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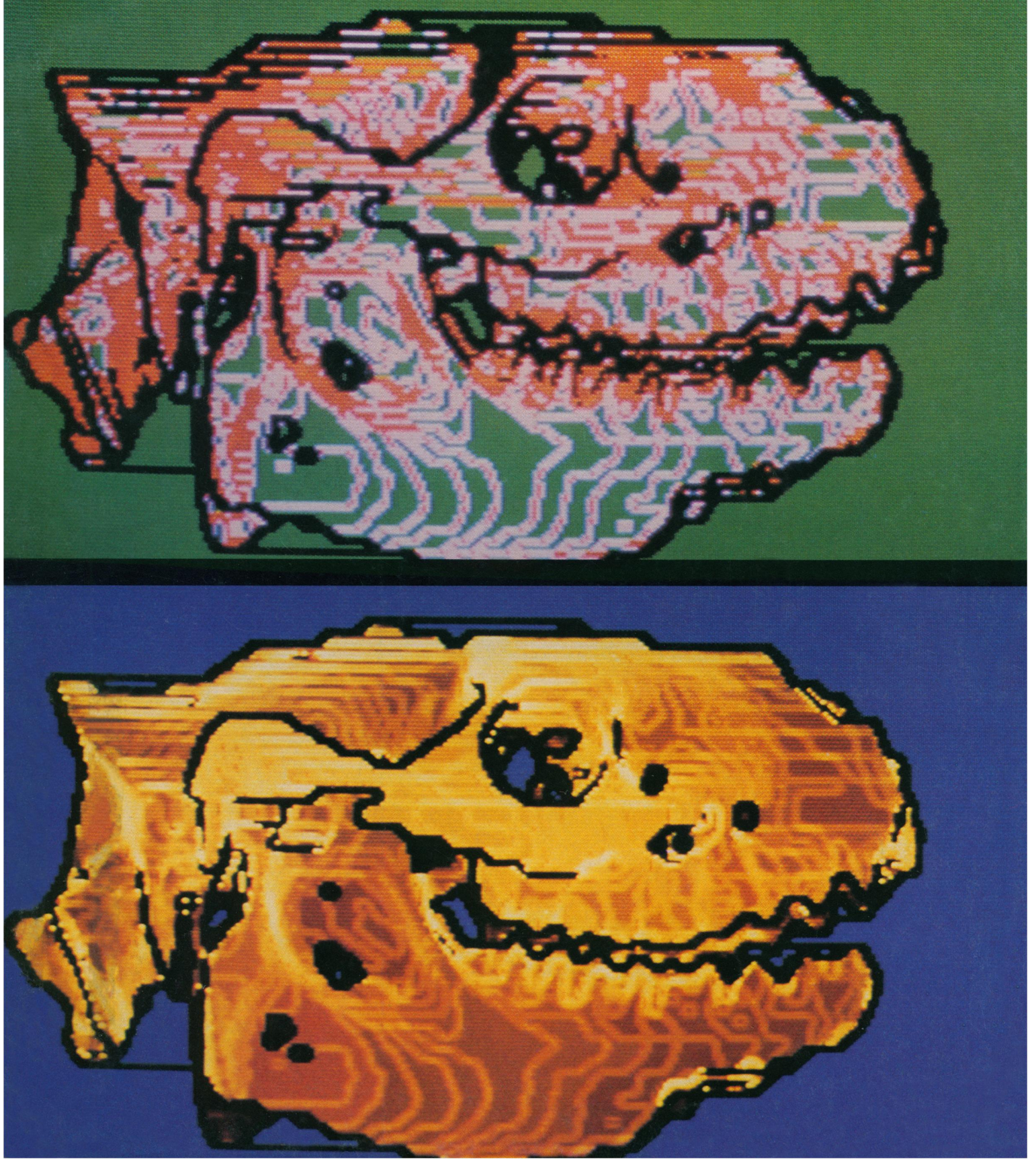

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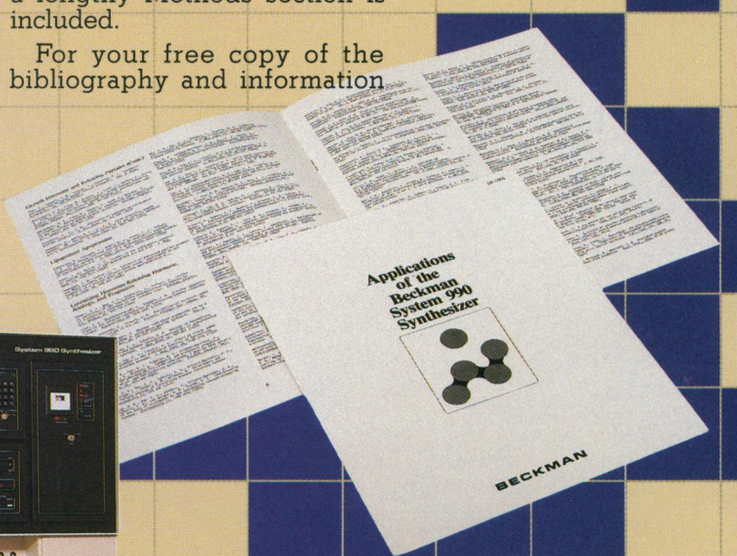

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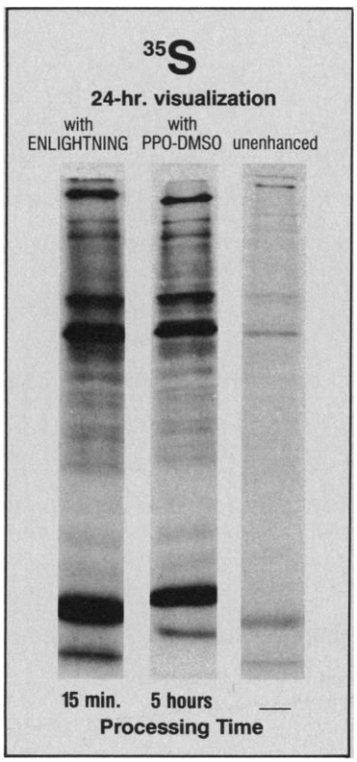

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26 October 1984

Volume 226, No. 4673

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COVER

Three-dimensional surface reconstructions of a 30-million-year-old fossil mammal (*Stenopsochoerus*) generated from serial computerized tomography scans of its skull. Both ecto- and endocranial stone matrix have been "removed" (made transparent) by computer methods to reveal the true osseous contours of the fossil. See page 456. [Photograph by C. Ungar in collaboration with M. Vannier, Washington University School of Medicine, St. Louis, Missouri 63110]

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The LKB DNA/RNA Sequencing System successfully combines all the relevant technology and know-how into a single integrated system dedicated to and optimized for nucleotide electrophoresis. Basically, the amount of sequencing information that can be obtained from one experiment is limited primarily by the resolution of the electrophoresis system used. This resolving power is in turn limited mainly by the length and thickness of the gel, by thermal and mechanical distortion, and by the so-called 'smile effect'.

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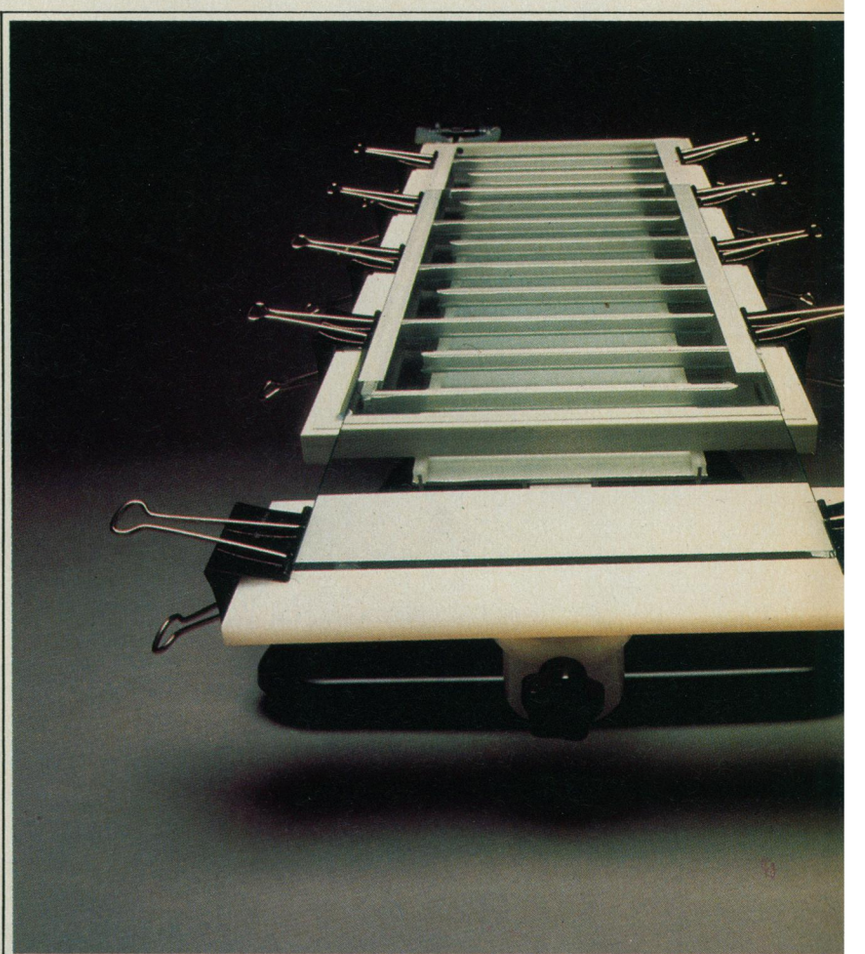

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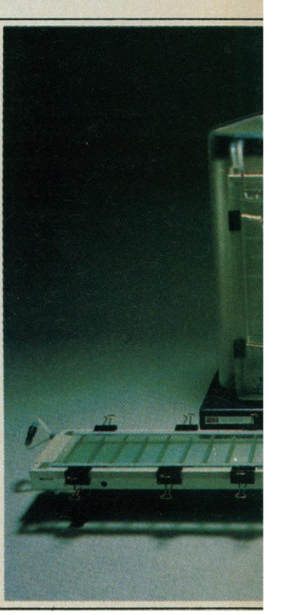

Vertical Electrophoresis Systems

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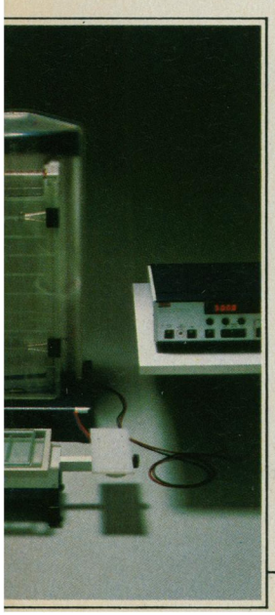
◀ *The LKB Macromould Gel Casting Unit makes it easy for you to cast reproducible and bubble-free ultrathin gels of different sizes and thicknesses in less than one minute*



Seriously, no smiling
It is desirable to carry out electrophoresis runs at a high gel temperature in order to resolve clearly those nucleotides that are still compressed in conventional systems. In the LKB Macrophor, the thermostatic plate onto which the gel is cast is used to maintain the gel temperature. Warm water is circulated through the unit by the LKB MultiTemp II, thus ensuring that the temperature is constant everywhere in the gel and that all samples are run under identical conditions. The 'smile effect' is thereby eliminated, making the bands straight and much easier to read. You can use the full width of the gel and obtain equally good results from all your samples.

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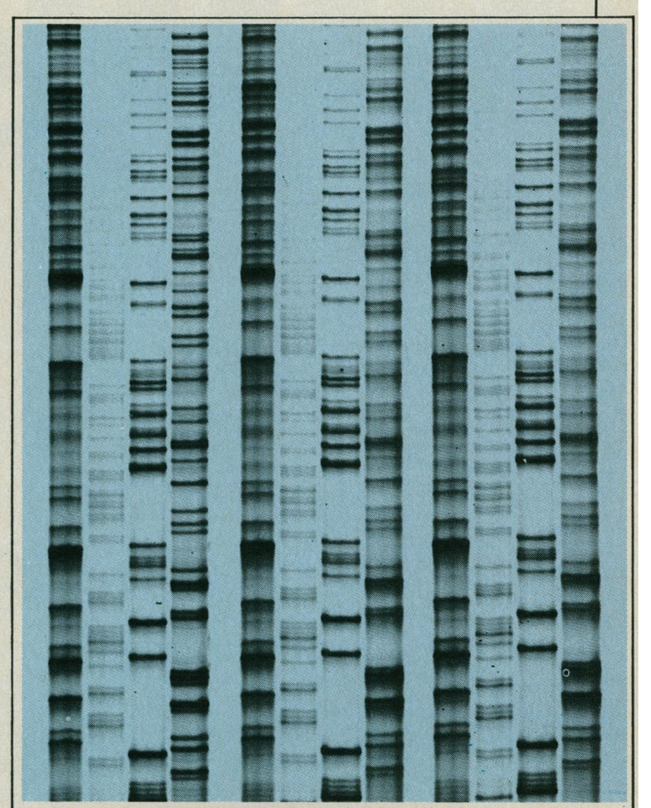

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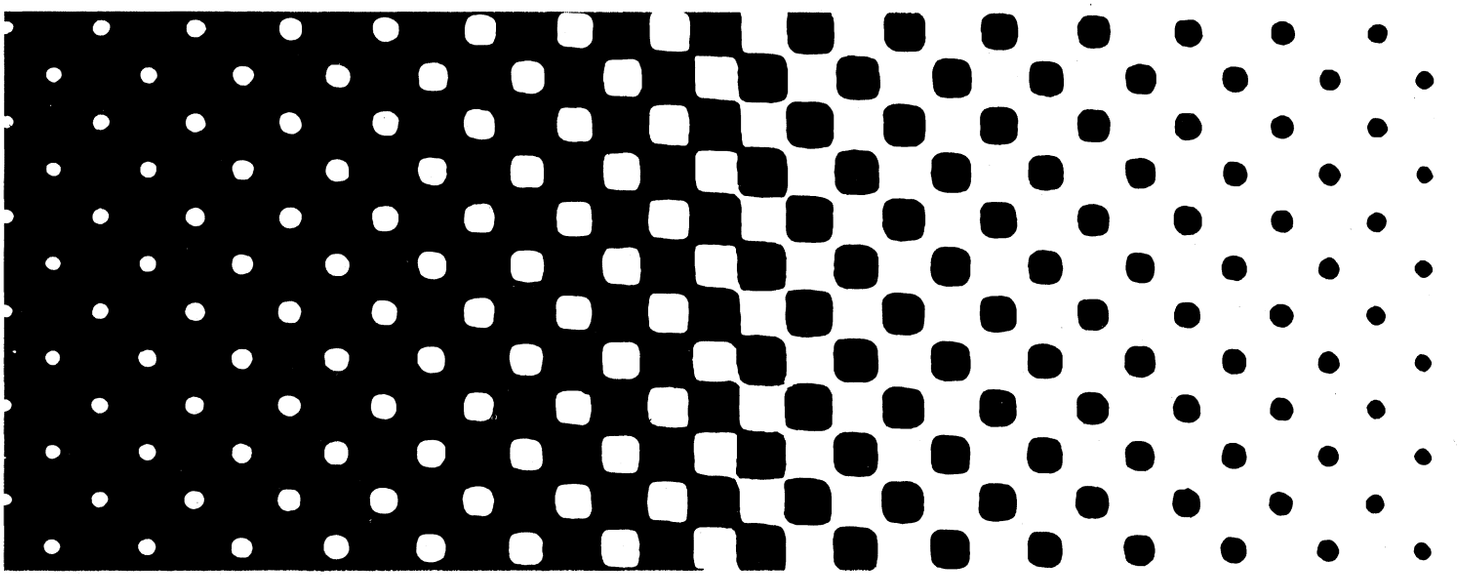

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WORKSHOP VI - TO BE ANNOUNCED

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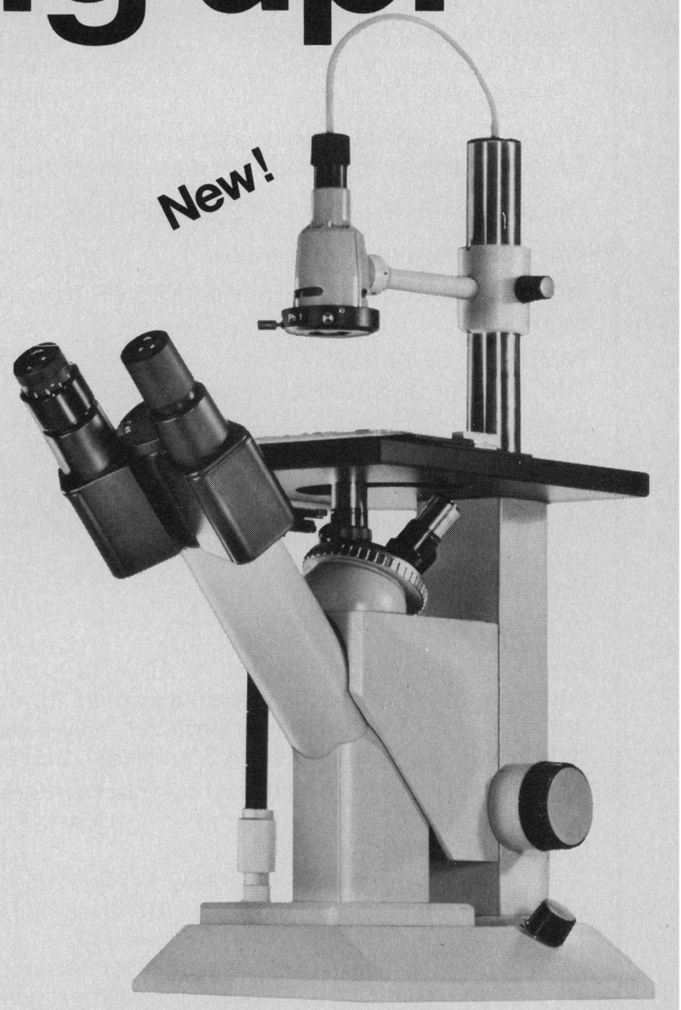

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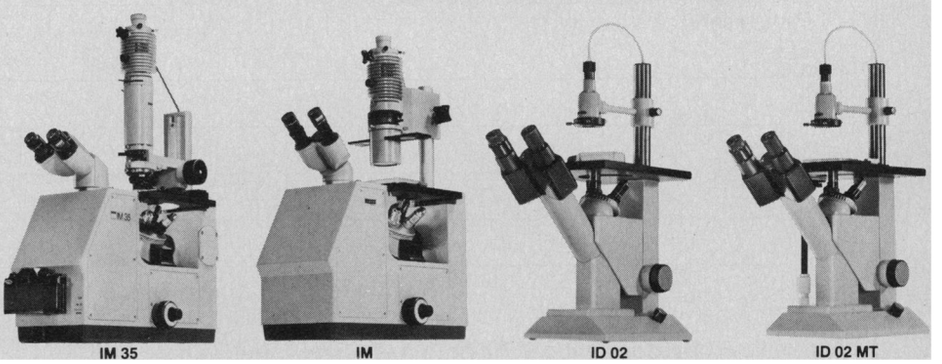

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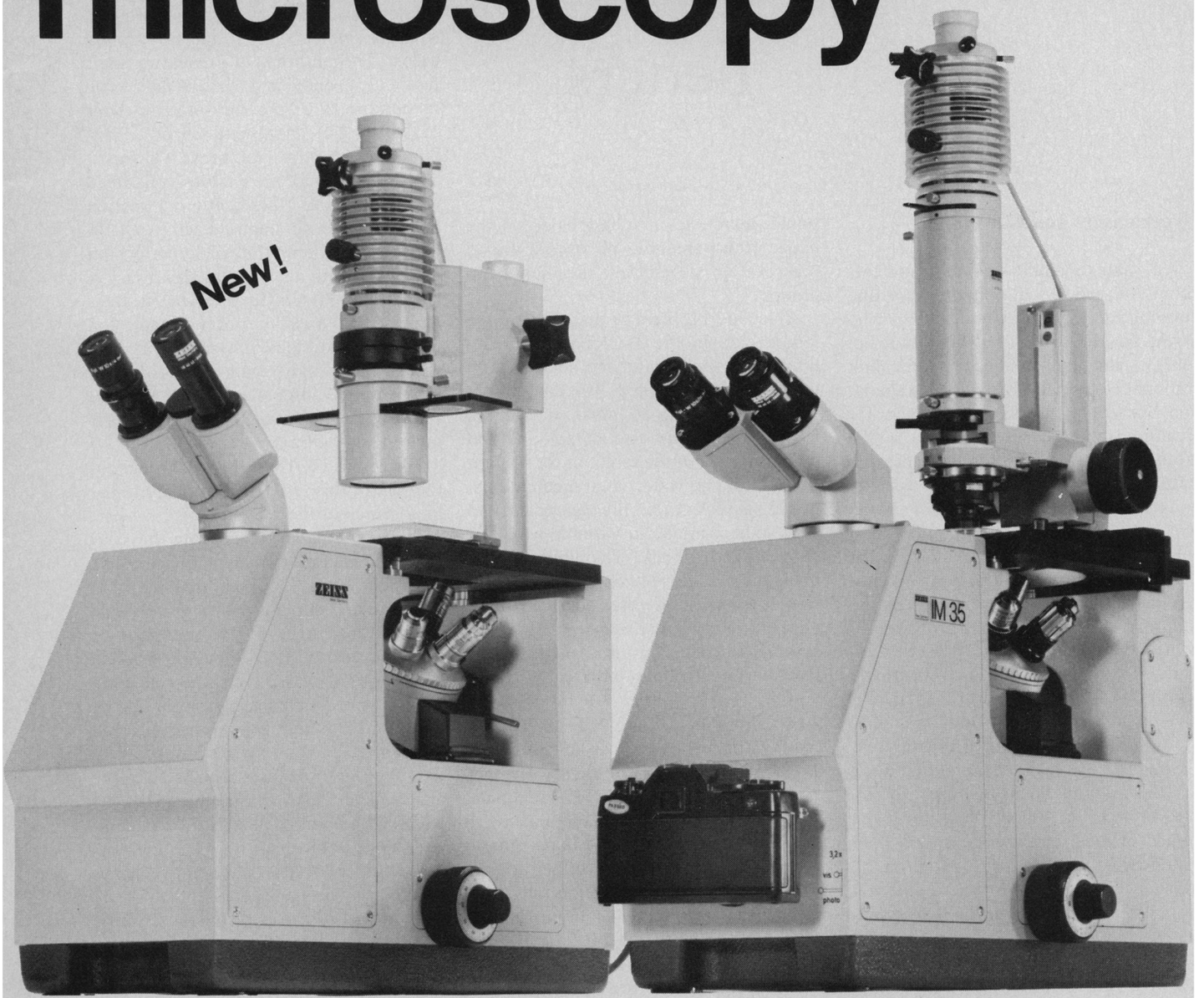

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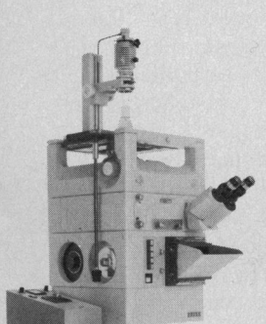

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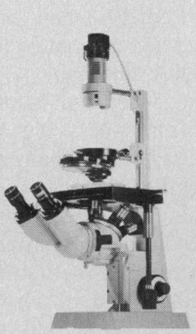

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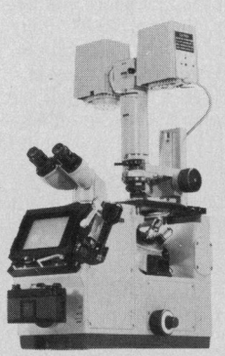

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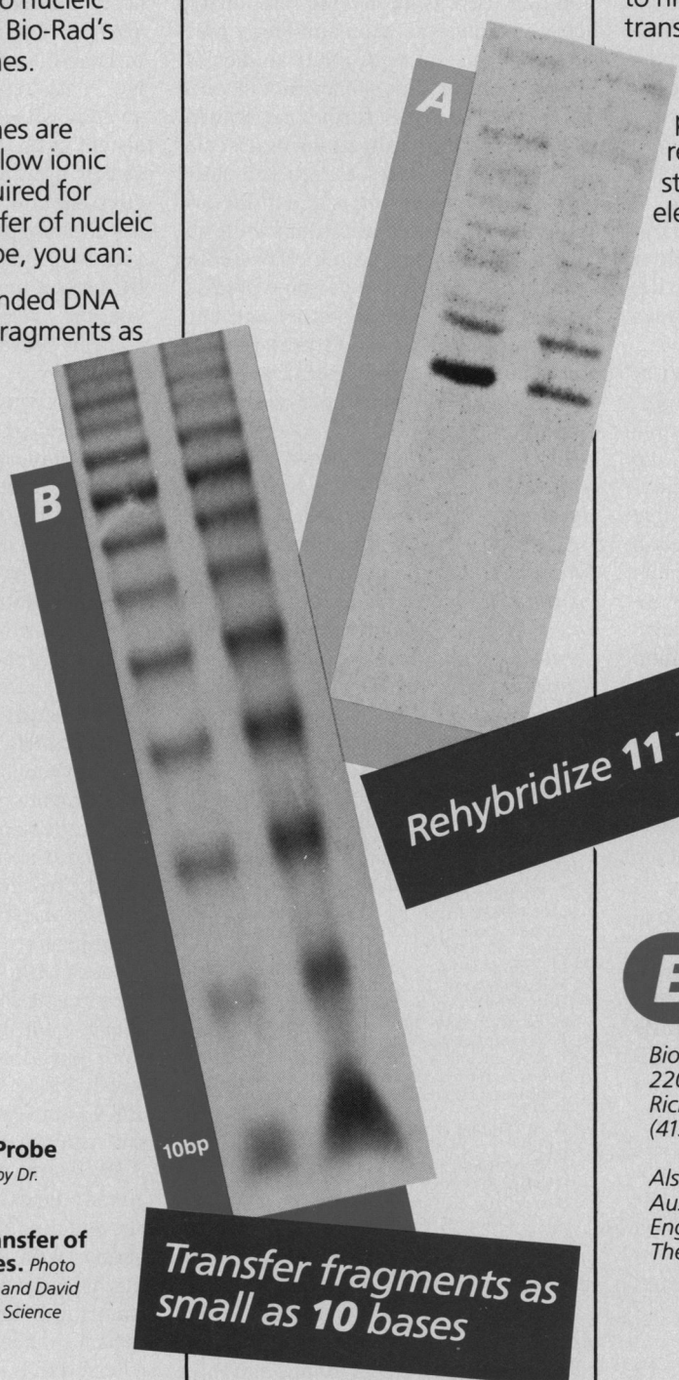

A. Eleventh round of hybridization on Zeta-Probe membrane. *Data provided by Dr. Richard A. Gatti, UCLA.*

B. Electrophoretic transfer of DNA, 10-125 nucleotides. *Photo courtesy of Drs. William Garrard and David Gross, University of Texas Health Science Center, Dallas, Texas.*

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sured, because saltshaker hole size and number of shakes are required for quantification. In fact, having nondiscretionary sodium values and not total sodium values, which would have been better estimated from 24-hour urine collections, does not necessarily bias the data. Bias could only occur if discretionary sodium did not parallel nondiscretionary intake, for example, if high sodium intakes were associated with infrequent saltshaker use, and the converse. No data exist to suggest this is true. As an example of how remarkably good the data collection was in HANES I, the average sodium intake of Americans has been measured at 140 to 160 milliequivalents (meq) per day on the basis of 24-hour urinary excretion. Discretionary sodium typically may represent 25 to 40 percent of the overall intake, or an average of about 33 percent (*6*). When one uses this average, nondiscretionary sodium intake should be 100 meq. From our HANES I analysis, the average intake was 97 meq, an excellent agreement.

In regard to replication of our sample, we are under the impression, after recent contact with the National Center for Health Statistics, that our sample size was reproduced exactly.

There are a variety of issues involved in weighting that are not completely represented by Feinleib *et al.*, of which we were aware at publication. It should not be implied that there is only one correct method for dealing with these data, particularly as this is a censored subpopulation. Feinleib, Lenfant, and Miller acknowledge that using the weighted design does not change the results in their hands. More important, weighting affects only the variances and any subsequent statistical tests. As a result, the mean values we reported are not changed by weighting, and the associations we described are therefore unaffected.

We employed discriminant analysis rather than multiple regression analysis because we believe the relation between nutrients and blood pressure may not involve a continuous outcome (in terms of blood pressure), and that a threshold may exist for a nutrient beyond which point hypertension occurs. Therefore, while still a linear method, discriminant analysis may be most appropriate with a dichotomous dependent variable such as hypertension.

The contention that our analysis did not account for age, race, and sex is incorrect. In our table 2, all three confounding factors were accounted for in the first analysis, as stated on page 1393.

As with any major confounder, it is critical to adjust for these effects to demonstrate the observed relation. It may be important, however, to consider an interaction between a nutrient and aging, as nutrient intake (of, for example, calcium) and absorption frequently decrease with aging (*7*). This may be an important consideration when assessing the reasons for the increasing prevalence of hypertension in an older age group.

The intent of our article was to present an original analysis of the HANES I data base. Had we felt it was appropriate to "square" our conclusions with the abundance of population-based and experimental data suggesting that dietary sodium indeed plays an important role in hypertension, we would, of necessity, have included the now rather substantial body of newer information instead of the older information that has been the basis for the formulation of past policy. In fact, the lack of intrapopulation research indicating a positive association of sodium and blood pressure has often been noted (*8*). The most recent findings are consistent with those observed by us in HANES I. Development of high blood pressure in "salt sensitive" models of hypertension has been dissociated from the intake of sodium (*9*). In the most widely studied model of genetic hypertension, sodium restriction has resulted in growth retardation and possible acceleration of the hypertension (*10, 11*). In one of the studies (*11*), the level of sodium restriction was within the bounds currently recommended as the "safe" level of sodium reduction for the U.S. population (*12*). Finally, recently reported studies from abroad suggest no short-term benefits of moderate sodium restriction in hypertensive subjects studied under the tightest control reported to date (*13*).

We are encouraged by the acknowledgement of Feinleib *et al.* that "sufficient evidence has accrued to justify further experimental and clinical investigation of associations between dietary calcium and blood pressure." We trust that this portends a broadening from the narrow focus on sodium as the principle factor in the pathogenesis of hypertension. We hope that this new perspective will not simply encompass calcium, but will address the role of all nutrients, as well as the complicated interactions that characterize our diet. The complex issues we face in applying this information to our understanding of the pathogenesis of this common medical disorder should be a stimulus to intensify our research efforts rather than to formulate simplified and premature therapeutic recommendations to the public. Other established investigators in the research community share our perspective and have articulated it in recent public statements (*14*).

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Erratum: In the report "Cell sensitivity to gravity," by A. Cogoli *et al.* (13 July, p. 228), the legend for figure 1b should have read: "Glucose consumed by the lymphocyte cells during the experiment. The initial concentration of glucose in the medium was 1100 mg/liter; the glucose that remained in the medium after the experiment was measured by the glucose dehydrogenase method (*6*). The standard deviation of triplicate samples is shown."

Erratum: In the News and Comment article "Use of antibiotics in animal feed challenged" (12 Oct., p. 144) by Marjorie Sun, the rate of fatalities resulting from infections caused by drug-resistant *Salmonella* was incorrectly reported. The fatality rate resulting from these infections is 21 times higher than for disease caused by *Salmonella* strains that responded to conventional antibiotics. This finding was reported by Scott D. Holmberg *et al.* in *Science*, 24 Aug., p. 833.

Erratum: In two Research News articles by Arthur L. Robinson (24 Aug., p. 822; 14 Sept., p. 1137), the affiliations of three researchers were given incorrectly. Peter Smith and Thirumalai Venkatesan (24 Aug.) are with Bell Communications Research (Bellcore), not AT&T Bell Laboratories, as stated. David Hwang (14 Sept.) is also with Bellcore.

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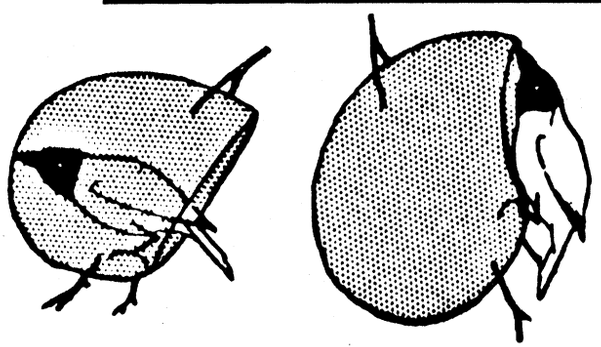

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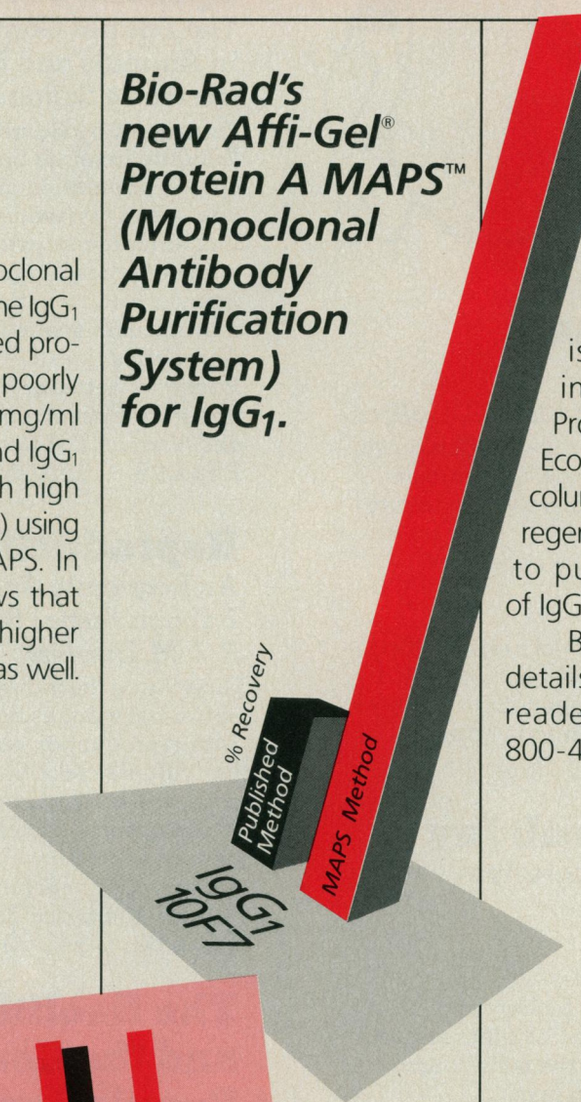

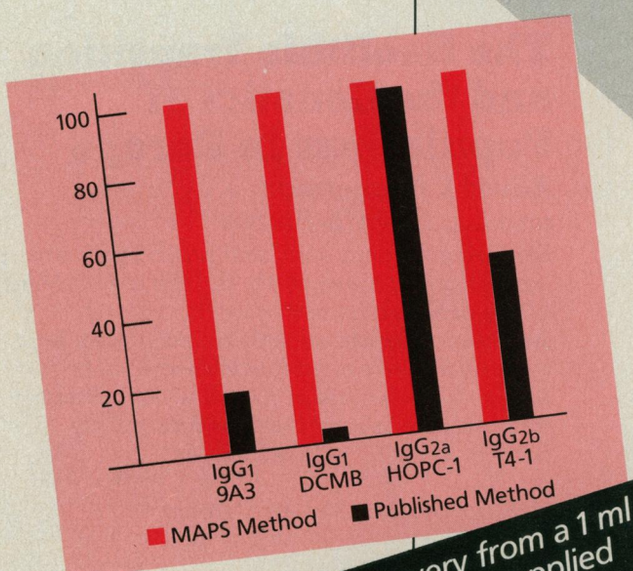

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26 October 1984, Volume 226, Number 4673 **SCIENCE**

Let Them Eat Cake

At the second United Nations conference on population, convened in August in Mexico City, the prestige and power and ostensibly the know-how of the United States went to advise the developing countries to let the play of supply and demand in the free market solve their economic and population problems. Population increase, our representatives declared, is not of itself a bad thing, and it sets up increase in demand. On the supply side, they argued, intervention by the state must not be allowed to inhibit the response of sufficiently motivated entrepreneurs.

This advice, not endorsed by the delegations of other market economies, carries the faults inherent in prescription from narrow ideology. It is not supported by the history of the industrial revolution nor by present arrangements in the societies that enjoy its benefits. In its practical import, it invites the industrial nations to renege on their unredeemed pledge, twice declared in unanimous votes in the U.N. Assembly, to help hasten the development of the preindustrial nations by providing substantial economic and technical assistance.

The ultimate size of the world population will be determined by the time it takes the poor countries of the world to complete the demographic transition: that is, the transition from near zero population growth at high death rates and high birth rates to near zero population growth at low death rates and birth rates. The people of Europe, at home and in the lands they settled around the world, made that transition in about 400 years. Until the 17th century, they had the same 25-year life expectancy as the rest of the world population. Over the years in which they carried through their scientific-industrial revolution, they multiplied their numbers 20 times. Enjoying at last the physical well-being that ensures the survival of their infants, they have brought their birth rates down to the low level of their death rates that gives them life expectancies in excess of 70 years.

The rest of the world population has meanwhile multiplied its numbers about ten times. With their death rates falling as they come through the first phase of the demographic transition, that ten times will double to 20 times in the next 30 years. Much less than a century remains to see the world population stabilize at a technologically, politically, and morally tolerable number.

The rate and scale of economic development required to secure the necessary popularization of physical well-being exceed by many times what can be expected or hoped for from the market process. Ahead of the developing countries still remains the immense task of putting in place the infrastructure of industrialization. In the United States—and they know this as well as we—it is a long time since the building of turnpikes was left to private enterprise. Our railroads are nationalized in fact if not in name. The building of the great dams and water distribution systems that sustain agriculture west of the 100th meridian is a sanctioned federal enterprise. With money earning double-digit interest, our smokestack industries quit investing in plants a decade ago. The two U.S. industries that earn foreign exchange—aircraft and electronics—enter into the domestic and world markets with the cutting edge of their technologies secured by subsidy by the purchasing power of the federal government, especially that of the Department of Defense.

For some substantial number of the world's poor, the United States still holds out the future to which they aspire. What they require from us is not advice—and surely not advice carrying the mindless condescension Marie Antoinette offered to the poor of Paris—but action alongside them in the task of hastening their economic development. Belonging to the same world population, we have as large a stake in the outcome as they do.—GERARD PIEL, *President-Elect, AAAS, and Chairman of the Board, Scientific American, Inc., New York 10017*

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