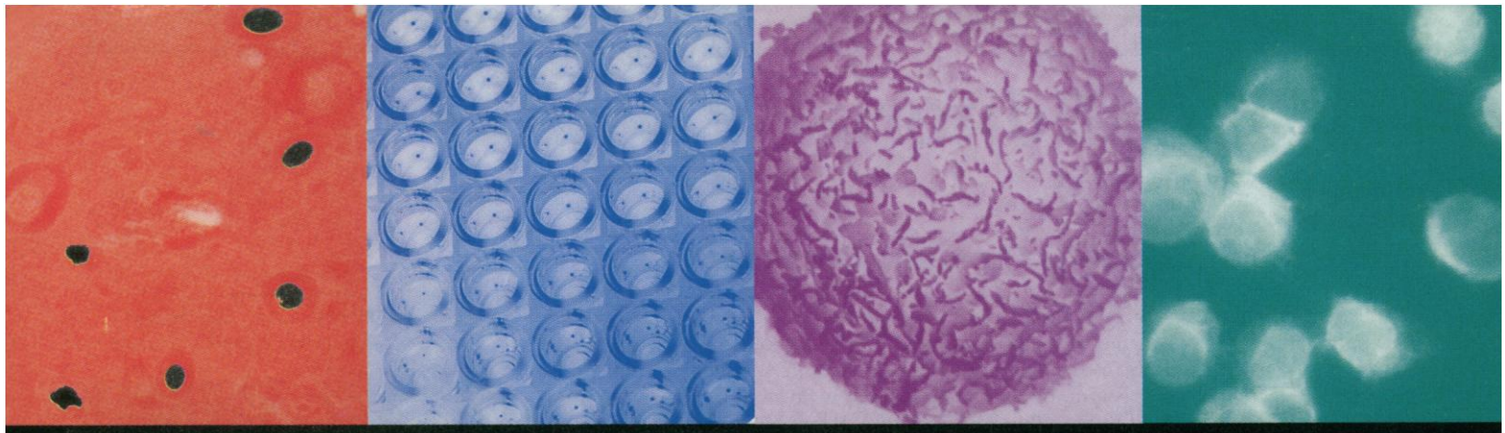

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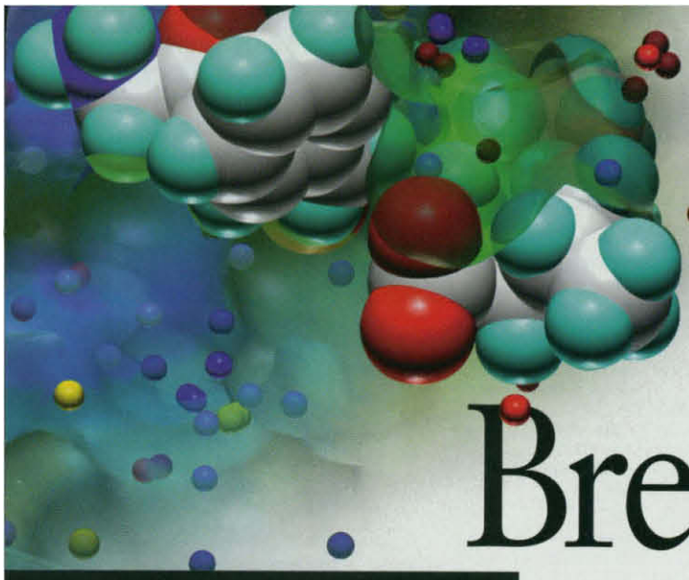

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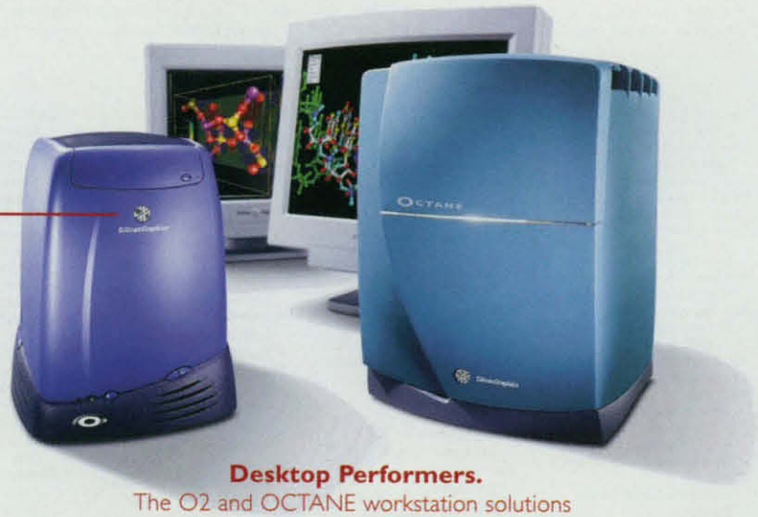

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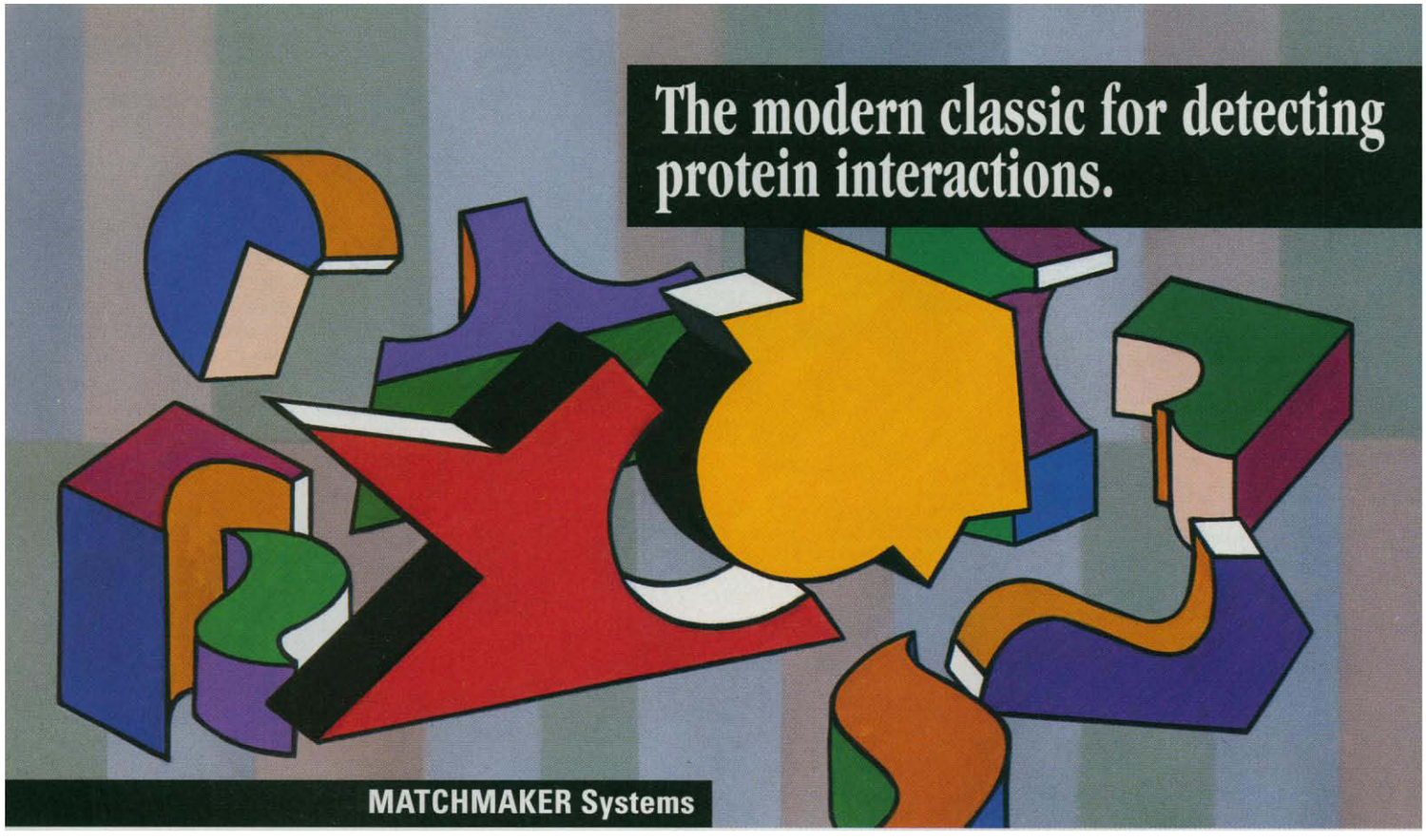

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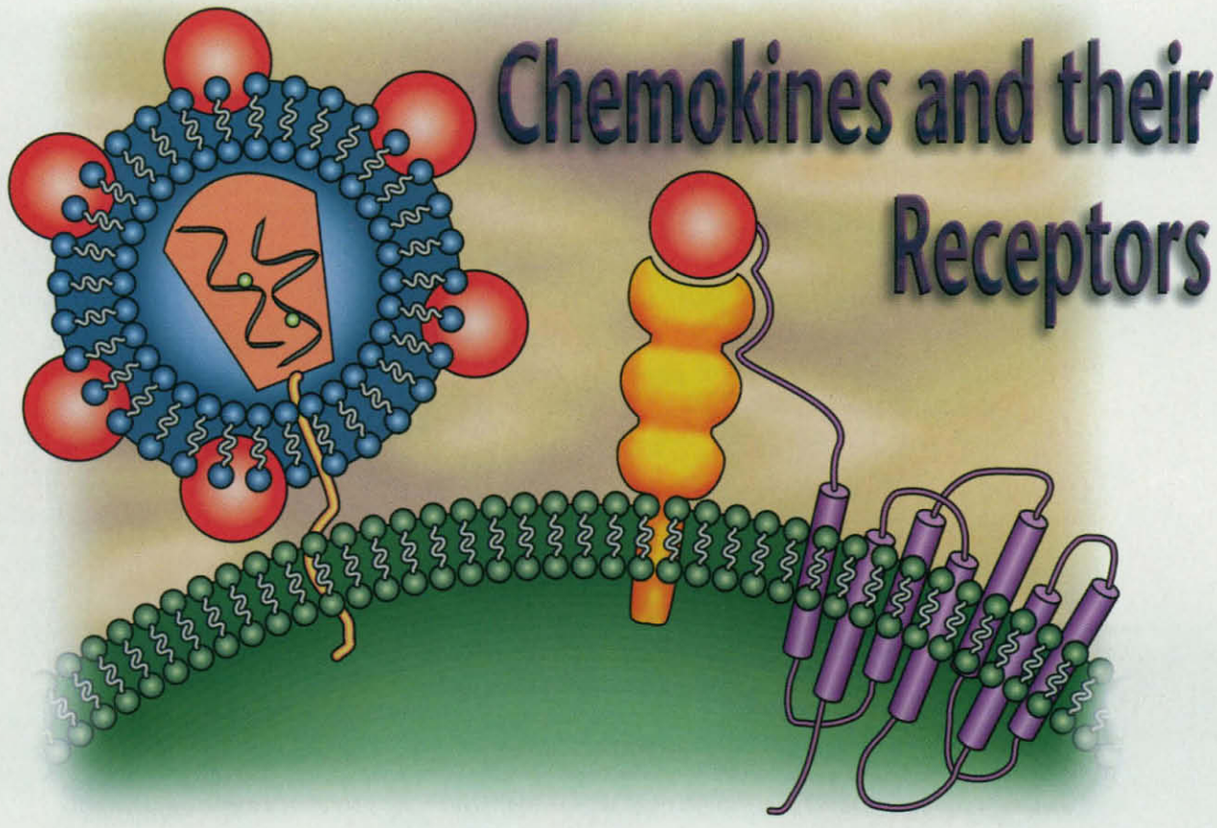

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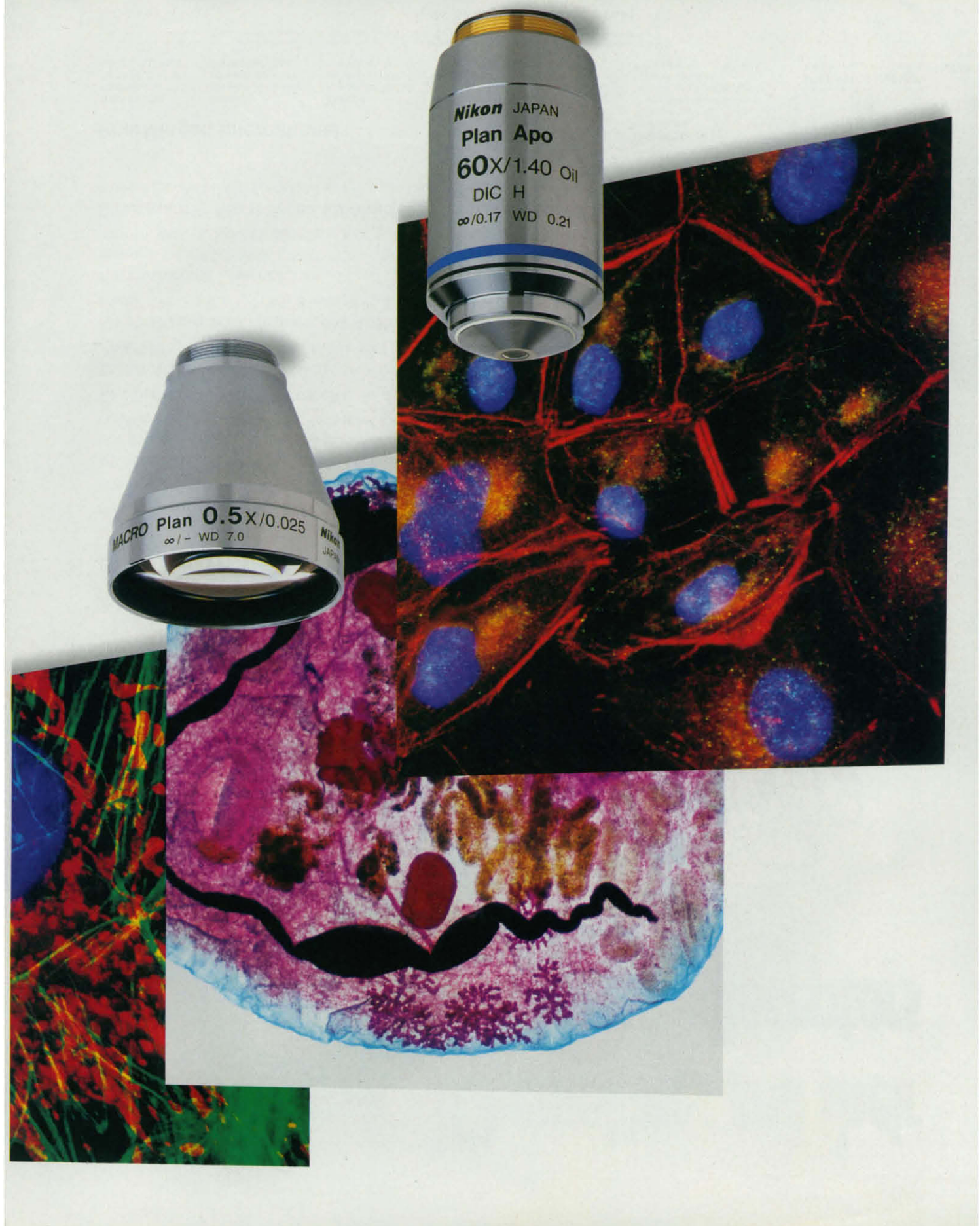
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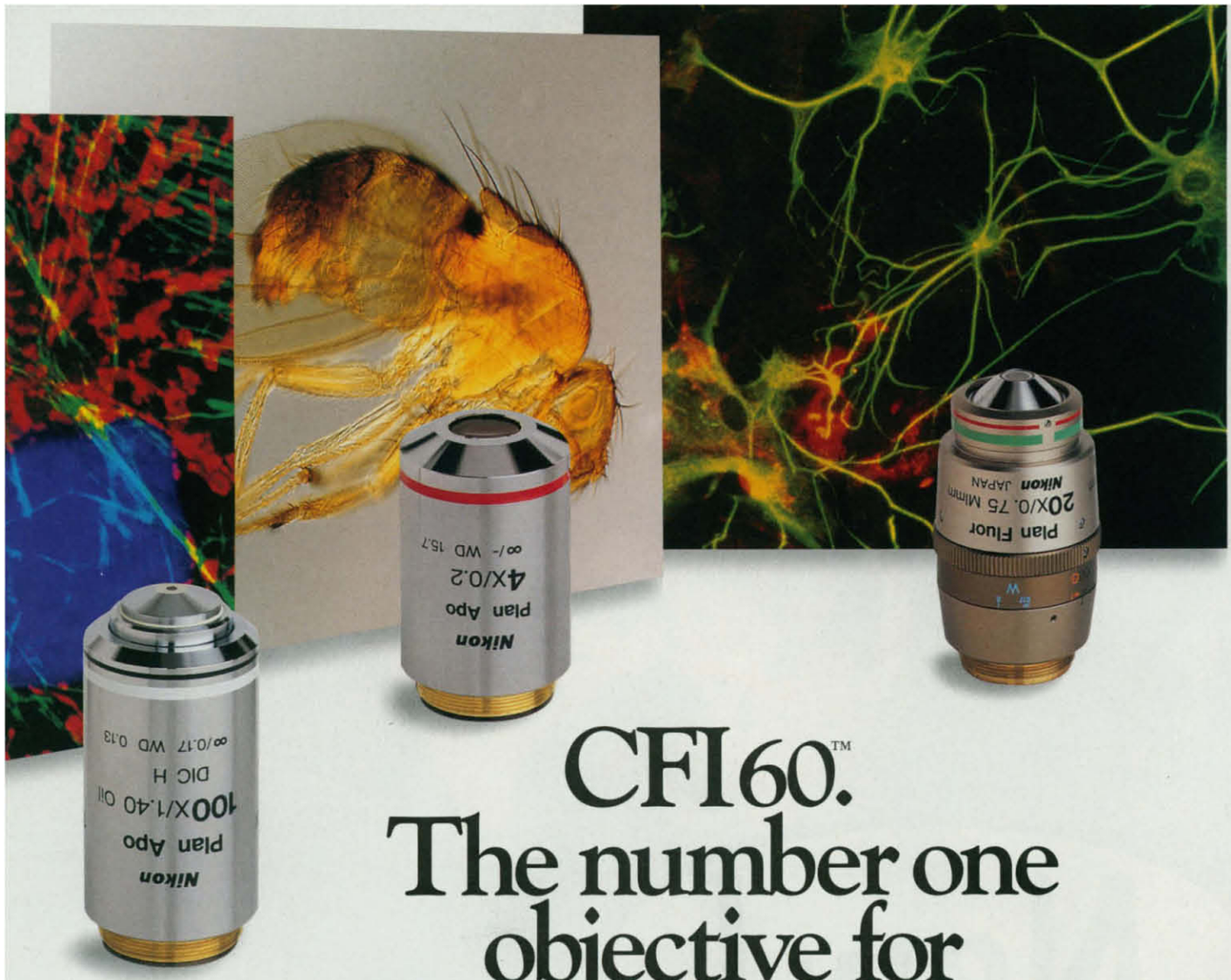

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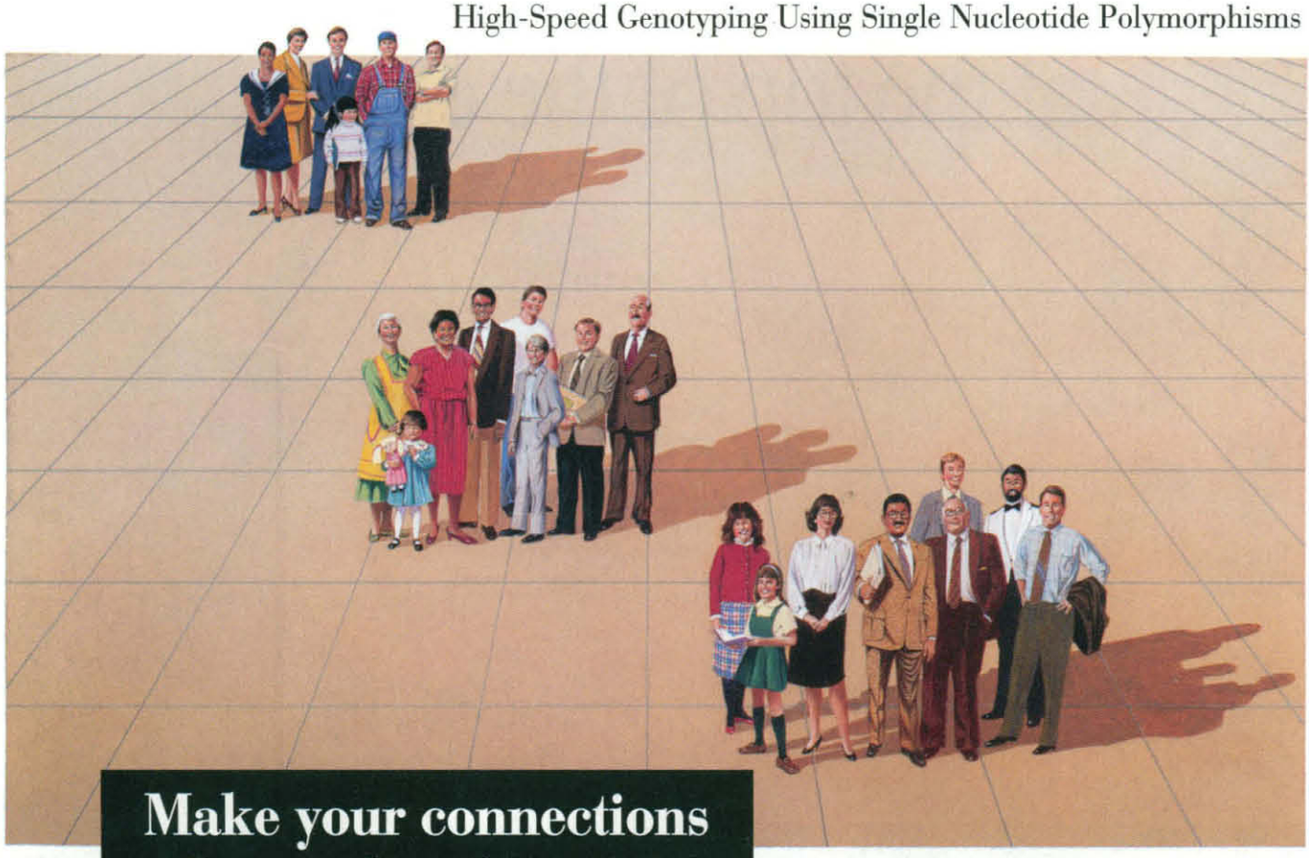

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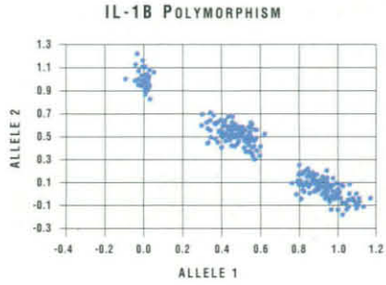

Genotyping of the single nucleotide IL-1B polymorphism: Genotyping of the two alleles of the human Interleukin-1 beta (IL-1B) promoter polymorphism at position −511 was performed using the fluorogenic 5′ nuclease assay. The graph shows genotyping results for 454 individuals. (Results courtesy of Franco di Giovine and Adeel Chaudhray, University of Sheffield, UK.)

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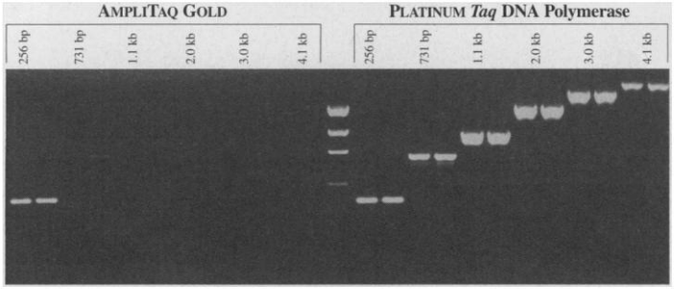

Automatic Hotstart setup at Room Temperature with 100 ng K562 Human DNA. Human betaglobin primers were used in 0.2 µM amounts. **PLATINUM *Taq* DNA Polymerase:** 1.5 mM $MgCl_2$, 0.2 mM dNTPs, 10X PCR buffer with PCR protocol–94°C, 1 min.; 94°C, 30 s; 55°C, 30 s; 72°C, 4 min 30 s. 35 Cycles of PCR performed. **AMPLITAQ GOLD™:** 2.5 mM $MgCl_2$ (as specified by PE), 0.2 mM dNTPs; 10X PCR buffer (PE); 2 units of AMPLITAQ GOLD. PCR protocol–PRE-INCUBATION 10 min @ 94°C prior to cycling; 94°C, 1 min; 94°C, 30 s; 55°C, 30 s; 72°C, 4 min 30 s; 35 Cycles of PCR performed.

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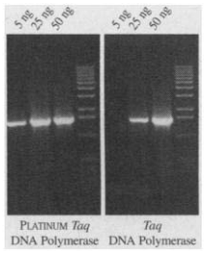

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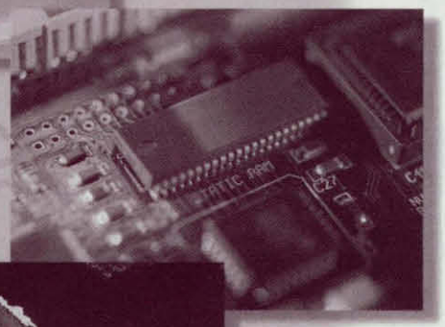

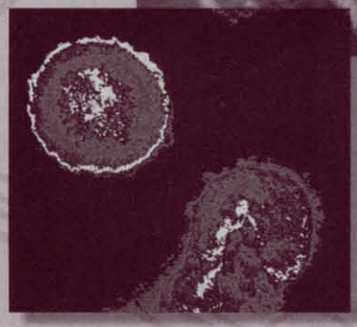

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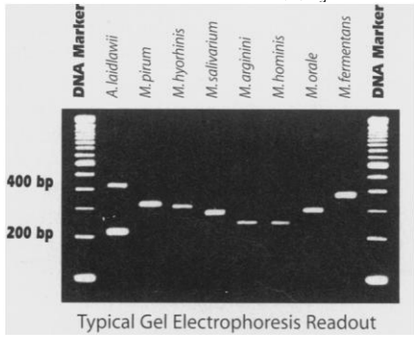

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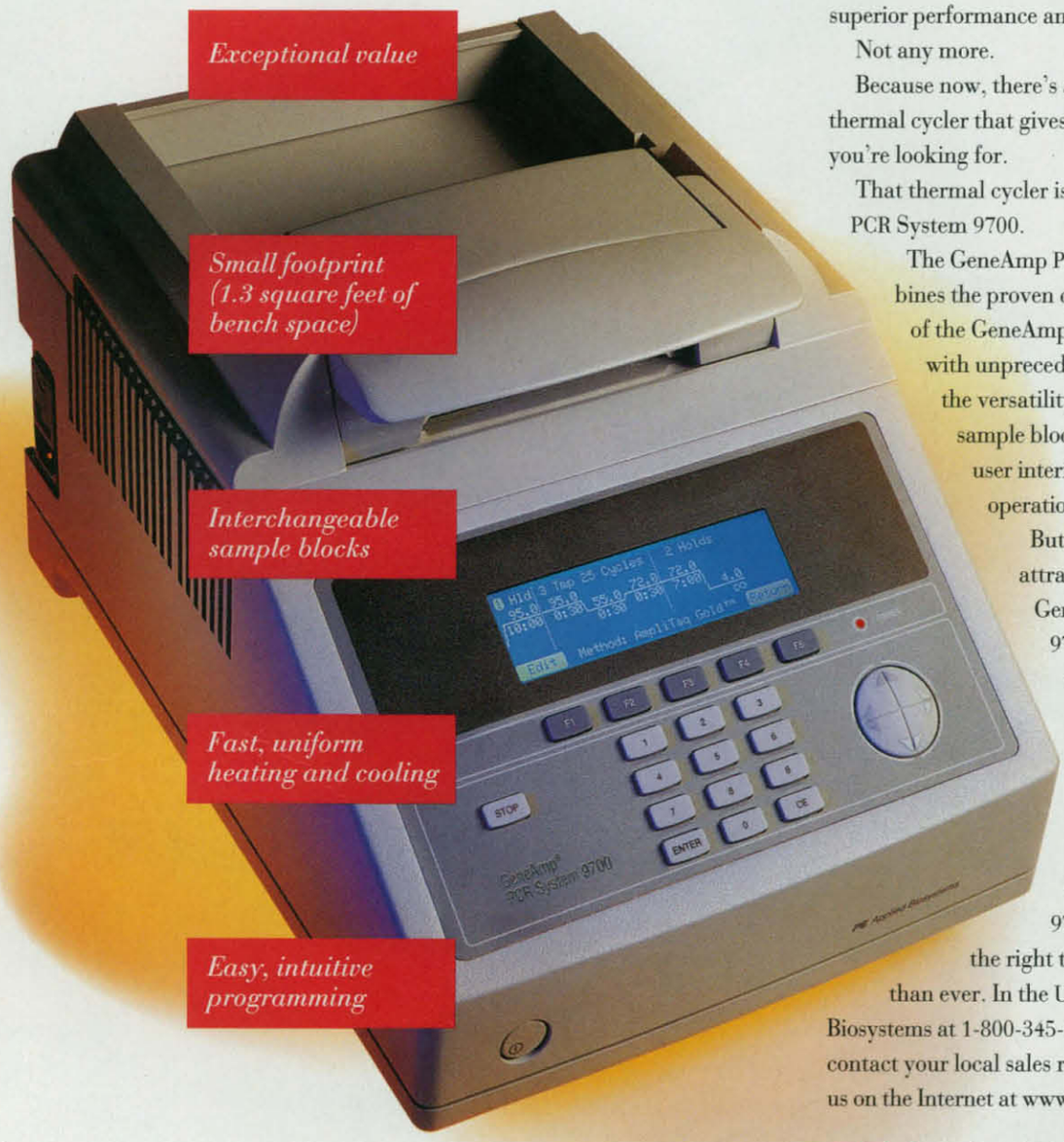

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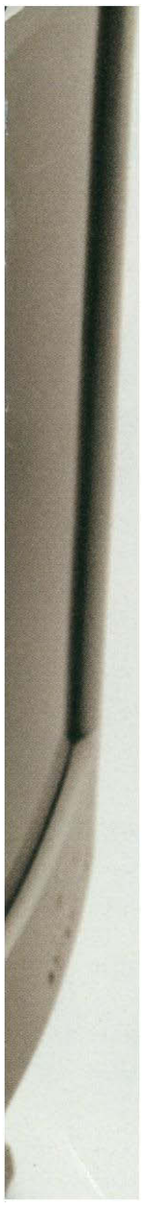

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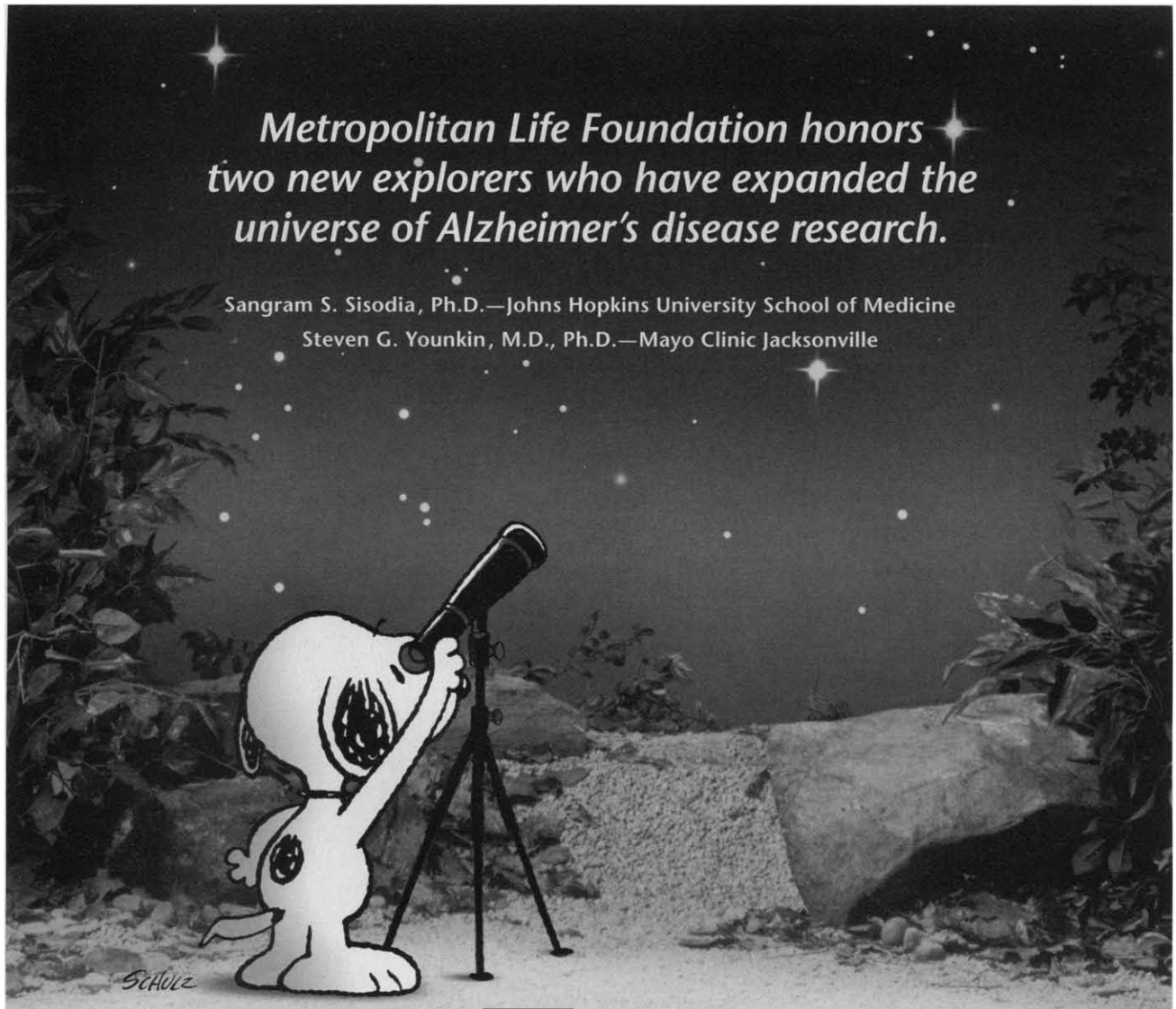
Sangram S. Sisodia, Ph.D.—Johns Hopkins University School of Medicine
Steven G. Younkin, M.D., Ph.D.—Mayo Clinic Jacksonville



About four million Americans now have Alzheimer's disease, and without the discovery of a cure or prevention, the number may reach 14 million by the middle of the next century.

The practical cost is enormous. The emotional cost is incalculable. The solution will be found only through the patience, perseverance and industry of the many men and women dedicated to a continual exploration and study of Alzheimer's disease.

Today, Metropolitan Life Foundation, through its Awards for Medical Research program, honors two of these explorers for their contributions to the understanding of Alzheimer's disease: Dr. Sangram S. Sisodia *(left)*, of Johns Hopkins University School of Medicine, and Dr. Steven G. Younkin *(right)* of the Mayo Clinic Jacksonville. They join a distinguished group of award winners who have increased our understanding of Alzheimer's disease.

Since the inception of the program in 1986, Metropolitan Life Foundation has awarded millions of dollars in grants for Alzheimer's disease research. Through these awards it is our hope to help researchers expand the universe of knowledge and move us closer to a cure for Alzheimer's disease.

Congratulations and thanks to Drs. Sisodia and Younkin.

PREVIOUS WINNERS:

1986 Peter Davies, Ph.D., Dennis J. Selkoe, M.D. 1987 James F. Gusella, Ph.D., Peter H. St. George-Hyslop, M.D. 1988 Carl W. Cotman, Ph.D., George G. Glenner, M.D. 1989 Donald Lowell Price, M.D. 1990 Konrad T. Beyreuther, Ph.D., Robert D. Terry, M.D. 1991 Stanley B. Prusiner, M.D. 1993 Blas Frangione, M.D., Ph.D., Allen D. Roses, M.D. 1994 John Hardy, Ph.D., Alison Goate, D.Phil., Robert W. Mahley, M.D., Ph.D., Karl H. Weisgraber, Ph.D. 1995 Gerard D. Schellenberg, Ph.D., Thomas D. Bird, M.D., Ellen M. Wijsman, Ph.D., Rudolph E. Tanzi, Ph.D. 1996 Brenda Milner, Sc.D., Michel Goedert, M.D., Ph.D., Yasuo Ihara, M.D., Virginia M.-Y. Lee, Ph.D., John Q. Trojanowski, M.D., Ph.D

Metropolitan Life Foundation

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HUMAN FRONTIER SCIENCE PROGRAM (HFSP)

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CALL FOR APPLICATIONS FOR 1999 AWARD YEAR
(Deadline for receipt of applications is 1 September 1998)

The aim of the Human Frontier Science Program (HFSP) is to promote, through international collaboration, basic research to elucidate the complex mechanisms of living organisms, including man. Applications are solicited for the support of research grants, fellowships and workshops in the areas set out below.

RESEARCH AREAS OF THE HFSP
Basic Research for the Elucidation of

(B) Brain Functions

1. Elementary Processes
2. Perception & Cognition
3. Movement & Behavior
4. Memory & Learning
5. Language & Thinking

(M) Biological Functions through Molecular Level Approaches

1. Expression of Genetic Information
2. Morphogenesis
3. Molecular Recognition & Responses
4. Energy Conversion

TYPES OF SUPPORT

The program will only support research that **transcends national boundaries**. Thus, **research grants** will be awarded for programs that involve collaboration between teams in different countries; **fellowships** are available to young post-doctoral scientists who wish to work in a different country; international **workshops** are organized in Strasbourg.

RESEARCH GRANTS Grants for basic research *(up to 3 years)* carried out jointly by research teams in different countries. The principal applicant must be from one of the eligible countries*.

FELLOWSHIPS **Long Term** *(1- 2 years)* and **Short-Term** *(up to 3 months)* Fellowships for researchers early in their careers and from the eligible countries* who wish to do post-doctoral research in foreign countries, or for young researchers from outside the eligible countries who wish to do research in one of the eligible countries*.

WORKSHOPS International workshops can be organized by researchers from the eligible countries*.

*** Current eligible countries are Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Italy, Japan, Luxembourg, the Netherlands, Portugal, the Republic of Ireland, Spain, Sweden, Switzerland, the United Kingdom and the United States.**

RESEARCH GRANTS AND LONG-TERM FELLOWSHIPS : APPLICATION DEADLINE IS 1 SEPTEMBER 1998

(awards to be announced in April 1999)

Applications for **Short-Term Fellowships** and **Workshops** can be submitted **throughout the year**

Scientists interested in organizing a workshop should contact the Secretariat about possible dates

Guidebooks and application forms will be available in mid-April 1998 and may be obtained upon written request by addressing the form below to the HFSP or by E-mail. Applications using previous year's forms are not accepted. You can retrieve copies of our guidebooks and application forms from our World Wide Web site at **http://www.hfsp.org**

Surname _____ *First name* _____

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☐ **Research Grant**
☐ **Long-Term Fellowship**
☐ **Short-Term Fellowship**
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(please check the relevant box(es)

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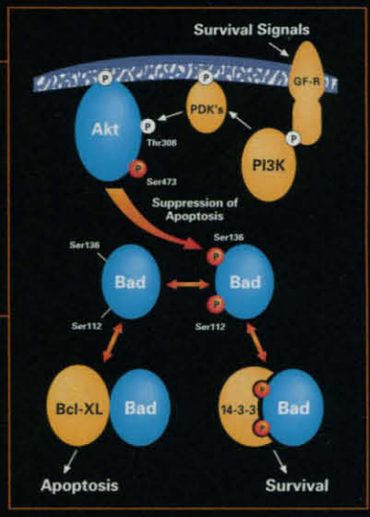
NEW ENGLAND BIOLABS

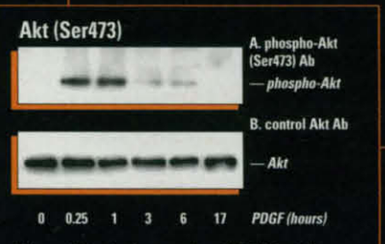
PHOSPHO-SPECIFIC ANTIBODIES

Cell Death/Survival Signaling

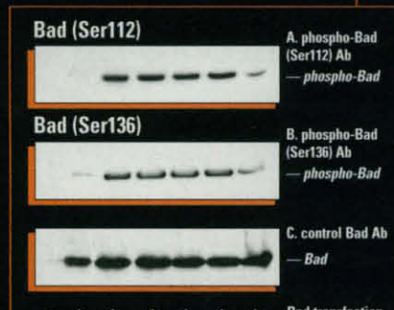
FOR ANALYSIS OF Akt & Bad

See NEB's 98/99 catalog to learn more about how **marine protected areas** are helping to conserve the ocean's rich biodiversity.





Western blots of cell extracts from PDGF (50 ng/ml) treated NIH 3T3 cells using (A) phospho-Akt or (B) control Akt Antibodies.



Western analysis of cell extracts from 293 cells transfected with GST-Bad and treated with TPA using (A) Phospho-Bad (Ser112) Antibody (B) Phospho-Bad (Ser136) Antibody and (C) Bad Antibody.

Cell Death/Survival related Phospho-specific Antibodies

Akt (S473)	#9270
Bad (S112, S136)	#9290
c-Myc (T58/S62)	#9401
Rb (S249/252, T373, S780, S795, S807/811)	#9300
IκB-α (S32)	#9240
p38 MAP Kinase (T180/Y182)	#9210
SAPK/JNK *monoclonal* (T183/Y185)	#9255
p44/p42 MAPK *monoclonal* (T202/Y204)	#9105

See the new NEB catalog and web site for a complete list of phospho-specific antibodies for Protein Kinases, Transcription Factors and Receptors.

Many in easy-to-use Kit format. Phospho-specific and control antibodies also available separately.

For more information visit the NEB web site: http://www.neb.com or call 1-800-NEB-LABS

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