

MEXICO

Student Strike Engulfs Research Activities

A student strike that has gripped Mexico's main university for 6 months has now spread to the school's research institutions. Faculty members have already been hampered by months of delays at student-controlled checkpoints, thefts of equipment, and other hassles, and last week some scientists spent hours negotiating for the right to keep their labs open. "The damage ... will be difficult to repair," says Jaime Urrutia, director of the Institute of Geophysics.



Striking out. UNAM students have taken their protest from the streets to labs on campus.

The protests at the Mexico City campus of UNAM—the 260,000-student National Autonomous University of Mexico—began on 20 April when student activists protested a proposed hike in tuition from pennies to about \$150 a year. In June the university abandoned that plan, but the strike has continued, with students now demanding an end to all fees, looser admissions and graduation standards, and much more power on UNAM's governing council. More turmoil could lie ahead if university workers follow through on a threat to strike for higher wages. The school's 2000 researchers publish about half the papers by Mexico's scientists, and hundreds of faculty members have signed a letter asking the government to enforce the law and restore order. But Mexican President Ernesto Zedillo so far has refused to intervene in apparent fear of stirring public opposition in an election year.

Although students have occupied schools and teaching buildings for months, it was

not until 18 October that they began to invade some of the 24 research institutes and centers, shutting down parts of the geography, geology, and geophysics buildings. Physicists and applied mathematicians convinced the activists to keep their buildings open, however, and several geophysicists told *Science* by e-mail that their colleagues had argued successfully that work such as monitoring of the nearby Popocatepetl volcano should go on. The geosciences, says Urrutia, "are particularly important because of the [recent] earthquakes, eruptive activity, and flooding of the past months."

Even though most research labs are still functioning, scientists say the prolonged shutdown of much of the campus has impeded their work. "The major inconvenience [for us] is traffic and communication problems," says Fernando Lopez-Casillas of the Institute of Cell Physiology. Each day researchers must pass through student-controlled gates, where they face verbal harassment. The strikers have also made it difficult to leave campus with equipment, including new seismographs for monitoring Popo. Overnight deliveries can only be picked up off-campus. The strikes have also touched off a wave of vandalism and robberies, including reported thefts of computers and several vehicles used by scientists.

An open letter from UNAM faculty and many international colleagues calls on the Mexican government to impose the "rule of law" (www.ibt.unam.mx/sos). The letter says that complying with strikers' demands, coupled with proposed cuts in UNAM's budget, "would leave us with a totally devalued institution, putting at grave risk one of the most ambitious and successful public and national university projects in Latin America." But Zedillo, according to Jose Antonio Zabalgoitia, a spokesperson at the Mexican embassy in Washington, D.C., "will not order any police or public force to drive the strikers out without strong evidence from the university community itself that they are not supporting the strikers and they want the strike over." Some scientists say they may have to hunker down until the political climate is more favorable: "We hope the problem will clear up once the primaries are over" next month, notes UNAM seismologist Cinna Lomnitz.

Meanwhile, Lopez-Casillas says he plans to take a computer home to write papers on his work characterizing the transforming growth factor- β receptor if his institute is closed. But labs working with higher animals, such as a colleague's highly regarded

Tucson, discovered that a sample of Dorn's that he was processing contained coal and charcoal grains of vastly different ages. Those variations, he and co-authors later wrote in *Science*, made the dates obtained by the technique "ambiguous" (*Science*, 26 June 1998, p. 2132). They also noted that they were unable to find the grains in samples that were not prepared in Dorn's lab. Beck, geochemist Wallace Broecker of Columbia University's Lamont-Doherty Earth Observatory in Palisades, New York, and other researchers co-authored a paper that was eventually submitted to *Science*, which published it last June as a Technical Comment accompanied by a response from Dorn.

Although the authors did not accuse Dorn of misconduct, several shared their findings with officials at ASU and the National Science Foundation (NSF), which had funded some of Dorn's work. Both organizations began investigations of whether Dorn had manipulated the ages of his samples by adding the carbon grains.

This month ASU and NSF cleared Dorn. A faculty panel established by ASU concluded that "the evidence did not support allegations that Dorn added coal or charcoal to rock varnish samples" and that studies showed the materials occurred together naturally. In June, even before the finding was released, however, Dorn moved to file suit against the authors of the *Science* paper, charging that their statements were "published with an 'evil heart.'" His amended complaint, filed on 5 October in Maricopa County Superior Court, cites remarks attributed to Beck and Broecker by the *Arizona Daily Star*, and letters from Broecker to ASU and NSF, that deal with the feasibility of doctoring samples. In the complaint, Dorn says such remarks "clearly implied professional misconduct" and "seriously damaged" his ability to win grants, although the suit claims no specific amount for damages. In addition to Beck and Broecker, Dorn is suing Douglas Donahue, A.J.T. Jull, and George Burr of the University of Arizona's AMS Laboratory; Broecker's employer, Columbia University; linguist and rock art researcher Ekkehart Malotki of Northern Arizona University in Flagstaff; and Georges Bonani and Irka Hajdas of the Swiss Federal Institute of Technology in Zurich.

Lawyers say Dorn's case may rest on whether he can show that the authors went beyond normal academic discourse in criticizing him. Gilbert Whittemore, whose firm, Stalter & Kennedy in Boston, is not involved in the case, says the possibility of such litigation could prompt researchers to avoid future controversies. "Scientific disputes normally get worked out by a rip-roaring debate in the literature," he says, not in the courtroom.

—DAVID MALAKOFF

research using monkeys to study sensory perception, "would probably be totally destroyed" if the strike encompassed them, he says. Adds Lourival Possani, a biochemist at the biotechnology institute in Cuernavaca, 100 kilometers from Mexico City, "If they shut down the research, it's going to be a disaster for the entire country."—**JOCELYN KAISER**

MOLECULAR BIOLOGY

Candidate 'Gene Silencers' Found

Sometimes genes don't add up. About a decade ago, researchers added additional copies of pigment genes to petunias, hoping to darken their purple flowers. Instead, the petals turned white. Biologists now know that in many organisms, including plants, worms, and flies, adding an extra dose of a gene can have the paradoxical effect of slashing that gene's expression. This phenomenon, which goes by the name posttranscriptional gene silencing (PTGS) and helps organisms defend themselves against viral and other foreign nucleic acids, occurs because the added gene somehow causes destruction of the messenger RNA (mRNA) made by both it and the corresponding cellular gene. As a result, production of the gene's protein product shuts down. Now, researchers may have found the tracking system that homes in on the mRNA and triggers its destruction.

Because gene silencing targets specific mRNAs, many people have thought that so-called antisense RNA—RNA with a nucleotide sequence complementary to the gene's mRNA—might be involved, possibly as a tag that marks the mRNA for degradation. They have been unable to identify those antisense RNAs or any other nucleic acid involved in silencing, however. But on page 950, molecular geneticists Andrew Hamilton and David Baulcombe of the John Innes Centre in Norwich, U.K., report that they have come up with a likely prospect: short RNA snippets, 25 nucleotides long, that match the gene being silenced.

"This could be just what we're looking for. It's the first good candidate for RNA molecules that have a role in PTGS," says Richard Jorgensen, a molecular geneticist at the University of Arizona, Tucson. The work should "lead to a much more mechanistic understanding of the process than we currently have."

Baulcombe and Hamilton, a postdoc in Baulcombe's lab, suspected that previous workers had failed to find antisense RNAs in silencing because the molecules were so small that they were running through the analytical gels too quickly for researchers to detect them. For the new search, Hamilton first added a gene that encodes a plant enzyme, called ACO, to each of five tomato plant lines. In two of them, the added gene led to the silencing of the endogenous ACO gene as indicated by the disappearance of its RNA.

The researchers extracted nucleic acids from the plant leaves, enriched the cellular mixtures for low-molecular-weight molecules, and separated the components using a gel system designed to retain small molecules. Hamilton then probed the nucleic acids with a piece of radioactive RNA that specifically binds to antisense ACO sequences. This probe picked up a 25-nucleotide molecule from the two lines where the gene was silenced but not from the three others. The researchers proved that the molecule was in fact RNA by showing that it disappeared from samples subjected to enzymes and chemicals that destroy RNA but not DNA.

To see whether similar RNAs would turn up in other silencing examples, Hamilton added a nucleic acid containing green fluorescent protein sequences to a leaf of a tobacco-related plant that already had a gene for GFP engineered into all its cells. A gene introduced in one place in an organism can silence the corresponding RNA at distant locations, and, in keeping with that, by several weeks later the GFP flu-

orescence had disappeared throughout the plant. Again, the team detected 25-nucleotide GFP antisense RNA in tissues exhibiting PTGS but not in control plants. The researchers analyzed several other examples of PTGS and in all cases they found a 25-nucleotide antisense RNA specific for the silenced gene.

The findings don't distinguish whether these antisense molecules cause silencing or are byproducts of it. If they're the cause, they may be made by an enzyme called RNA-dependent RNA polymerase (RdRP), which copies one RNA from another, creating antisense fragments. Researchers note, for example, that RdRP levels in plant cells rise upon viral infection, when gene silencing takes place. Alternatively, the 25-nucleotide RNAs may be debris left by an enzyme that chews RNA down to precisely that size. Either way, the existence of such uniform-sized RNAs

provides insight into silencing, says Phillip Sharp, a biochemist at the Massachusetts Institute of Technology: "That's really remarkable. ... There's a very precise biochemical mechanism in there."

Besides pinning down what the RNAs are doing and how they are made, researchers would like to use them to enter the natural world of PTGS. Beyond acting as a defense against foreign nucleic acids, the normal role of PTGS is largely a mystery. "Can we find these 25-nucleotide RNAs in plants that don't contain any foreign DNA at all?" asks Baulcombe. "If so, what are they specific for? That will give us ideas about the processes they control."

And of course, scientists wonder whether the findings in plants apply to other organisms. "I can guarantee there will be a lot of flies and worms ground up" to look for a small RNA, Sharp says.

—**EVELYN STRAUSS**

NEUROBIOLOGY

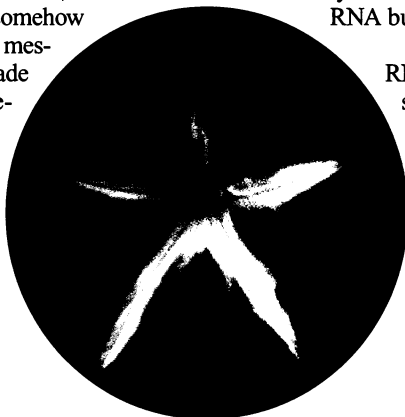
Fetal Cells Help Parkinson's Patients

MIAMI BEACH, FLORIDA—A controversial therapy that involves injecting fetal cells into the brains of Parkinson's patients can slow down the progression of the disease, according to the first double-blind, placebo-controlled clinical study of the procedure. The study, presented here on 24 October at the Society for Neuroscience's annual meeting, shows that the fetal cells can produce a critical neurotransmitter, reducing patients' tremors and paralysis.

Parkinson's disease is marked by the death of brain cells that make the neurotransmitter dopamine. Since the 1980s, researchers have been developing a technique to substitute those brain cells with fetal cells destined to produce dopamine. In 1994, a team led by Curt Freed of the University of Colorado, Denver, received the first grant from the National Institutes of Health for a double-blind, placebo-controlled study of fetal cell transplants in human patients.

Forty patients with advanced Parkinson's disease underwent an operation in which a long needle was inserted through the forehead in four places, under local anesthesia. In half of the patients, the needles delivered small amounts of brain tissue—derived from four 7- to 8-week-old embryos—to the putamen, one of the brain areas affected by Parkinson's. The other patients constituted a control group. For them, the operation was a sham; nothing was injected into their brains.

One year after the operation, the control group hadn't improved. But the fetal tissue seemed to have taken hold in the patients who received a transplant: Positron emission to-



Quiet zone. Silenced pigment genes created the white stripes of this petunia.

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