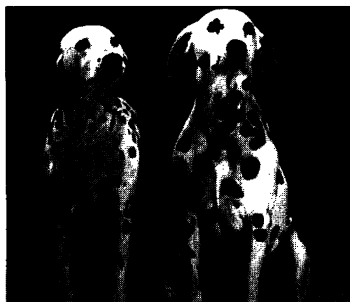


Investigator™

Electrophoresis Brought To Life



Spot the difference...

The power of 2-D electrophoresis is now brought to both analytical and preparative scale applications. Using Oxford GlycoSystems' Investigator™ 2-D electrophoresis system, you can move easily from 1st dimension isoelectric focusing (IEF) to 2nd dimension PAGE gels, then to blotting and sequencing your target protein.

...an obvious choice

Investigator 2-D systems are the most widely used complete 2-D electrophoresis systems.

- Duracryl™ high performance gels
- Dedicated reagents
- High resolution and reproducibility
- Integrated temperature control system
- Up to 10 gels per run
- Preparative gels

With a capacity of up to 1 mg protein total, and recovery of up to 10 µg from a single spot, you'll find the Investigator the obvious choice.

For a FREE information pack on how Investigator™ systems can improve your electrophoresis applications contact us today.



USA and Canada: Tel: (617) 533 0100,
Fax: (617) 533 0101, Tollfree: 1-800 722 2597
UK and Europe: Tel: +44 (0)1235 553066,
Fax: +44 (0)1235 554701, Tollfree: 0800 212061
To reach us from Switzerland 155 2739;
France 0590 86 08; Germany 0130 81 37 48
Japan: Tel: 3-3862-2108, Fax: 3-3866-5165

Circle No. 30 on Readers' Service Card

most of human history. Evidence to support the first statement is based on studies of skeletal remains (1). However, there are problems with interpreting paleodemographic data, including whether the skeletons reflect actual mortality by age and sex in the population being studied and whether mortality in the populations being studied reflects mortality more generally. To construct an accurate life table from skeletons would require all or a random sample of deaths to be found at the burial sites, either a stationary population so that the age distribution of deaths in the population is the same as that in the life table or a stable population with known rate of growth, and accurate estimation of age at death and determination of sex. Evidence against both the first and the second statements is provided by estimated mortality rates from more recent populations with very high mortality but with data whose reliability is far more certain. In Sweden, the country with the longest historical series of reliably recorded mortality data, expectation of life at birth (e_0) for females has exceeded that for males since the first period (1751–1790, when e_0 was 36.6 years for females and 33.7 years for males) that official estimates are available (2). Estimated life tables for other high-mortality populations also show a female advantage; in India [from the first period (1872–1881) for which estimates are available, when e_0 was 25.6 years for females and 23.7 years for males, through 1911–1921 (3)], among immigrants to Liberia [1820–1843, when e_1 was 24.6 years for females and 22.9 years for males who survived the calendar year of arrival (4)], and among the British peerage [from the first period (1550–1574) when estimates are available, when e_0 was 38.2 years for females and 37.8 years for males, through 1700–1724 (5)]. The statistics Mann gives for female and male life expectancy for the United States in 1920 are correct, but the differential favoring females also existed for each year from 1900 to 1919 (6). Although there are no official national estimates before 1900, when the national death registration area was established, estimated life expectancy for females has exceeded that for males in Massachusetts since the first year (1850) that estimates are available (5). In 1995, expectation of life at birth for females is estimated to exceed that for males in every country except Bangladesh, Bhutan, India, Nepal, and Pakistan (7). Discrimination against females in South Asia has long been recognized by demographers as the source of this anomaly.

James Trussell

Office of Population Research,
Princeton University,
Princeton, NJ 08544–5263, USA
E-mail: trussell@princeton.edu

References

1. G. Y. Acsádi and J. Nemeskéri, *History of Human Life Span and Mortality* (Akadémiai Kiadó, Budapest, Hungary, 1970).
2. National Central Bureau of Statistics, *Historical Statistics of Sweden. Part 1. Population. 1720–1967* (Beckmans, Stockholm, ed. 2, 1969).
3. K. Davis, *The Population of India and Pakistan* (Princeton Univ. Press, Princeton, NJ, 1951).
4. A. McDaniel, *Demography* **29**, 581 (1992).
5. T. H. Hollingsworth, *Population* **32** (numéro spécial), 323 (1977).
6. Bureau of the Census, *Historical Statistics of the United States: Colonial Times to 1970, Part 1* (U.S. Department of Commerce, Washington, DC, 1975).
7. C. Haub and M. Yanagishita, *1995 World Population Data Sheet* (Population Reference Bureau, Washington, DC, 1995).

MHC Class I Gene Expression

Harald Neumann *et al.* (Reports, 28 July, p. 549) elegantly demonstrate that major histocompatibility complex (MHC) class I genes are expressed by a portion of neurons cultured from the rat hippocampus. Cells expressing the MHC class I antigen did not exhibit spontaneous action potentials, while neurons that did not express the antigen had spontaneous action potentials. Treatment of electrically "silent" cells with gamma interferon increased expression of MHC.

We have previously demonstrated (1) that neurons of murine trisomy 16 (mts16) fetuses (16 to 18 days after conception) in vivo expressed large amounts of class I MHC H-2Kk antigen and increased synthesis of messenger RNA that binds a 33-base antisense complementary DNA probe to a region in exon 2 of the H-2Kk sequence. The reactive neurons were from the trigeminal ganglion, thalamus, and cerebellum. This finding is related to the report by Neumann *et al.* because mts16 animals have an increased gene dosage for interferon alpha and beta receptors (2); both interferons increase expression of class I MHC antigens, and an increased gene dosage for the receptor may cause cells to respond in a manner similar to that observed when high doses of interferons are administered.

An implication of the findings of Neumann *et al.* is that expression of class I MHC molecules occurs in functionally impaired neurons. The cerebellum is one of the more developmentally disturbed regions in mts16 brain (1). The neural dysgenesis seen in mts16 conceptuses may be a consequence of high numbers of interferon receptors on these cells and the resulting increase in MHC class I expression. Alternatively, it may result from other factors.

Steven Kornguth
Edward Bersu
Kenneth Mack

University of Wisconsin Medical School,
Madison, WI 53705, USA

References

1. S. Kornuth, K. Mack, E. Bersu, *Biochem. Biophys. Res. Commun.* **179**, 102 (1991); S. Kornuth *et al.*, *Ann. N.Y. Acad. Sci.* **477**, 160 (1986).
2. D. R. Cox, L. B. Epstein, C. J. Epstein, *Proc. Natl. Acad. Sci. U.S.A.* **77**, 2168 (1980).

Corrections and Clarifications

In the editorial "In transition" by Barbara Jasny (20 Oct., p. 359), the genus of the organism *Mycoplasma genitalium* was inadvertently given as "Mycobacterium" in the last line of the first paragraph.

In the first report listed in the Table of Contents in the issue of 13 October (p. 211), the name of author C. M. Lieber was misspelled.

The URL address listed for Pete Goldie in his letter of 13 October (p. 218) was incorrect. It should have read, "<http://lbin.com>"

In the Research News article "Designer tissues take hold" by Robert F. Service (13 Oct., p. 230), Katherine Tweden's name was spelled incorrectly. Also, her experimental results were completed in 3 weeks, not 33.

In Paul Selvin's article "The future university: Leaner and meaner?" (Careers '95: The future

of the Ph.D., 6 Oct., p. 135) it was reported that the University of Michigan's Institute for Mental Health Research was closed during the 1980s. In fact, the institute is now celebrating its 40th anniversary. The institute that was closed was the Institute for the Study of Mental Retardation and Related Disabilities, which had formerly been the Institute for the Study of Mental Retardation. *Science* regrets the error.

In the Book Reviews section of 13 October, the two photographs at the bottom of page 319 were inadvertently interchanged.

The News article "Wing scales may help beat the heat" by Wade Roush (29 Sept., p. 1816), should have described *Urania fulgens* as a moth, not a butterfly.

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-289-7562), or regular mail (*Science*, 1333 H Street, NW, Washington, DC 20005). Letters will not be routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. Beginning in October 1995, our previous policy of consulting with all letter authors before publication will be discontinued.

• DNA Sequencing Fully Documented

• Custom Oligo Synthesis \$1.50 Base

Fast Turn-around
Cost Effective
Reliable

SSR

Molecular Biology Into the 21st. Century

SOUTHWEST
SCIENTIFIC RESOURCES
5300 Sequoia NW, Suite 150
Albuquerque, NM 87120

Phone: (800) 343-8998
Fax: (505) 831-2635

Circle No. 65 on Readers' Service Card



Custom Transgenic Rat and Mouse Production

DNX offers comprehensive transgenic mouse and rat production programs which include the microinjection of your gene construct and DNA analysis to confirm the presence of your gene construct.

- Transgenic Mice and Rats Guaranteed
- Mice Delivered in Less Than 12 Weeks
- Proven Capability with Over 700 Genes
- Specific Murine Pathogen Free Animal Colony
- Shipments to Locations Worldwide

Transgenic Model Development

In addition to producing your transgenic animals DNX will work in collaboration with your scientists to characterize your transgenic animals. DNX will assist in gene construct design, establish your transgenic lines, characterize mRNA expression and protein expression. Our experience will result in valuable transgenic animal models for your research programs.

DNX Transgenic Technology Group

303B College Road East
Princeton Forrestal Ctr.
Princeton, NJ 08540

DNX is the exclusive commercial licensee under U.S. Patent #4,873,191 DNA Pronuclear Microinjection

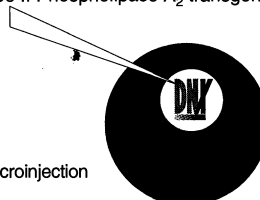
(609) 520-0300 Phone
(609) 520-9864 FAX

Add Value to Your Drug Discovery Programs With In-Vivo Compound Screening and Evaluation in Transgenic Animal Models

DNX affiliate Pharmakon Research International, offers in-vivo compound screening and evaluation with your compounds in custom DNX transgenic mouse or rat models or in our BIODIGM® Animal Models. Transgenic models expressing disease related genes and gene products will give you greater confidence in your in-vivo data than traditional animal models.

BIODIGM® Animal Models now available:

- BIODIGM®-CETP: Transgenic mice expressing human Cholesteryl Ester Transfer Protein.
- BIODIGM®-ApoB: Human Apolipoprotein B100 transgenic mice.
- BIODIGM®-CETP/ApoB: Transgenic mice with high LDL-Cholesterol, and low HDL-Cholesterol.
- BIODIGM®-ApoE3: Receptor binding deficient human ApoE3 transgenic mice.
- BIODIGM®-CETP/ApoE3: Transgenic mice expressing both the human CETP and mutant ApoE3 genes.
- BIODIGM®-PLA₂: Type II Phospholipase A₂ transgenic mice.



Circle No. 2 on Readers' Service Card