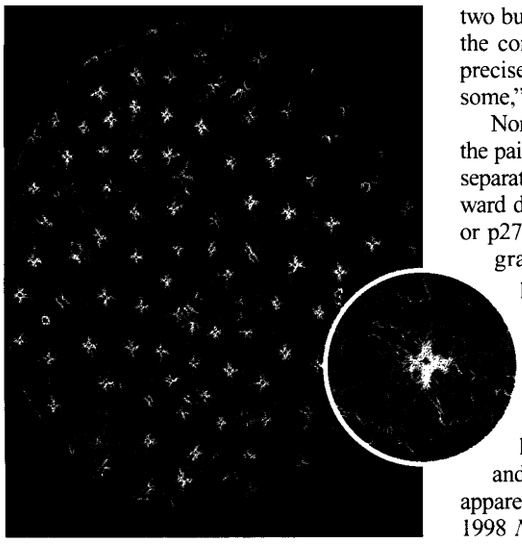


Centrosome galaxy. Cdk2-E causes proliferation of star-shaped centrosomes in an egg extract.

CREDIT: E. HINCHCLIFFE

kick in as the cell cycle progresses.

The first substance the Sluder team tested was the Cdk2–cyclin E (Cdk2-E) complex, reasoning that because the complex is involved in prodding cells to begin making new DNA, it might also regulate the centrosome duplication that seems to happen at about the same time. To test that idea, Sluder's postdoctoral fellow, Edward Hinchcliffe, obtained a specific inhibitor of Cdk2-E activity, a modified version of a frog protein called Xic1, from James Maller at the University of Colorado School of Medicine in Denver.

The team monitored the inhibitor's effects by using time-lapse photography to follow the increase in the numbers of centrosomes over time in their microscope's field of view. They had already learned that, without the inhibitor, three-quarters of the aster-shaped centrosomes replicate three times in a 6-hour period, and most of the rest replicate twice. The inhibitor greatly reduced this centrosome copying; 79% doubled just once and none doubled three times. Conversely, adding extra Cdk2-E overcame this effect, allowing the centrosomes to replicate multiple times. "This is clean evidence that we have one very important set of [proteins] that are essential for [centrosome] replication," says Brinkley.

Meanwhile, at Stanford, Stearns's group had taken a slightly different approach. The researchers had decided to look closely at the Cdk2-E complex after first finding that two naturally occurring Cdk2 inhibitors, proteins called p21 and p27, block centrosome replication in developing frog embryos. But rather than observing the effect of these inhibitors on the overall increase in the numbers of centrosomes, the Stanford team used deconvolution microscopy to watch what happens to the centrioles, the two bundles of short microtubules that form the core of the centrosome. We could "see precisely what's going on inside the centrosome," says Stearns.

Normally, after 1 hour in frog-egg extract, the paired centrioles in each centrosome have separated, presumably taking the first step toward duplication. But in the presence of p21 or p27, Stearns and his Stanford colleagues, graduate student Kathleen Lacey and pathologist Peter Jackson, found that the centrioles stayed put. "We both showed that Cdk2-E is probably the thing that's driving centrosome duplication," Stearns says.

Many questions remain, including how Cdk2-E triggers the duplication and what its molecular partners are, as it apparently doesn't act alone. In the October 1998 *Nature Genetics*, Brinkley and Subrata Sen at the M. D. Anderson Cancer Center in Houston reported that they had cloned a gene that when overexpressed in mouse cells resulted in extra centrosomes. This gene is also overexpressed in cancer patients. It may act in conjunction with Cdk2-E and, when in excess, "lead to a lot of chaos and genetic instability" and eventually, cancer, Brinkley notes. And that, he adds, "was Boveri's original notion."

–ELIZABETH PENNISI

POLYMER ELECTRONICS

Insulator Gives Plastic Transistors a Boost

Anyone who has dropped a laptop computer or mobile phone knows, to their cost, that they are not tough. But the glass and brittle semiconductors that make their displays prone to shattering could one day give way to a material that is cheap, easy to manufacture, and tough—a material pretty much like plastic. Before an all-plastic display makes a commercial debut, however, researchers will have to overcome a major drawback of polymer electronics: Polymer transistors, which would be needed by the thousands in a display, require impractically high voltages to make them work. Now, by simply changing an insulating material in a polymer transistor, a team of IBM researchers reports on page 822 that they have cut the voltage it needs to a level comparable with the amorphous silicon used in today's displays.

"This is excellent work," says plastic transistor pioneer Francis Garnier of the CNRS Laboratory of Molecular Materials in Thiais, France. Says Cambridge University physicist Richard Friend, "[Such] molecular semiconductors have now been built up as very credible materials for technologists."

The team, led by Christos Dimitrakopoulos of IBM's T. J. Watson Research Center in Yorktown Heights, New York, skirted a long-

ScienceScope

Let Them Debate! Like outlaws itching for a showdown with the sheriff, angry French scientists have been gunning for research minister Claude Allègre ever since he proposed controversial reforms of the nation's research agencies last year (*Science*, 23 October 1998, p. 607). Allègre has spurned scientists' demands for a formal national debate on the future of French science. Now, the scientists are plotting the next stage of their insurgency.

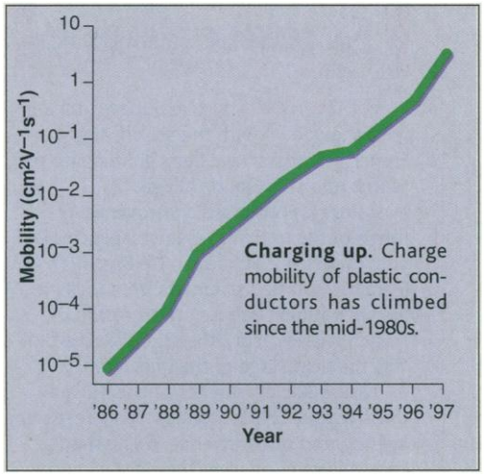
Last week, presidents of the 40 sections within CNRS, France's basic research agency, and other science VIPs issued a communiqué insisting that "the circumstances demand" a national debate. The research ministry's answer came swiftly: *Non*. Instead, the ministry wants to continue ongoing discussions of Allègre's plans within its science agencies. "We don't believe a national debate is the best solution," says the ministry's director-general for research, Vincent Courtillot.

The next move is up to the scientists, who have already shown some fighting spirit. "We all agree changes are necessary, but there is no reason not to [debate]," says neurophysiologist Rose Katz, president of the French biomedical agency INSERM's scientific council. CNRS historian Denis Peschanski vows that his colleagues will organize a national debate—"with the agreement of the minister or without it."

Magnetic Makeover Dutch scientists are turning dreams of upgrading their High Field Magnet Laboratory in Nijmegen into reality. The Dutch Foundation for Fundamental Research on Matter and the University of Nijmegen have signed off on a $23 million plan to refurbish the lab, which probes materials such as superconductors and studies the effects of magnetic fields on living organisms. A new power supply will boost the 20-tesla fields of two existing magnets to 34 and 41 teslas, says lab director Jan-Kees Maan, and the lab will install a new pulsed magnet, capable of producing an 80-tesla field—800,000 times as strong as Earth's magnetic field.

The additions will allow the lab to better compete with facilities in Tallahassee, Florida, and Grenoble, France. Elsewhere on the European magnet front, scientists face a 15 February deadline for commenting on a European Science Foundation report calling for a jointly funded, continent-wide magnet lab that would be home to even more powerful devices.

Contributors: Govert Schilling, Judy Redfearn, Michael Balter, Alexander Hellemans



standing problem in polymer electronics. Polymers suffer from low mobility—essentially the speed at which charges, either electrons or electron gaps called holes, move through the material when a voltage is applied. By tweaking polymers' chemical structure, researchers had managed to improve their mobility by five orders of magnitude—enough to make plastic semiconductors. That development, in turn, opened the way to polymer transistors.

The basic design starts with a substrate carrying a metal electrode called a gate. Over the gate and substrate goes a layer of insulator followed by a layer of organic semiconductor such as pentacene, topped off by two more contacts, one on either side of the buried gate, known as the source and the drain. Normally, a voltage between the source and drain will produce only a trickle of current because charge carriers get caught in "traps," current-impeding locations in the polymer. "We expect that these traps are related to structural defects, such as grain boundaries or dislocations, and to impurities," says Dimitrakopoulos.

Applying a voltage to the gate, however, attracts charge carriers—holes in pentacene's case—from elsewhere in the semiconductor layer into the region above the gate, where they fill up some of the traps, allowing a freer flow of charge carriers from the source to the drain. Although this gate voltage effectively "switches on" the transistor, it still requires voltages in the region of 100 volts at all three electrodes to achieve this result, because the mobility of charge carriers is so low in a polymer. "Such voltages are incompatible with real applications," says Garnier.

So Dimitrakopoulos's team dodged the problem: Instead of trying to improve the mobility of the pentacene semiconductor directly, they sought to fill more traps by changing the insulating layer. "We replaced silicon dioxide, which was the gate insulator used, with an insulator with a much higher dielectric constant," says Dimitrakopoulos. The team used barium zirconate titanate, which has a dielectric constant of 17.3 compared to the 3.9 of silicon dioxide. Dielectric constant is a measure of a material's ability to transmit an electric field. A higher constant will channel more of the gate's electric field to the semiconductor and so pull in many more holes—"enough to fill [all] the trapping states [and leave] extra carriers that are free to travel," says Dimitrakopoulos.

With the new insulator, it took a change in gate voltage of just a few volts, rather than a few hundred volts, to alter the source-drain current by more than five orders of magnitude. The performance of these transistors now rivals that of the amorphous-silicon transistors, the type of low-cost transistor used in active-matrix displays, says Dimitrakopoulos, who adds that his group now hopes to integrate their transistors into similar displays. Friend says such work can only heighten industry's interest. "The level of interest is of an entirely different order than it was 2 years ago."

–ALEXANDER HELLEMANS

Alexander Hellemans is a writer in Naples, Italy.

VIROLOGY

AIDS Virus Traced to Chimp Subspecies

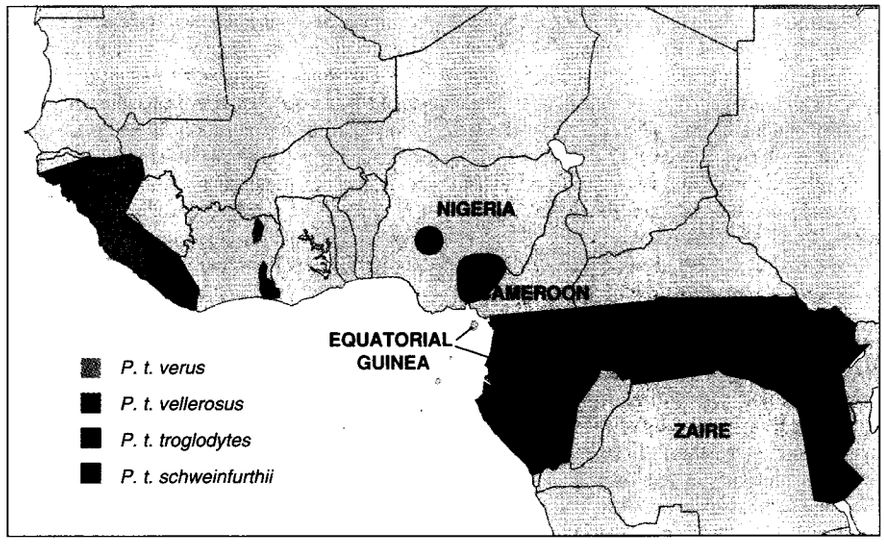
CHICAGO, ILLINOIS—Most AIDS researchers have long believed that HIV-1, the main form of the AIDS virus, jumped from chimpanzees into humans. But there have been scant data to support this thesis, which has allowed theories to flourish ranging from the ridiculous (the government made the virus) to the scientifically implausible (a poliovirus vaccine introduced it) to the highly speculative (an unidentified species is the main host). Now Beatrice Hahn from the University of Alabama, Birmingham, and co-workers have pieced together what is being hailed as the best case yet for the chimpanzee connection.

Hahn's genetic detective work—which she described in the keynote speech here at the opening of the largest annual AIDS conference held in the United States* and is published in this week's issue of *Nature*—indicates that different subspecies of chimps harbor different strains of HIV-like viruses, and that one particular chimp subspecies found in a region that includes Gabon, Cameroon, and Equatorial Guinea is the source of human HIV-1 infections.

That region had been identified before as the likely epicenter of the human disease (*Science*, 15 May 1992, p. 966). But some researchers, including Hahn, doubted that chimps were the original reservoir of HIV-1 because a virus isolated from one chimp bore little resemblance to human strains, and some regions where chimps live do not have HIV-1 epidemics. The new analysis changed her mind: She now argues that some subspecies may not harbor the virus, and others may be infected with a strain that is not as likely to spread epidemically in humans. Furthering the case, Hahn noted in her talk that a French group led by the Pasteur Institute's Françoise Barre-Sinoussi will report later at the meeting that they have found three chimps from Cameroon infected with an HIV-like virus.

Vanessa Hirsch, a primate researcher at the U.S. National Institute of Allergy and Infectious Diseases who, like Hahn, helped establish the link between HIV-2—a much rarer

* Sixth Conference on Retroviruses and Opportunistic Infections, 31 January to 4 February 1999, Chicago, Illinois.



CREDIT: (TOP) IBM; SOURCE (BOTTOM) B. HAHN *ET AL.*

Chimp ranges. All three close relatives of HIV-1 were found in *P. t. troglodytes*.