the edge closest to the tracheal cells. So, Trimmer suggests, when the lantern is off, these mitochondria "are a barrier" that soaks up oxygen before it reaches luciferin.

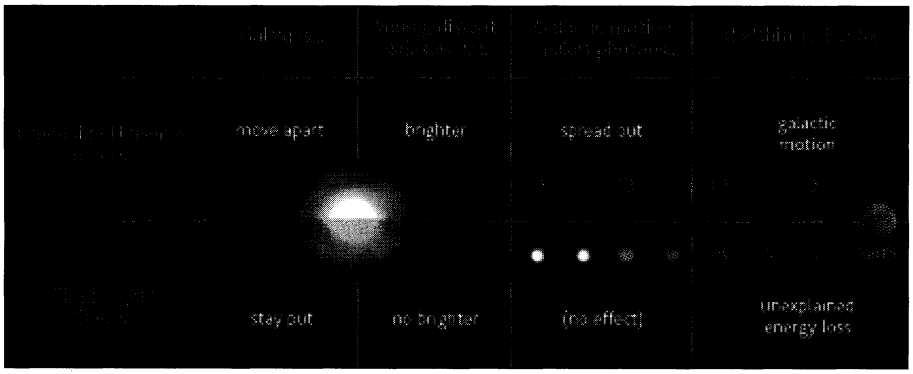
When it's time to flash, however, the NO concentration increases in the air-duct cells and diffuses over the mitochondria, briefly shutting down this oxygen barrier. In response, the oxygen concentration rises in the photocytes, setting off the light. Then "the lantern in and of itself turns off the NO reaction," says William Sessa, a pharmacologist at Yale University, because, as other research has shown, light inhibits NO.

Trimmer has yet to prove that this is how the lantern works. But to Gerd Bicker, an NO specialist at the Hannover School of Veterinary Medicine in Germany, the work stands on its own merits. "I am very pleased," he says, "that NO appears to be involved in such an esthetical aspect of cellular communication." **–ELIZABETH PENNISI**

ASTROPHYSICS

'Tired-Light' Hypothesis Gets Re-Tired

The "tired-light" hypothesis, mainstay of a dwindling band of contrarians who deny the big bang and its corollary, the expanding universe, has suffered a one-two punch. Observations of supernovae and of galaxies provide the best direct evidence that the universe is truly expanding and promise to shed light on the evolution of galaxies to boot.



Beyond the fringe. "Tired light"—a radical alternative to the standard expanding-universe model of the cosmos—has just failed two crucial tests.

"The expansion is real. It's not due to an unknown physical process. That is the conclusion," says Allan Sandage, an astrophysicist at the Carnegie Observatories in Pasadena, California, and leader of the galaxy study.

It's a conclusion that most astronomers reached long ago. In 1929, Edwin Hubble announced that light from distant galaxies is redder than light from nearby ones. Hubble and others took the redshifts as evidence that the universe is expanding, causing distant galaxies to speed away faster than nearby ones. To an observer on Earth, they reasoned, this would appear to stretch the wavelength of their light, just as the sound of a police-car siren seems to drop in frequency as it speeds away. However, within a few months of the publication of Hubble's paper, astrophysicist Franz Zwicky came up with an alternative explanation: that galaxies' light reddens because it loses energy as it passes through space. In Zwicky's tired-light scenario, the universe doesn't expand at all. Distant galaxies are red not because they are moving, but because their light has traveled farther and gotten pooped along the way.

When experimenters first measured the cosmic microwave background more than 30 years ago, they found that the radiation was too dim to be explained by Zwicky's hypothesis. That realization relegated "tired light" firmly to the fringe of physics, but scientists still sought more direct proofs of the expansion of the cosmos.

Two new papers provide the best direct evidence yet. The first, slated to appear in *Astrophysical Journal*, measures the brightening and dimming of a certain type of supernova. Thanks to Einstein's theory of relativity, if distant supernovae are speeding away from us, they will appear to flare and fade at a more leisurely pace than close-by ones. A team of scientists led by Gerson Goldhaber of the Lawrence Berkeley National Laboratory (LBNL) in Berkeley, California, has shown that this is, indeed, the case with 42 recently analyzed supernovae. "It's such a clean-looking curve," says Saul Perlmutter, a member of the LBNL team. "It's very unambiguous."

In the second study, Sandage and Lori Lubin of Johns Hopkins University in Baltimore analyzed space-based measurements of the surface brightness of galaxies. Both the standard expanding universe and the tired-light theory, they realized, agree that red-shifted light should make distant galaxies look dimmer than they really are. In an expanding universe, however, time dilation and other relativistic distortions will also dim distant galaxies, making them appear much fainter than tired-light theory dictates. What's more, young stars—and thus young galaxies—tend to be considerably brighter than old ones. When that extra brightness is taken into account, the observations match expanding-universe predictions, as Lubin and Sandage will report in *Astronomical Journal*. For the tired-light theory to be correct, young galaxies would have to be dimmer, rather than brighter, than old ones. "There's no way to explain that," says Lubin.

Although not surprising in themselves, the results are useful for "tidying things up in our cosmology," says Michael Pahre, an astronomer at the Harvard-Smithsonian Center for Astrophysics in Cambridge, Massachusetts, who performed a similar surface-brightness experiment in the mid-1990s. By comparing the expanding-universe theory's predictions with observed values of the surface brightness of distant galaxies, scientists can work backward and figure out how much brighter those galaxies must have been earlier in the history of the universe.

Even so, researchers doubt whether the results will convert tired-light diehards. "I don't think it's possible to convince people who are holding on to tired light," says Ned Wright, an astrophysicist at the University of California, Los Angeles. "I would say it is more a problem for a psychological journal than for *Science*." **–CHARLES SEIFE**

PARTICLE PHYSICS

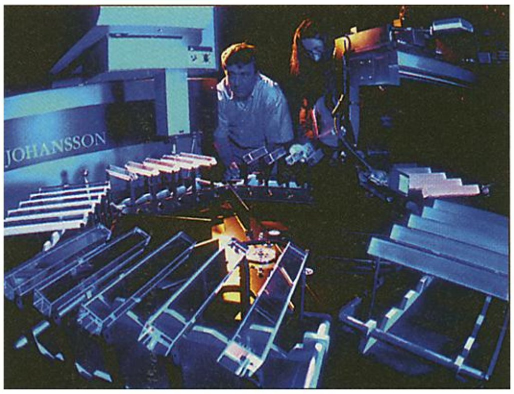
Russian Turmoil Rattles CERN

MOSCOW—Discord over KGB-style rules that require Russian academics to report contacts with foreign scientists—as well as a management change at a key institute—are raising caution flags about Russia's collaboration on a major international particle physics project being built at CERN, the European laboratory for particle physics near Geneva.

Last month, the Russian Academy of Sciences issued a directive ordering its 55,000 researchers to report any international activities and contacts to the academy's governing presidium. Some observers see the directive as a benign effort to protect researchers from inadvertently divulging classified information. Others, however, view it as a thinly veiled attempt to allow the KGB's successor agency, the Federal Security Service, to exert more control over the scientific community (*Science*, 8 June, p. 1810). Now, similar restrictions are roiling the waters at nonacademy institutes.

At issue are rules requiring all institutes overseen by the Ministry of Atomic Energy

(Minatom) to inform it about visits from foreign researchers no less than 45 days prior to their arrival. Although the rules were put in place 3 years ago, Minatom had enforced them only at sensitive facilities such as the nuclear weapons design centers in Sarov and Snezhinsk. Now, purely civilian outfits like the Institute for Theoretical and Experimental Physics (ITEP) in Moscow are under pressure to toe the line, says ITEP physicist Boris Ioffe.



Imperiled? Russia's contribution to the LHC includes top minds and materials such as these lead tungstate crystals.

In the increasingly tense atmosphere last week ITEP director Mikhail Danilov, a key figure in Russia's delegation to CERN, tendered his resignation. When contacted by *Science*, Danilov declined to comment. However, some ITEP staff members assert that the resignation stems from Danilov's frustration over Minatom's recent restrictions on foreign contacts. Besides bleeding off some of Danilov's authority, the rules could put a chill on foreign collaboration, Ioffe predicts. Others insist that the administrative burden of the directorship, rather than any increased pressure from Minatom bosses, spurred Danilov's resignation. Either way, Ioffe says, "the situation is very difficult."

That's alarming news to CERN, which is relying heavily on Russian researchers to help build the $1.5 billion Large Hadron Collider (LHC), a machine that will explore fundamental questions such as why particles have mass. Roughly 600 Russian physicists are working on the LHC, providing an invaluable contribution to the collider and its associated experiments (*Science*, 13 October 2000, p. 250). About one-quarter of the Russian contingent comes from ITEP. "If, as I suspect, the security pressure will increase," Ioffe warns, "then ITEP's contacts with CERN will shrink, and consequently ITEP's participation in CERN projects will shrink." Others have a less dire take. "I don't see any danger for the time being," says ITEP deputy director Vitali Kaftanov. The most important wildcard, he argues, is whether Danilov's successor will be able to convince Minatom that the CERN collaboration is worth supporting.

—VLADIMIR POKROVSKY AND ANDREY ALLAKHVERDOV

Vladimir Pokrovsky and Andrey Allakhverdov are writers in Moscow. With reporting by Richard Stone.

CELL CYCLE RESEARCH

DNA: Once Copied, Thrice Blocked

When it comes to cell division, nature demands close tolerances. A dividing cell must keep a tight rein on DNA replication to ensure that each daughter receives exactly one copy. Indeed, failure of that control and the genetic instability that results can lead to cell death or even cancer. Although more than a decade ago cell biologists identified the master protein that guarantees that DNA replication occurs only once, they don't understand how it exerts its power. Now, part of that puzzle has been solved.

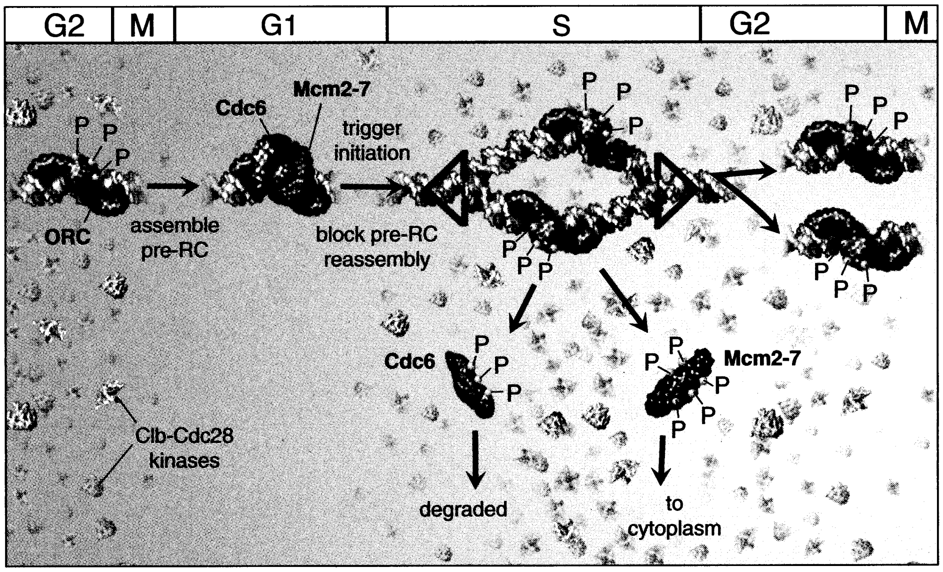
In the 28 June issue of *Nature*, a team led by Joachim Li of the University of California, San Francisco, reports that three separate pathways, all under the control of the master coordinator, a protein kinase known as Clb-Cdc28, must work together to prevent a second round of DNA synthesis from getting under way before the cell divides.

The current findings are an outgrowth of work done over the last 5 years indicating that Clb-Cdc28 does double duty in controlling DNA replication. When cell division begins, a group of proteins called the prereplicative complex (pre-RC) comes together on each of the many origins of replication where DNA synthesis begins. Once assembled, the pre-RCs stand poised to initiate replication but don't do so until the cell suddenly activates the Clb-Cdc28 kinase. That's when the enzyme shows its versatility.

It first triggers replication by the assembled pre-RCs, but once they fall off the origins of replication, it inhibits assembly of new pre-RCs that would reinitiate DNA synthesis. As a result, Clb-Cdc28 triggers one and only one round of replication. Then, once the cell divides, Clb-Cdc28 shuts off and new pre-RCs can assemble for the next round of cell division.

Although the details of how Clb-Cdc28 prevents reinitiation remain sketchy, researchers over the past few years have found several potential targets through which it might prevent reassembly. As a kinase, Clb-Cdc28 has the ability to add phosphate groups to other proteins, including three components of the pre-RC. Two of these are so-called initiator proteins: Cdc6, which is targeted for degradation when phosphorylated by Clb-Cdc28, and the protein complex Mcm2-7, which is kicked out of the nucleus when thus modified. The origin recognition complex (ORC), a group of proteins that is also part of the pre-RC, harbors sequences that look like Clb-Cdc28 phosphorylation sites, but researchers haven't proved that. They have also had trouble proving that the three targets help prevent reinitiation, because separately rendering each of them immune to Clb-Cdc28's influence doesn't remove the block. That led them to postulate that all the proteins, and perhaps others, have to work together to prevent reinitiation.

Now, Li's team has provided the experimental proof for the cooperation by making yeast cells in which all three potential targets



Safeguard. Activation of the Clb-Cdc28 protein kinase triggers DNA replication (S) and acts through three targets (ORC, Cdc6, and Mcm2-7) to prevent a second round of replication. Inactivation of the kinase after mitosis (M) allows a new round of replication.

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