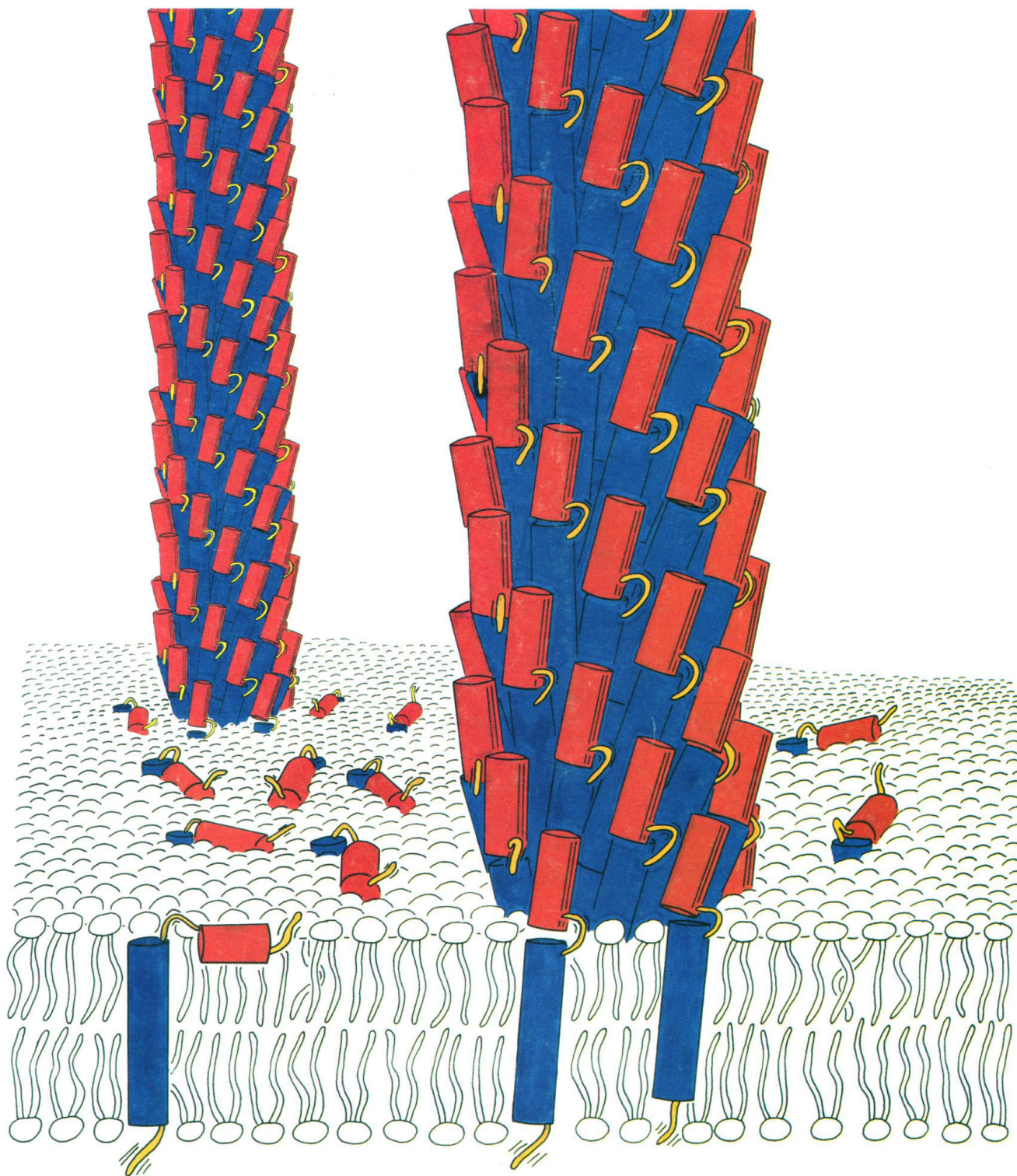


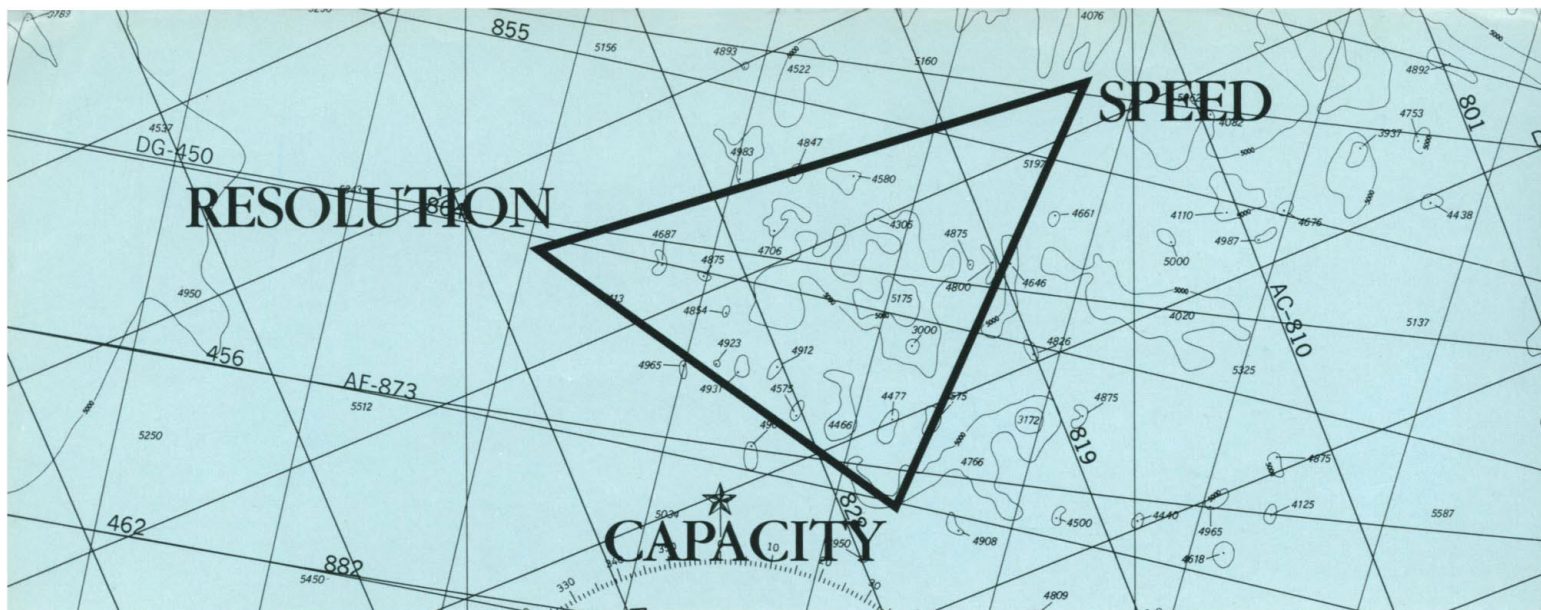
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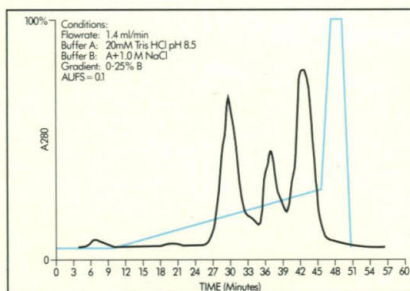
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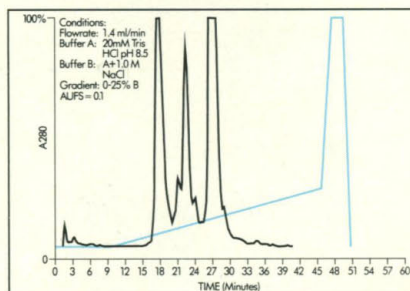
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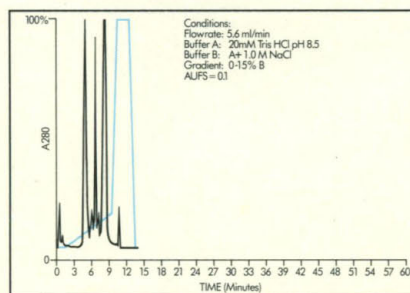
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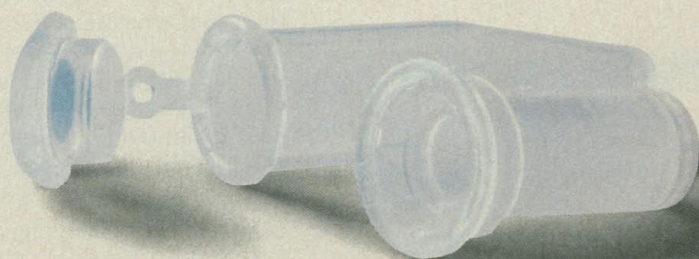
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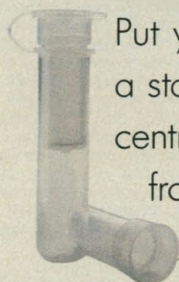
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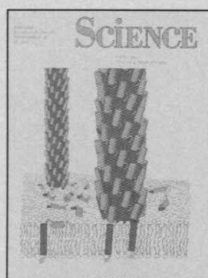
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COVER A drawing of viral particle assembly at a membrane surface. Assembly of the filamentous bacteriophage Pfl takes place by the addition of coat protein subunits to the growing virus cylinder as the DNA extrudes through the host cell membrane without incorporating lipids. The coat protein undergoes a structural transition during this translocation process in which its secondary structure remains essentially unchanged while its tertiary structure changes substantially. See pages 1303 and 1305. [Drawing by L. Makowski]

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This Week in SCIENCE

Arctic ozone depletion

THE catalytic destruction of ozone by chlorine and bromine produces an ozone hole over Antarctica annually; in the past several years, the chemical species, stratospheric conditions, and nature of the interactions required for ozone depletion have been greatly clarified. Is the Arctic stratosphere similarly susceptible to ozone depletion and can any depletion be documented there? Data collected in early 1989 indicate that the distinctive combination of physical, chemical, and dynamical conditions in the stratosphere required for ozone depletion can exist in the Arctic stratosphere (page 1260). Although temperatures over the Arctic are generally warmer than those over Antarctica, most years the Arctic stratosphere becomes cold enough for polar stratospheric clouds to form. Furthermore, polar vortices have formed often over the Arctic, although they are smaller than those that form over Antarctica. The depletion of ozone inside the vortex in 1989 was estimated at 5 to 8%. Brune and colleagues speculate that the extent of ozone depletion over the Arctic will increase in the coming years as chlorine release into the atmosphere continues; they point out that another variable in the ozone-destruction equation is human compliance with international policies to limit halogen release.

Enzyme analysis

THE enzyme adenosine deaminase is found in all mammalian cells and participates in the metabolism of purines. It appears to be crucial for proper immune functioning. In various diseases—including severe combined immunodeficiency disease (SCID), AIDS, anemias, leukemias, and certain lymphomas—this enzyme is defective or nonfunctional. New clues to how this enzyme performs its catalytic functions have been obtained by Wilson *et al.* (page 1278). They studied the three-dimensional crystal structure of adenosine deaminase when it was complexed with a transition-state analog, HDPR, a hydrated form of the

purine ribonucleoside that is the enzyme's ground-state substrate. The enzyme folds into an α/β -barrel motif that has already been characterized in a number of other enzymes; a newly identified zinc atom in the active site plays a pivotal role in enzyme activity, which could explain previously noted associations of reduced immune function with zinc deficiencies. The adenosine deaminase molecules in patients with SCID have various point mutations; from these structural studies it is clear how certain amino acids that are in the active site can have such profound effects on enzyme function.

Neuron-silicon junction

THE goal of developing neural biosensors and various forms of neuroelectronic circuitry is now somewhat closer: a tight electric junction has been successfully formed between a nerve cell and silicon (page 1290). Fromherz *et al.* dissociated individual neurons (called Retzius cells) from segmental ganglions of leeches and connected them to oxidized silicon; the junction behaved like a field-effect transistor. Both spontaneous and induced signals were recorded and were found to resemble typical electric discharges—action potentials—of firing neurons. The attachment of neuron and silicon, which occurs through the neuron's plasma membrane and the gate oxide of the silicon, is intimate, allowing for electric transmission by the junction. If this single-neuron technology can be applied to neurons in networks, it should facilitate analyses of complex neural systems.

Life-history experiments

NORMALLY side-blotched lizards produce an average of 4.6 eggs per clutch, although they can produce as many as 9 eggs at a time. However, if yolk is removed from some developing follicles of lizard ovaries (a procedure that prevents the eggs from developing further), clutches are produced that contain on average just 1 to

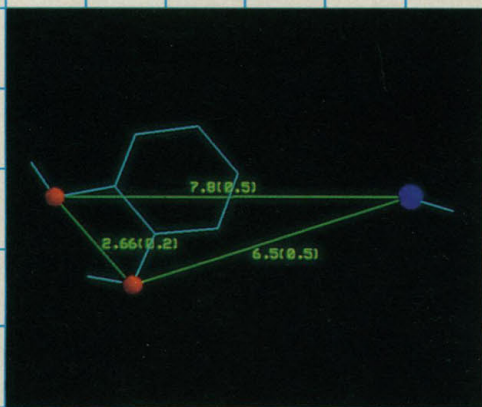
3 eggs (page 1300). Life history theory predicts that a balance of egg size and egg number is needed to keep the total mass of the egg clutch constant, and the remaining eggs in the clutch do grow to larger than normal size. Sinervo and Licht found that some of the over-sized eggs that formed after yolkectomy burst at oviposition or were unable to traverse the oviduct. Eggs that block the oviduct not only fail to hatch and block hatching of other eggs behind them in the oviduct but may also kill the female. Egg size thus appears to be limited also by the diameter of the lizard's pelvic girdle. The maximum lizard successfully attained by eggs in these experiments matched the maximum size of eggs in lizards that can normally lay only 1 egg. The coordinate evolution of clutch mass and egg size and number is likely to have been regulated and constrained by a combination of genetic, physiologic, and biochemical (hormonal) factors.

Brain cells

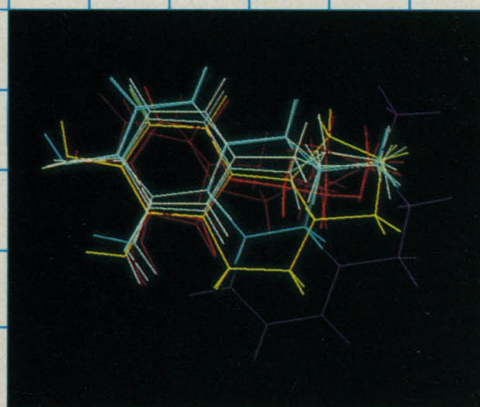
A small subpopulation of brain cells is active both during healthy REM (rapid eye movement) sleep and during episodes of cataplexy. Cataplexy—abrupt loss of muscle tone—is one component of the pathologic condition known as narcolepsy, which also includes sleepiness and other symptoms (page 1315). In brains of narcoleptic dogs, the “cataplexy-on” cells were situated in the ventromedial and caudal portions of the nucleus magnocellularis, which is where suppression of muscle tone during REM sleep is thought to be regulated. Because the cataplexy-on cells showed increased discharge rates in conjunction with cataplexy and REM sleep, Siegel *et al.* propose that these cells may be directly involved in the common feature of these two conditions—loss of muscle tone. The cells were not active when the animals were awake. Most other nearby cells were also active during REM sleep but were inactive during cataplexy; the neurologic underpinnings of cataplexy are thus unlike those of other known conditions of sleep or wakefulness.

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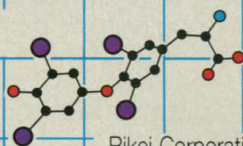
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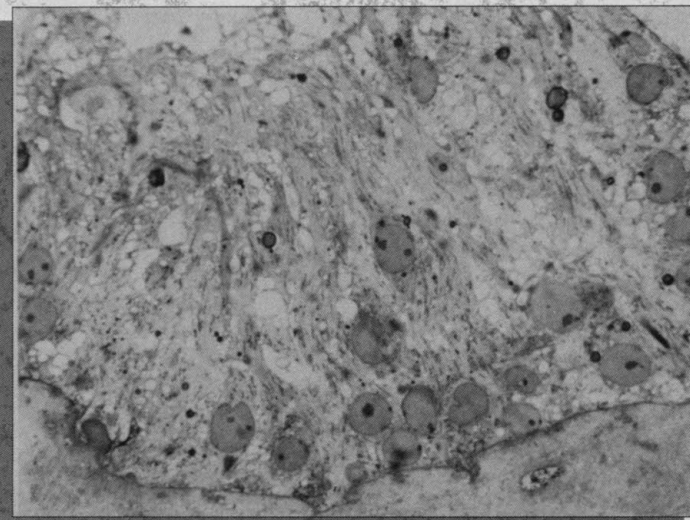
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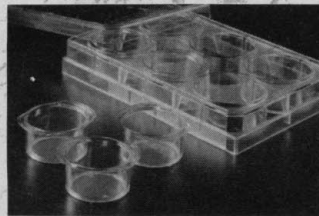


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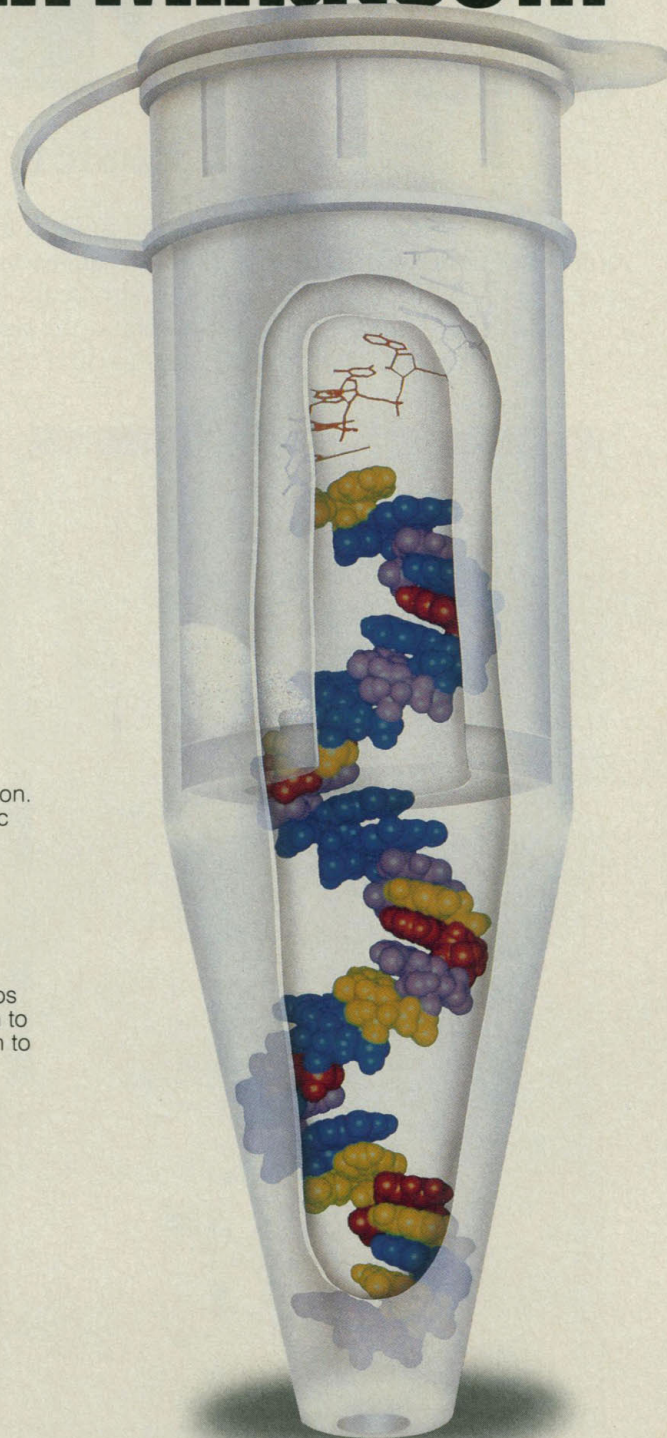


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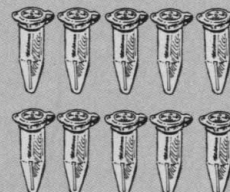


Total RNA Isolation Kit

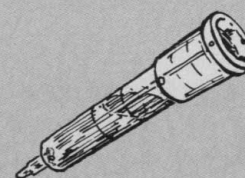
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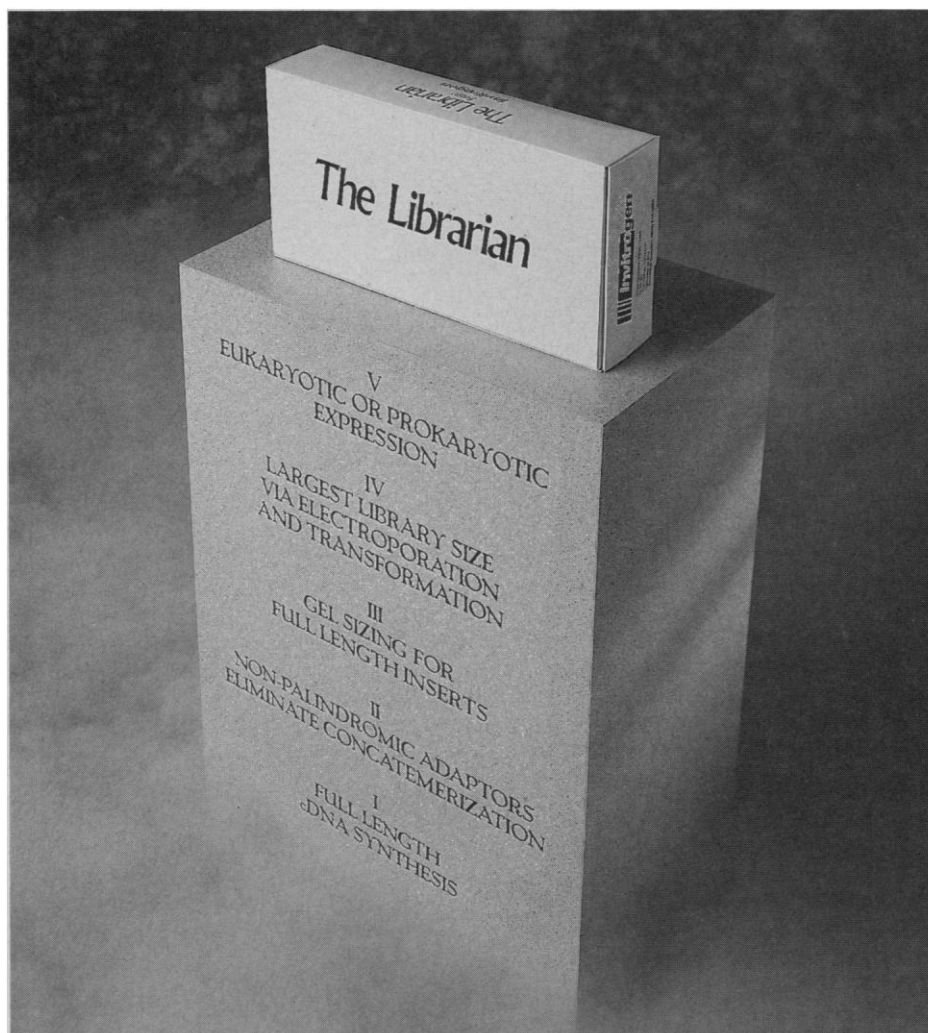
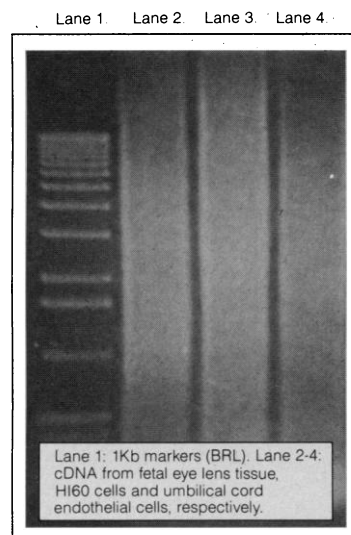
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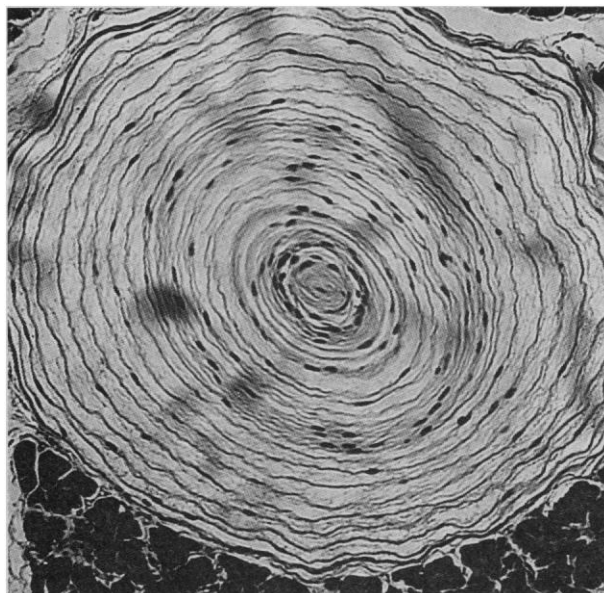
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