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SCIENCE

17 APRIL 1987
VOL. 236 ■ PAGES 233-364

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■ **SCIENCE** is published weekly on Friday, except the last week in December, and with an extra issue in February by the American Association for the Advancement of Science, 1333 H Street, NW, Washington, DC 20005. Second-class postage (publication No. 484460) paid at Washington, DC, and at an additional entry. Now combined with **The Scientific Monthly**. Copyright © 1987 by the American Association for the Advancement of Science. The title **SCIENCE** is a registered trademark of the AAAS. Domestic individual membership and subscription (51 issues): \$65. Domestic institutional subscription (51 issues): \$98. Foreign postage extra: Canada \$32, other (surface mail) \$27, air-surface via Amsterdam \$65. First class, airmail, school-year, and student rates on request. Single copies \$2.50 (\$3 by mail); back issues \$4 (\$4.50 by mail); Biotechnology issue, \$5.50 (\$6 by mail); classroom rates on request; Guide to Biotechnology Products and Instruments \$16 (\$17 by mail). **Change of address**: allow 6 weeks, giving old and new addresses and seven-digit account number. Authorization to photocopy material for internal or personal use under circumstances not falling within the fair use provisions of the Copyright Act is granted by AAAS to libraries and other users registered with the Copyright Clearance Center (CCC) Transactional Reporting Service, provided that the base fee of \$1 per copy plus \$0.10 per page is paid directly to CCC, 21 Congress Street, Salem, Massachusetts 01970. The identification code for *Science* is 0036-8075/83 \$1 + .10. **Postmaster**: Send Form 3579 to *Science*, 1333 H Street, NW, Washington, DC 20005. *Science* is indexed in the *Reader's Guide to Periodical Literature* and in several specialized indexes.

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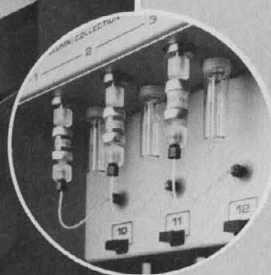
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This Week in SCIENCE

Risky business

NUMEROUS chemicals—both those that occur naturally and those that are man-made—have been tested at high doses for their carcinogenic effects on rats and mice; about half have proved to be carcinogenic (page 271). The validity of extrapolating from rodent experiments to humans in order to predict risks to human health from exposure for long periods to low doses of the same chemicals has been challenged. Ames *et al.* further argue that compared to the ubiquitous naturally occurring background carcinogens present in the environment (in foods, in soil, and elsewhere), the amounts of pesticide residues and of man-made pollutants in water are small and that the seriousness of the risks from any of the low-level chemicals, man-made or natural, may be greatly overstated. Risk assessment is discussed in several other articles and in Koshland's editorial (pages 267 to 300 and page 241).

The spider and the fly

JUMPING spiders prey on fruit flies, but, by mimicry, some vulnerable flies stay one jump ahead of their predators (pages 308 and 310). These flies have broad bands on their wings that look like spiders' legs; when a fly flaps its wings, the gait and display behavior (used in social interactions) of spiders are mimicked. Spiders typically stalk prey that come into their visual field; however, when a displaying spider enters the field, the first spider responds by displaying back. Greene *et al.* and Mather and Roitberg explored the abilities of flies and jumping spiders to deceive and be deceived. Mimicry was restricted: when a hungry spider encountered a fly in the experimental arena, spiders were most consistently put off by fruit flies that had the banded wing pattern (just as they were put off by other jumping spiders). House flies bearing, but not flapping, the banded wings of fruit flies did not deceive the spiders. Fruit flies that displayed plain wings (either transplanted from house

flies or with obliterated band patterns) became ready prey. Specific mimicry may be a phenomenon practiced by many flies whose predators, like the jumping spider, have retinal "feature detectors" that make them vulnerable to mimicry.

Chriacus: out on a limb

AN almost complete skeleton of the Eocene mammal *Chriacus*, which lived about 50 million years ago, has been recovered intact from the Willwood Formation of the Bighorn Basin of Wyoming (page 314). *Chriacus* was once thought to be ancestral to the Artiodactyla, a large and important order of mammals that includes even-toed ungulates such as camels, sheep, giraffes, hippos, and deer; the association was based on similarities in dental specimens. However, this association no longer appears credible: the lesson from the skeleton is that *Chriacus* was an animal much more like tree-dwelling civets, coatis, and lesser pandas. Rose describes the many adaptations of the skeleton for climbing in trees and on the ground for digging: mobile joints, flexible clawed feet, powerful limb muscles, and a long tail good for balancing and perhaps clinging. This discovery leaves the ancestry of the Artiodactyla still a mystery.

Chromosome deletion in acoustic tumors

BILATERAL acoustic neurofibromatosis (BANF) is a serious neurologic disorder that can lead to deafness, paralysis, other neurologic problems, and death (page 317). Several types of tumors can develop in the central and the peripheral nervous systems of people with BANF, but the most common finding is bilateral tumor development in the acoustic or eighth cranial nerves. BANF is inherited. Seizinger *et al.* studied the chromosomes that were present in tumors of patients with BANF. The unifying observation was that deletions occurred in chromosome 22, thus associating this chromo-

some with the pathogenesis of the several tumor types. Deletions of chromosome 22 have previously been implicated in "sporadic" BANF-type tumors, those typically unilateral tumors that develop in individuals not known to have the disorder in their families. As more probes are developed for chromosome 22, it should be possible to define precisely the portion of the chromosome that is involved in the development of disease. This could lead to new diagnostic and treatment strategies for both familial and sporadic cases of BANF.

Auxiliary heart pump

ONE of the skeletal muscles, the broad flat latissimus dorsi muscle of the back, can be shaped into a chamber that, upon electric stimulation, pumps blood much as does the heart (page 324). Experiments performed in beagles show that the muscle pumps (called skeletal muscle ventricles or SMVs) could sustain pressures comparable to those achieved by a healthy heart. Acker *et al.* fashioned SMVs on chest walls of dogs but waited several weeks before activating the muscle pumps with implanted pacemakers. During the rest period, a rich new blood supply to the muscle was established (a significant number of the original blood vessels, called collaterals, had been severed when the SMVs were being formed). The SMVs functioned without significant fatigue for up to 2 months. They did not need to be conditioned first with electrical stimulation as had been the case in previous experiments. Although there are physiologic dissimilarities in muscles of humans and dogs (for example, the dogs' muscles have an unusually oxidative character), it is possible that comparable muscle pumps could be constructed in people who are suffering from chronic heart failure (caused by past heart attacks or diseases of heart muscle) and in children who have congenital defects that result in underdeveloped heart chambers. An SMV could assist a failing heart by pumping a few extra liters of blood per minute.

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Immortality and Risk Assessment

The time has come, it seems, when I can realistically refocus on the goal of achieving immortality. I do not refer to the old-fashioned sort of immortality earned by performing monumental works in science or art, winning historic battles, procreating children, or adhering to a simple faith in the Almighty. I am talking about living forever.

From recent reports on television and in newspapers, my impression is that we are dying like flies from exposures to toxic chemicals, nuclear power stations, drunken drivers, and incompetent physicians. If one simply avoids such hazards and has a little help from an artificial organ here and there, dying seems to be pointless. All that needs to be done is to reduce life to zero risk. To provide guidance for those who would like to be immortal, this issue of *Science* generously shares with our readers some risk assessment analyses by the experts.

In the article by Richard Wilson and E. A. C. Crouch, the comparative listing of various risks makes it evident that I will have to give up being a policeman with a 2×10^{-4} annual risk of death (AR), driving motor vehicles (2×10^{-4} AR), and being a "frequent flying" professor (5×10^{-5} AR). I was, to say the least, stunned to find that by switching from city water (6×10^{-7} AR) to what the Environmental Protection Agency considers contaminated well water in Silicon Valley, I could actually lower my risk by a factor of 300. It was also distinctly unnerving to discover that the potassium in my body, which contains a radioactive isotope, gave me 1500 times the radiation level of that from the atmosphere within 20 miles of a nuclear plant, and 6 times that from a transcontinental air trip. Should we, I wondered, abandon Superfund and find a substitute for potassium in the body? Lester Lave informs us that hazards around the house are half as likely to cause injuries as motor vehicle accidents and that asbestos poses a small risk in most appropriately constructed buildings. Astonishingly, corn contains aflatoxin at appreciable levels as does peanut butter and, for me, giving up those two delicacies is not going to be an easy trade-off for mere immortality. Bruce Ames' article is a further shocker, ranking chemicals in terms of potency rather than simply labeling them as carcinogenic and noncarcinogenic. And, lo and behold, food ranks as a major hazard. Apparently plants learned through evolutionary time that chemical warfare is an extremely effective way to fight off fungi, insects, and animal predators. Unfortunately, these species have the same type of genetic code as I do, so that whenever I eat, I am consuming mutagens and carcinogens rated everywhere as hazardous to my health.

Clearly, to get to zero risk I must give up walking up and down stairs, drinking alcohol, living in Denver or other high-altitude locations, and innumerable other temptations. I am willing to sit in a rocking chair with a lead roof over my head and be fed amino acids intravenously in order to live forever.

Still, Paul Slovic points out that a scientist does not necessarily see risk the same way that the public does. The public regards deaths caused by mysterious technology or the simultaneous deaths of a large number of people (in airplane crashes, for example) as being far worse than those from well-known causes or the same number of deaths occurring in multiple locations (as in automobile accidents). Therefore, as I sit in my rocking chair, I become uneasy that the Nuclear Regulatory Commission, as described by David Okrent, may not really be doing its job, and that the regulatory agencies described by Milton Russell and Michael Gruber are making decisions based on politics rather than quantitative scientific appraisal. The government, I note, seems determined to remove the beautiful red color from maraschino cherries while supporting subsidies to tobacco growers and allowing cigarette advertisements, although cigarette smoking may cause as many as 350,000 premature deaths a year in the United States alone. Excessive worry about the competence of others can cause peptic ulcers and lead to my death from "natural causes."

Thus, although my commitment to the goal of immortality is unswerving, I am not positive that a zero risk society is yet in the scientific cards. This thought may force me to seek immortality in the arduous old-fashioned way, doing good deeds and taking care of my children.—DANIEL E. KOSHLAND, JR.

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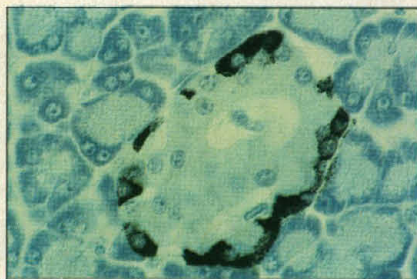
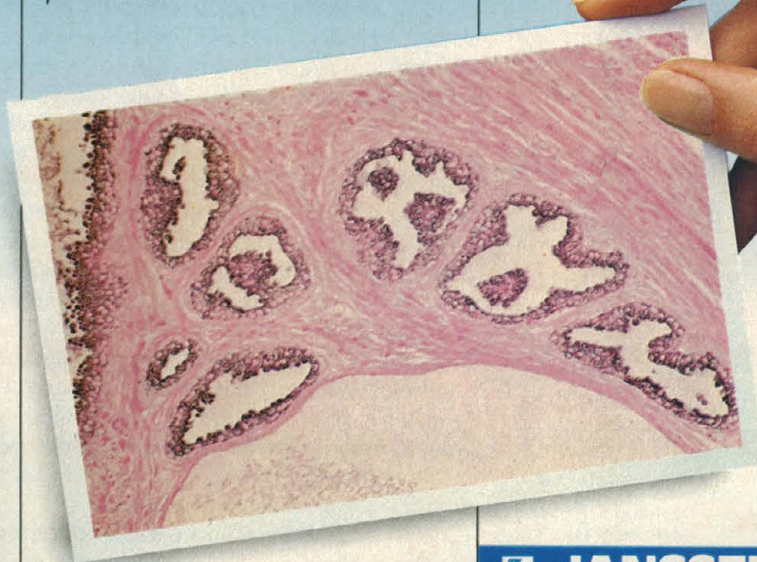
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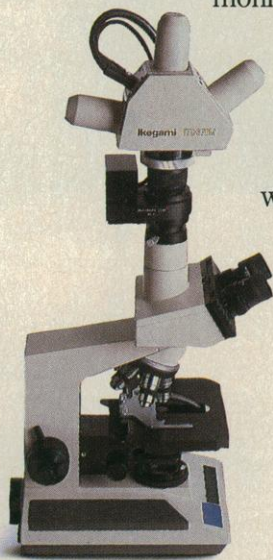
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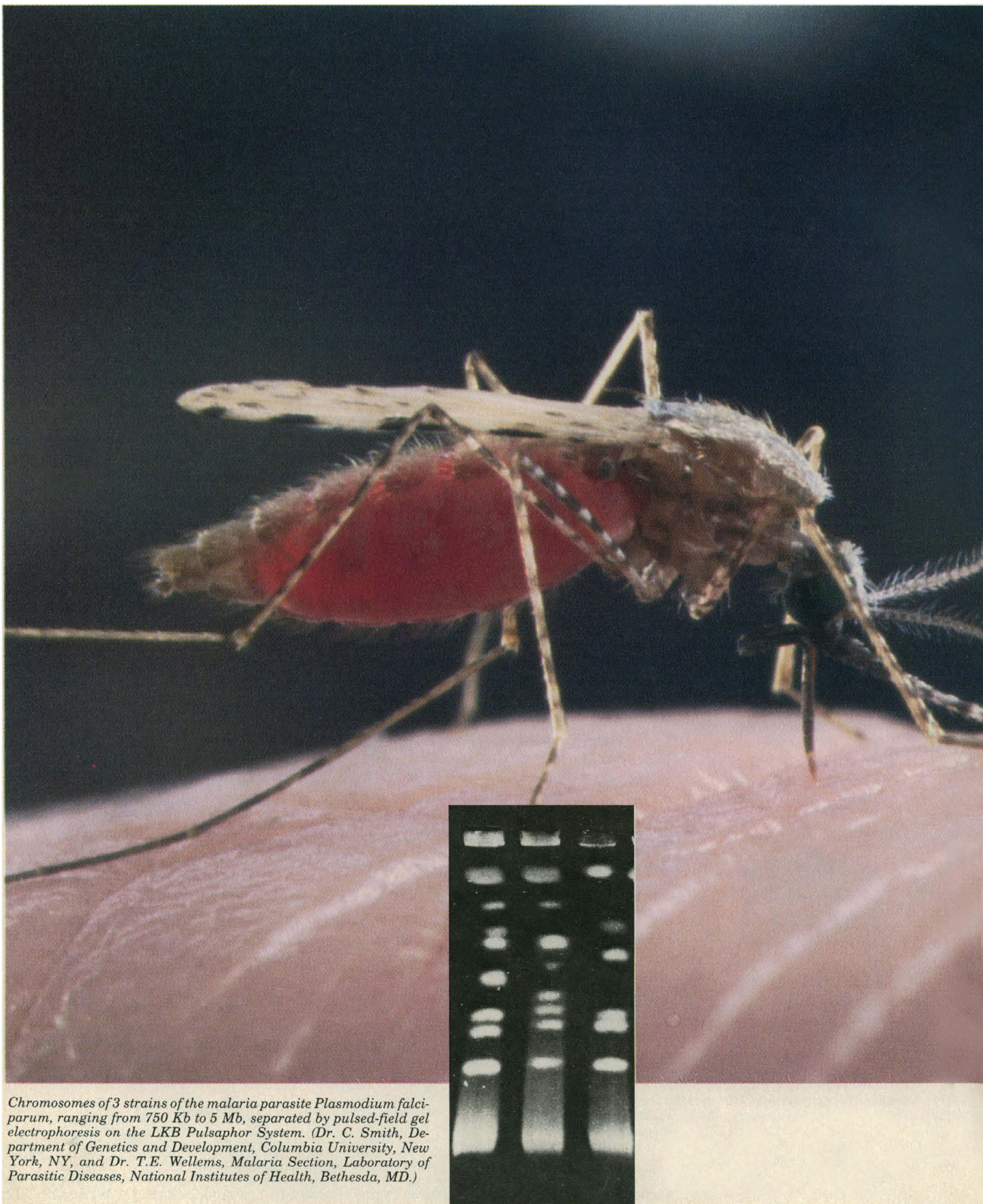
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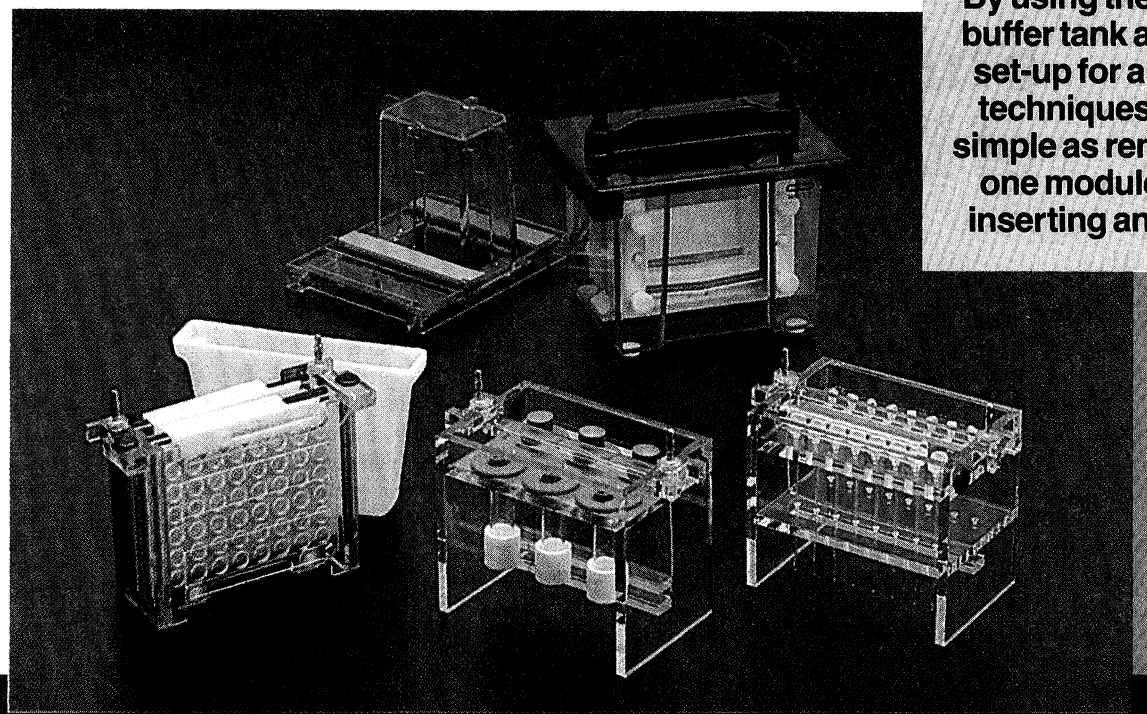
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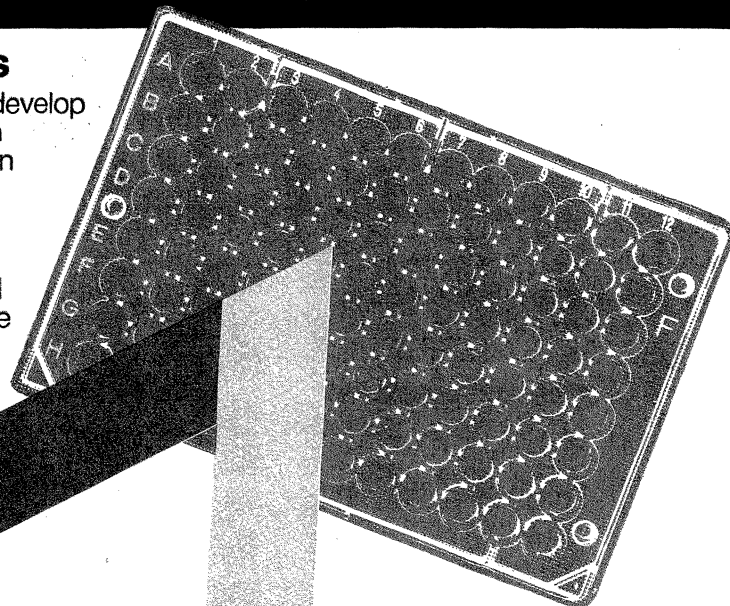
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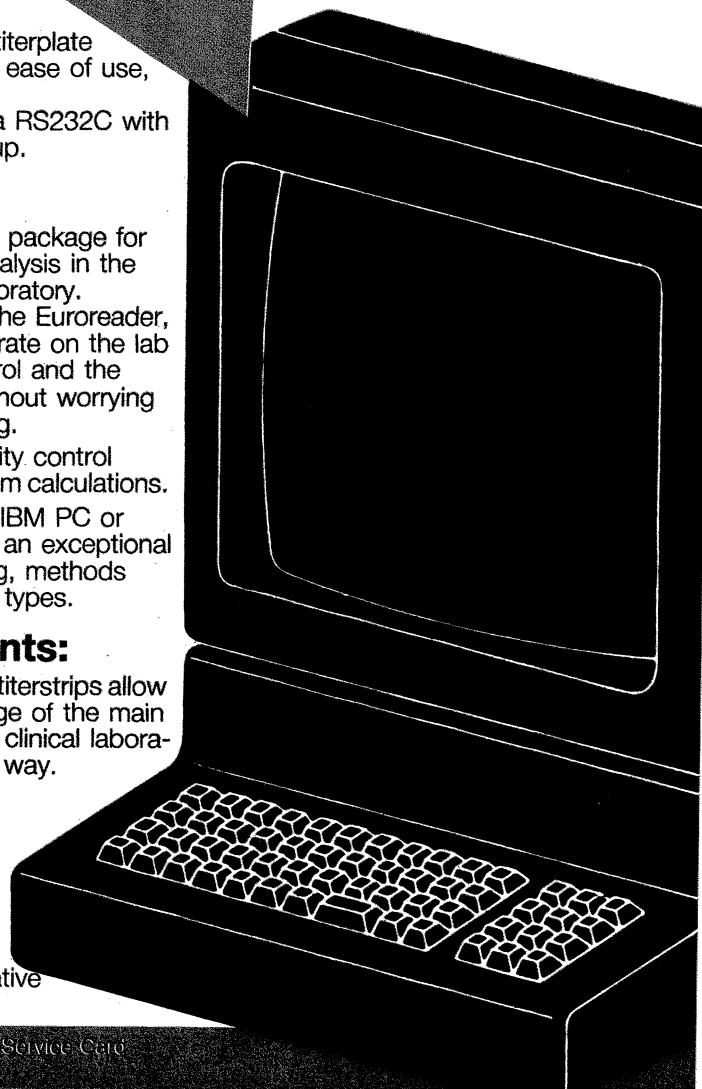
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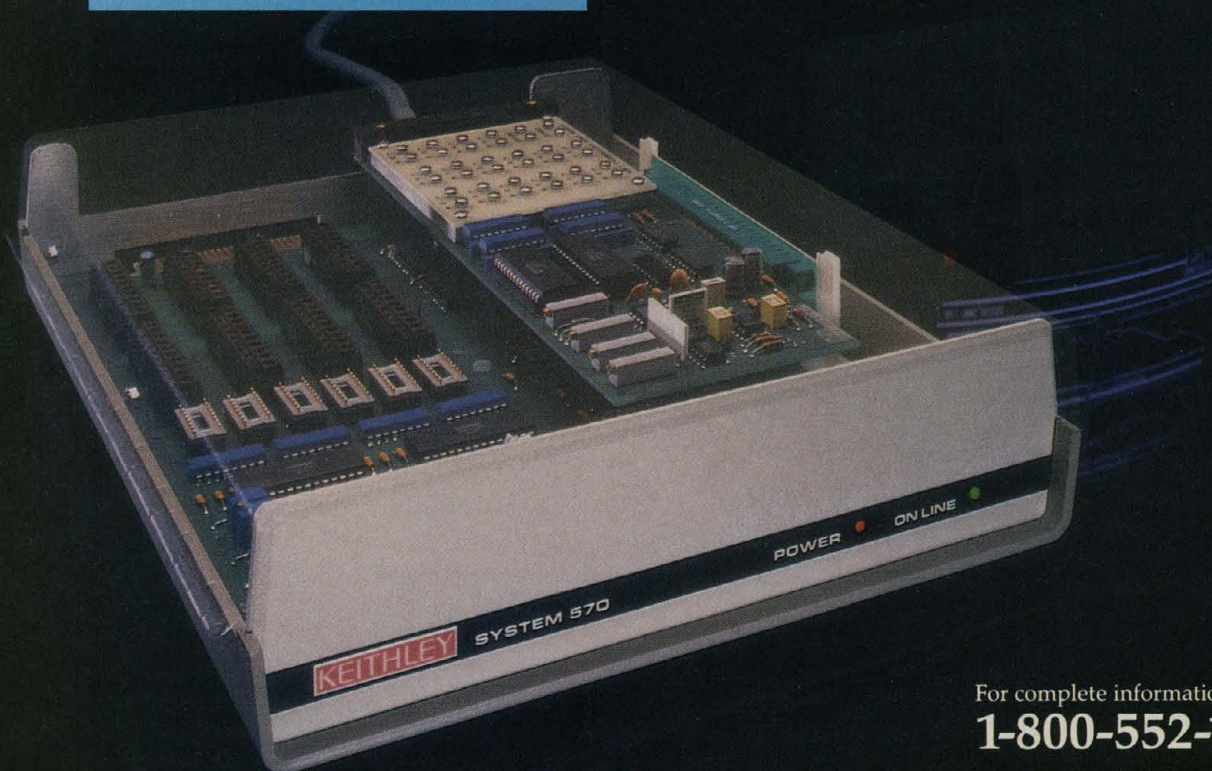
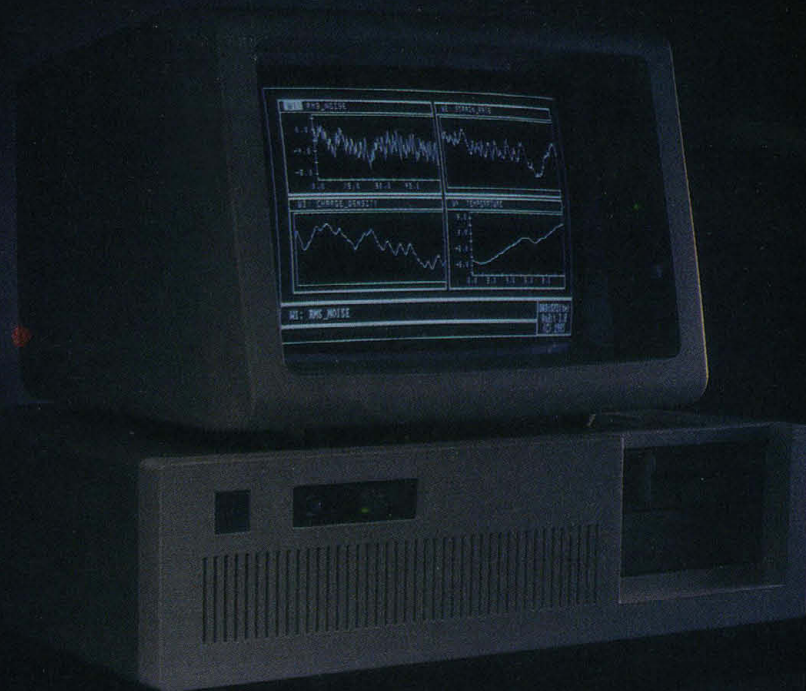


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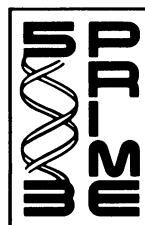
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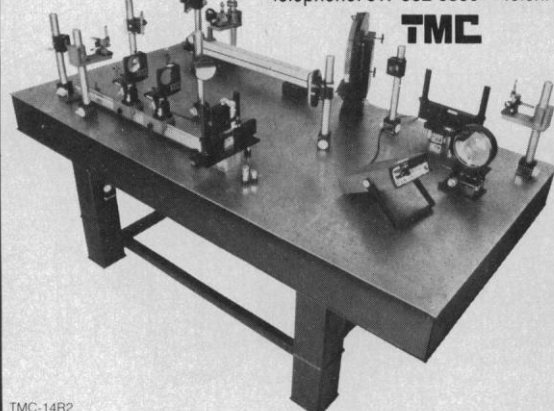
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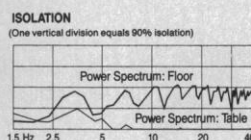
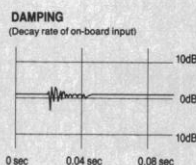
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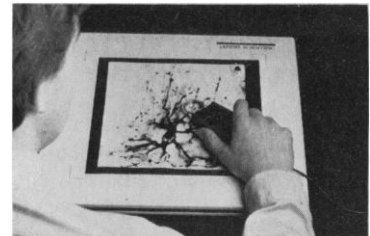


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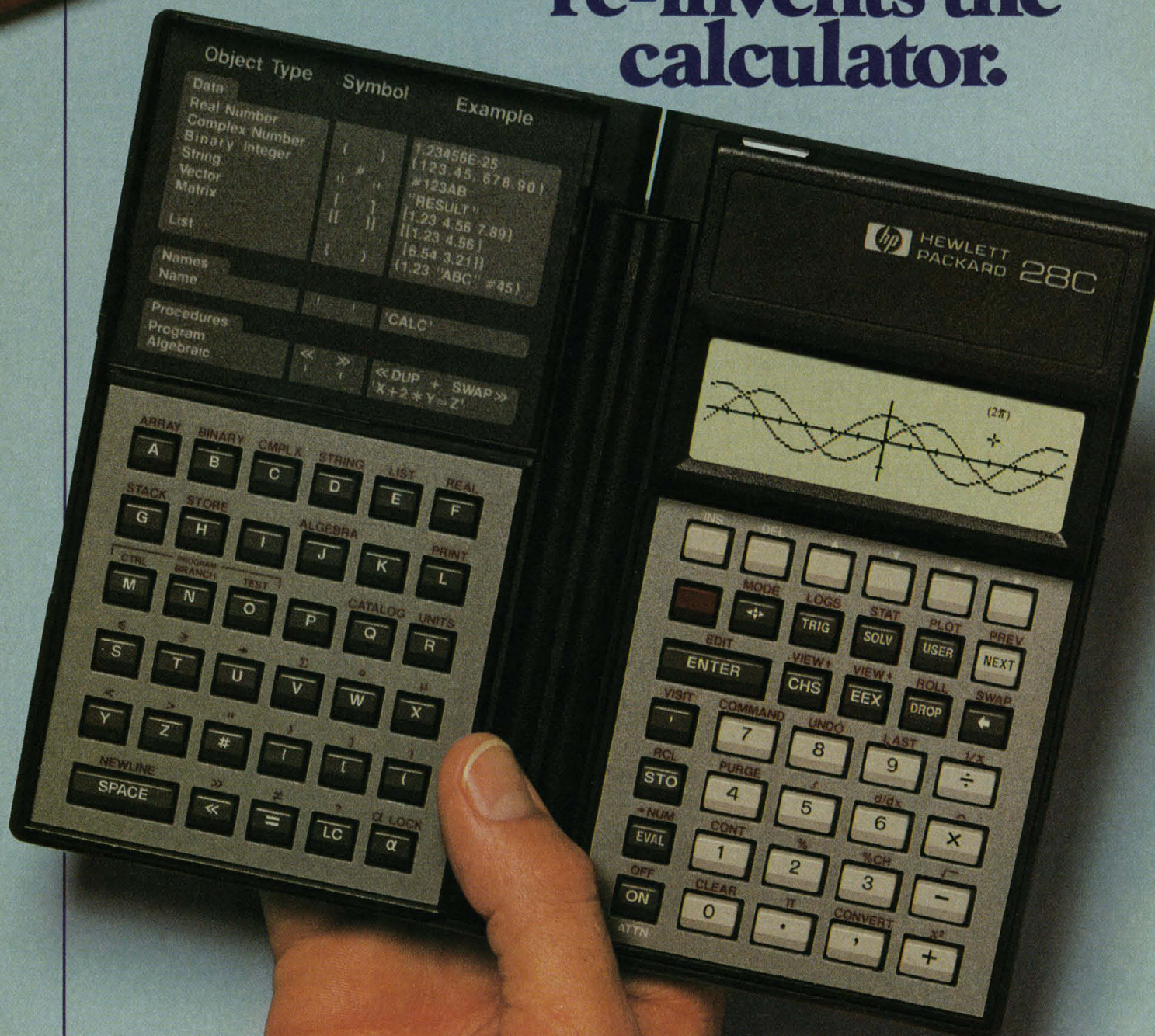
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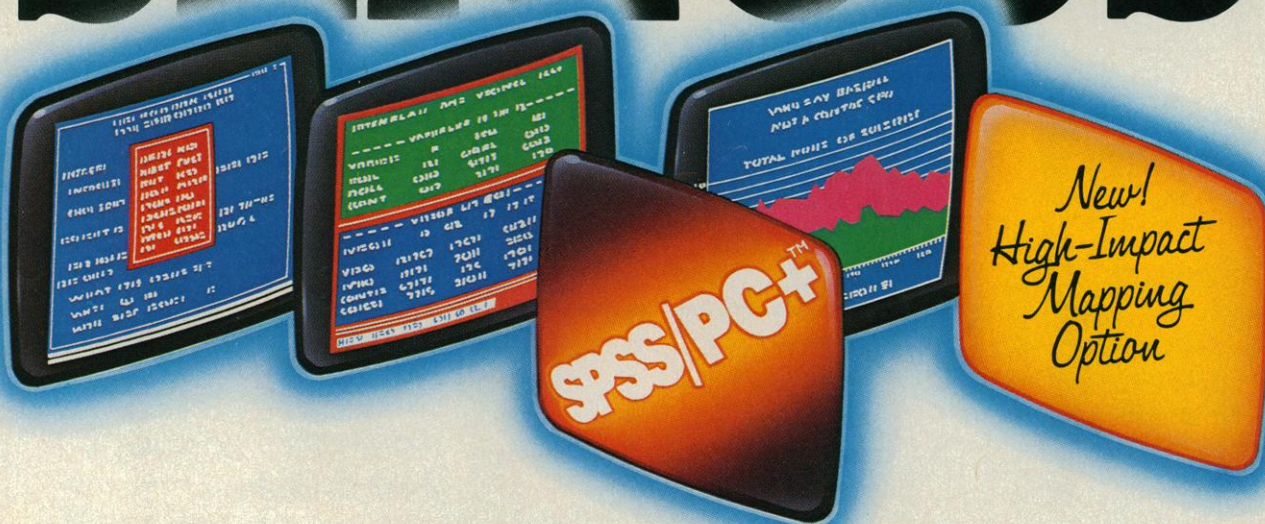
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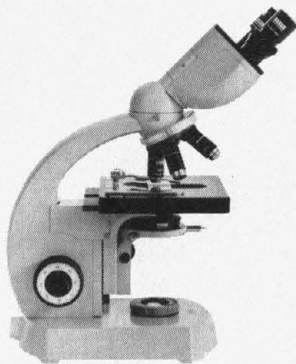
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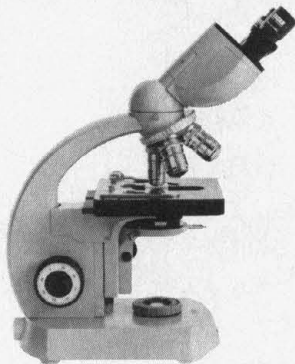
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- Porcine PDGF is structurally very different from hPDGF (i.e. differs in M.W., subunit composition, N-terminus), its activity is determined as in 3 above.
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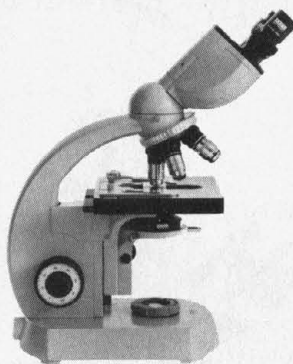
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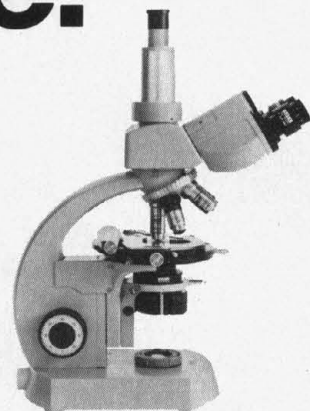
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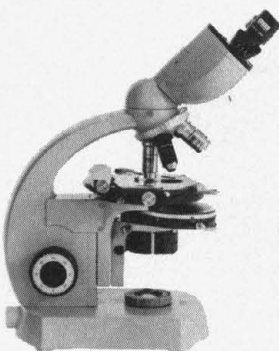
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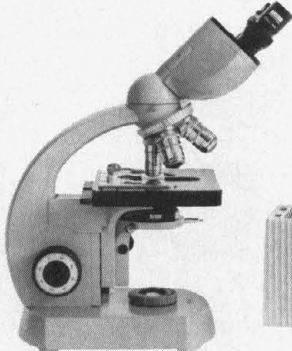
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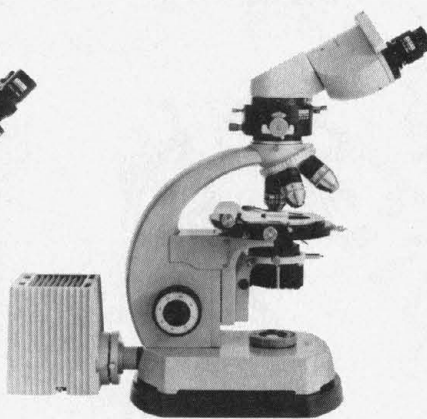
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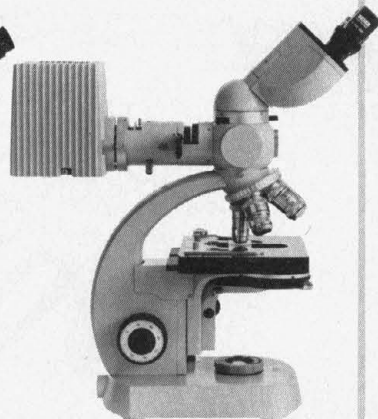
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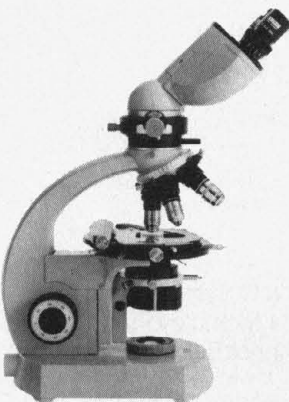
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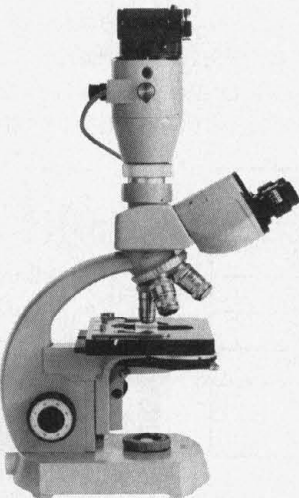
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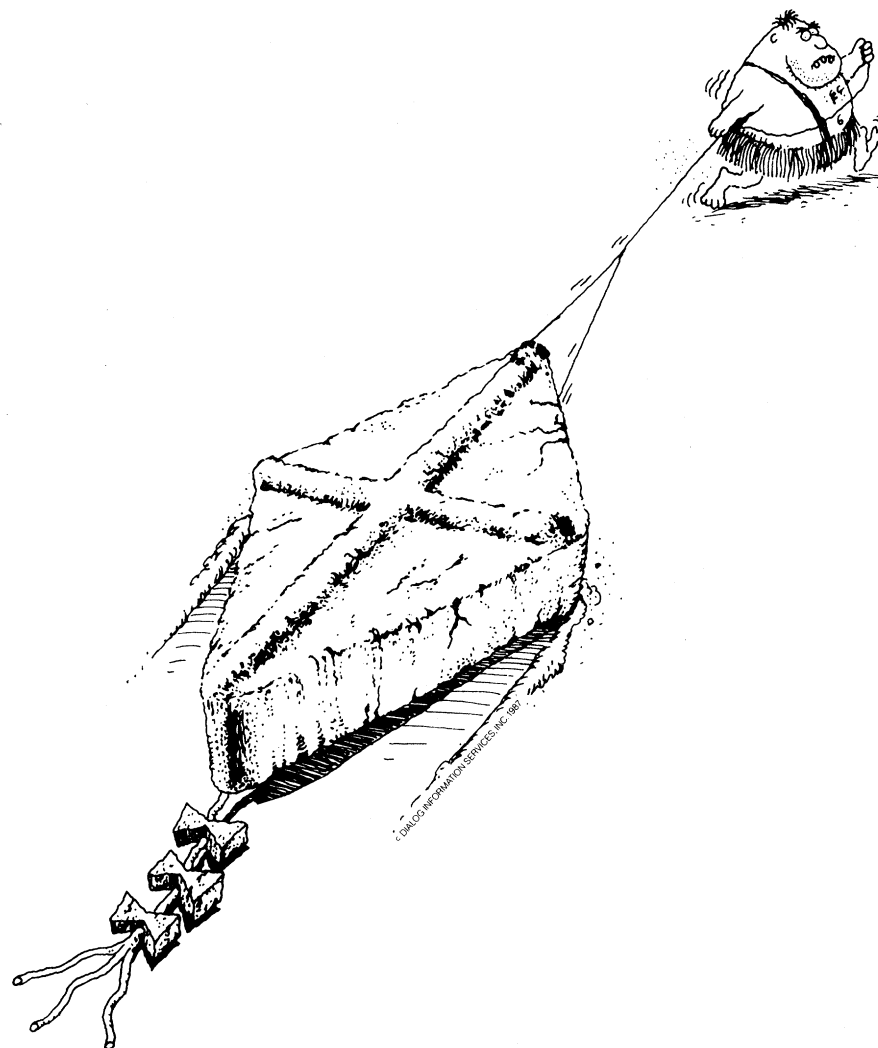
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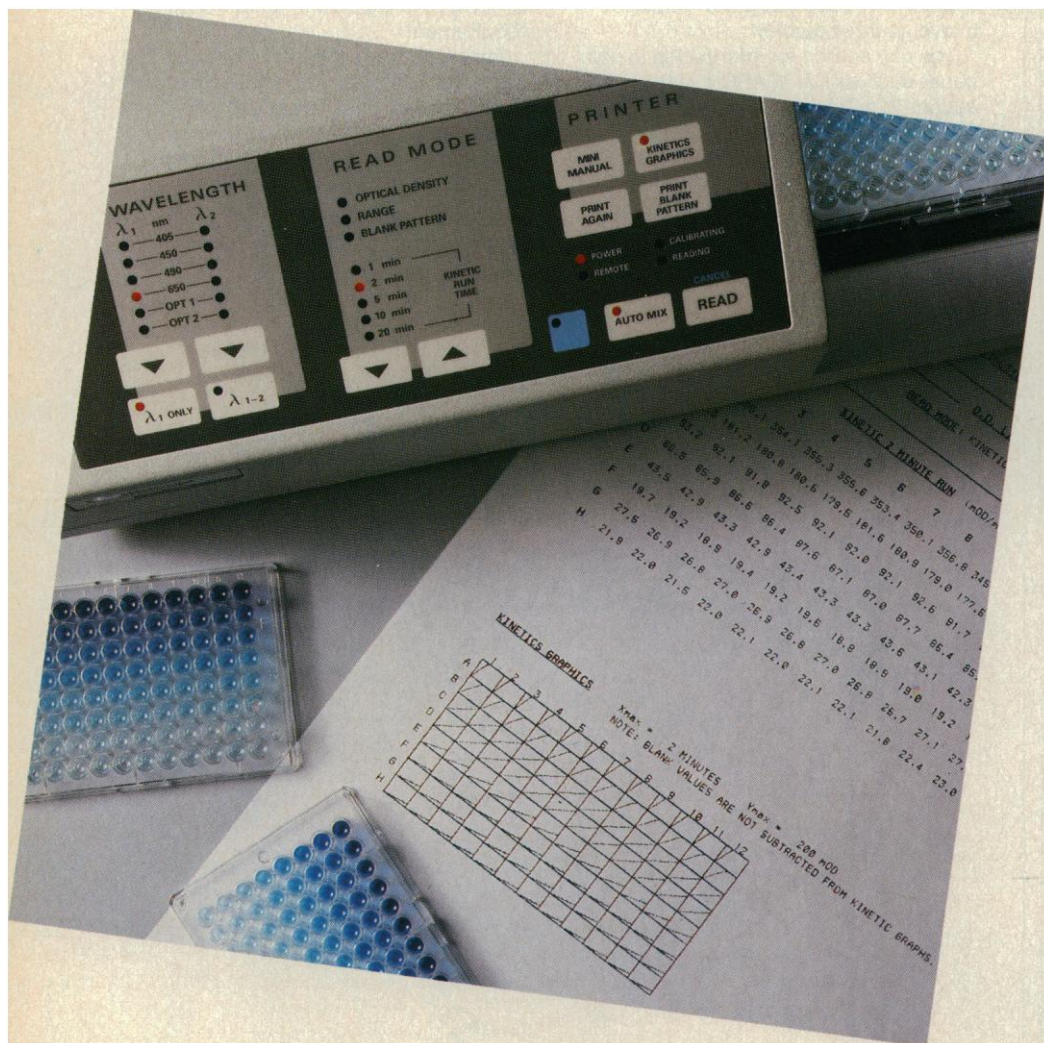
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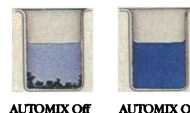


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