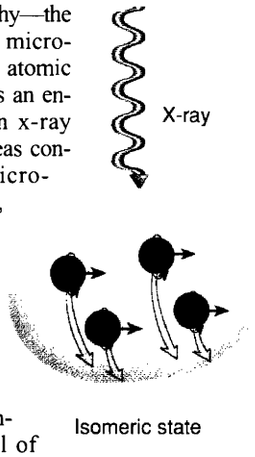
thin, crusty, plastic-encased film of hafnium were not coherent—their waves were not synchronized, which is a hallmark of a true laser. "This is more like a gamma ray flashbulb," remarks laser expert Paul Kepple of the Naval Research Laboratory in Washington, D.C. But he and physicist Carl Collins of the Center for Quantum Electronics at the University of Texas, Dallas, the group's leader, think that if researchers can unravel and control the complicated movement of energy within the nuclei, they could be on their way to the ultimate laser. Says Kepple: "Who knows where it is going to go?"

Even if it falls short of a laser, the phenomenon the researchers have observed, called induced gamma emission, could find plenty of uses. A tabletop gamma machine, with its supershort wavelengths, could push photolithography—the process that traces microcircuit patterns—to atomic dimensions, serve as an energy source for an x-ray laser, or sterilize areas contaminated by microorganisms released, for example, by terrorists. Says Collins: "You could set off something the size of a match head" to do the job. But team member Paul McDaniel of the Air Force Research Laboratory in Albuquerque, New Mexico, says the science is what has him going. "It's the newest physics that I have heard of."



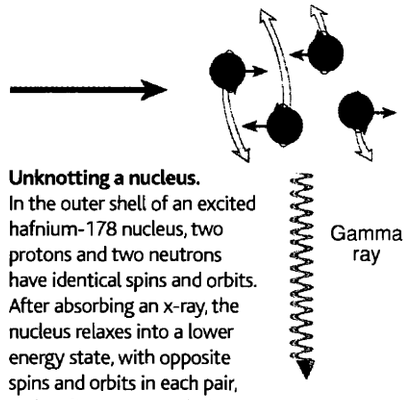
A gamma ray laser would work differently from existing laser types, which all pump electrons in some gaseous, liquid, or solid lasing medium to an excited state and then stimulate them to emit radiation coherently as they relax to their ground state en masse. The only way to get atoms to emit gamma-caliber photons is to achieve the same trick with their *nuclei*, pumping a large population of them into deformed, excited states called isomers and getting them to relax to their normal shapes all at once.

With most nuclei, the gamma-emitting isomers give off their energy too quickly for a large population to develop, no matter how fast more energy is pumped in. But isomers of some isotopes are much longer lived. Collins and colleagues had first managed to coax gamma emission from tantalum-180, a naturally occurring isotope with a proportion of its nuclei fixed in an excited isomeric state. A blast of x-rays triggered the tantalum-180 nuclei to relax into their ground-state arrangement and emit their stored energy.

The photons that emerged from the tantalum had about the same energy as the triggering x-ray photons, but an accelerator-produced isomer of hafnium-178 can emit gamma photons many times more energetic. The isomer normally leaks its energy over a half-life of 31 years. But calculations by Collins's team had shown that ordinary x-rays could discharge that pent-up energy, by triggering the complex nuclear rearrangements needed for the nuclei to relax to their ground states.

Two years ago French researchers reported that they had succeeded in triggering the hafnium-178 emission. They have not provided further details, however, and some researchers are skeptical. Now Collins and his colleagues have tested their own sample of hafnium-178, a waste product of the production of radioisotopes for medicine. Working in Dallas, the researchers aimed a dentist's x-ray machine at the sample and detected an answering pulse of gamma rays, at energies 60 times those of the triggering x-rays.

Now they are preparing follow-on experiments to map out how x-ray energy goes into the hafnium-178 isomers and then triggers the excited nuclei to relax. Collins says it could be just the beginning of a new research area some are calling quantum nucleonics, marked by precise manipulation of nuclear structure. Not to mention a step toward the ultimate laser beam.



Unknotting a nucleus. In the outer shell of an excited hafnium-178 nucleus, two protons and two neutrons have identical spins and orbits. After absorbing an x-ray, the nucleus relaxes into a lower energy state, with opposite spins and orbits in each pair, and emits a gamma photon.

—IVAN AMATO

Ivan Amato is a science reporter for National Public Radio and the author of *Stuff*.

CELL BIOLOGY

Trigger for Centrosome Replication Found

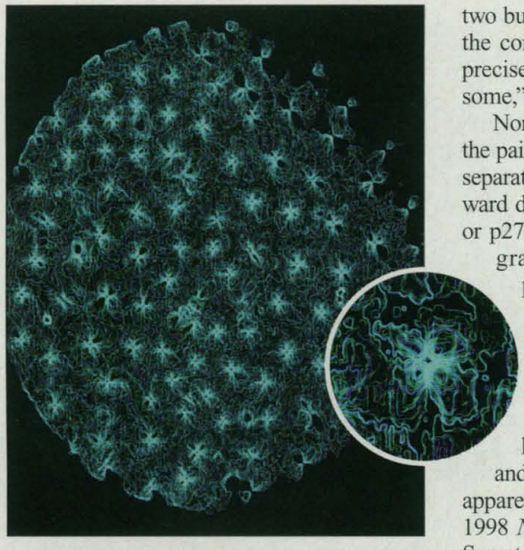
At the turn of the century, a European biologist named Theodor Boveri suggested that a small body near the cell nucleus might be a key to cancer. Called the centrosome, it replicates before cell division and then, via the protein cables that radiate from it, helps pull the duplicated chromosomes apart into the daughter cells. Boveri proposed that errors in this process could derange cells. Over the following decades, however, his idea got lost, as researchers concentrated on understanding the specific gene malfunctions that lead to cancer. New findings, some of which are described in this issue, may now help revive interest in Boveri's notion.

On page 851, Greenfield Sluder and his colleagues at the University of Massachusetts Medical School in Worcester report that they have identified a trigger that helps tell the dividing cell to copy its centrosome. It's a new role for a familiar character: an enzyme called Cdk2, already known to help drive cells through the division cycle when activated by another protein, known as cyclin E. Later this month, another team, led by cell biologist Tim Stearns of Stanford University, will publish similar findings in the *Proceedings of the National Academy of Sciences*. "It's a key discovery," says William Brinkley, a cell biologist at Baylor College of Medicine in Houston. "The biochemistry of how centrosomes replicate is beginning to come into view."

By helping explain how the cell cycle and centrosome replication are linked so that the centrosome is copied just once and at just the right time, the finding may help researchers figure out how the replication might go awry, with potentially disastrous consequences. Because "the balance in the genome is maintained through the [centrosomal] machinery," as Robert Palazzo, a cell biologist at the University of Kansas, Lawrence, puts it, a centrosome that replicates too often, or fails to replicate at all, can leave the daughter cells with abnormal chromosomal compositions. They might lose a tumor suppressor gene, for example, or acquire extra copies of growth-promoting oncogenes—conditions that predispose cells to cancer. Indeed, researchers have recently found that cancer cells, even in their earliest stages, have abnormal numbers of centrosomes.

The two teams succeeded in identifying a trigger for centrosome division because they came up with assays for studying centrosome replication outside living cells. "The ability to assay that is a significant achievement that should not be underestimated," comments Palazzo.

Both assays use extracts of frog eggs. In a test tube, the DNA and centrosomes in these extracts can still replicate just as they do in intact eggs. Normally, both activities are coordinated so that they occur at roughly the same time in the cell cycle. But Sluder's group found that a chemical inhibitor blocks DNA synthesis in the egg extracts yet somehow does not affect centrosome replication. With the cell cycle thus stalled out, the researchers could monitor how various substances affect centrosome duplication without worrying about the normal blocks to the copying that



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