tough question: how to bring the school bus–sized UARS back to Earth.

Launched in 1991, UARS is still beaming data from five of its 10 instruments that are monitoring global warming factors, such as water vapor and solar radiation, as well as chemicals, such as chlorine, that destroy stratospheric ozone. The observations have already revealed a mysterious rise in stratospheric water vapor having climate implications and confirmed the peaking of ozone-destroying chemicals due to an international agreement. Although the satellite is well past its 3-year design lifetime, project scientists had hoped to keep it operating until this fall, when the European Space Agency had planned to launch Envisat, a sophisticated environmental monitoring satellite. That would have provided some continuity of data. But the launch has been delayed because of the recent failure of an Ariane rocket.

NASA officials now intend to shut off UARS's instruments next week. "Giving up that overlap is difficult," says Anne Douglass, deputy project scientist. "I'm shocked," says Paul Crutzen of the Max Planck Institute for Chemistry in Mainz, Germany, who shared the Nobel Prize in chemistry for discovering the ozone threat. "It would be a tremendous loss."

NASA also must decide how to dispose of the 7-ton satellite. Most large satellites—such as the Mir space station—are designed so that they can be guided into the Pacific Ocean. But UARS was built in an era when engineers envisioned the space shuttle routinely orbiting and returning scientific spacecraft, and it lacks the thrust capacity to be placed on a path for controlled reentry. The shuttle is now busy building the international space station, however, and it may be tough to reserve one to reclaim a defunct satellite as well as find the $50 million needed for such a mission.

Left on its own, UARS would remain aloft for another 20 years. But a slow decay of its orbit would increase the chances that it would break into large chunks containing toxic batteries and fuel. Alternatively, NASA could adjust the orbit of the spacecraft in the coming year for the best possible flight path and vent the toxic fuel, but a truly controlled reentry is not possible. "There's no guarantee where it would come down," says Ondrus.

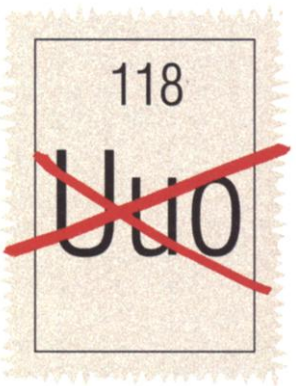
The pending shutdown of UARS "puts a downer on our [anniversary] party," says Douglass. "But we're still going ahead. This was a successful mission, and we have a lot to celebrate." **–ANDREW LAWLER**

With reporting by Richard A. Kerr.

HEAVY-ION PHYSICS

Berkeley Crew Unbags Element 118

The superheavy element 118 just displayed an exotic property that nobody predicted: the ability to vanish into thin air. Physicists who thought they had created the most massive chemical element have retracted their claim in a short statement submitted to *Physical Review Letters*.



Two years ago, scientists at Lawrence Berkeley National Laboratory in California presented evidence that they had bagged element 118 along with its slightly lighter cousin, element 116 (*Science*, 11 June 1999, p. 1751). The news came as a shock to many scientists in the field, who thought that the method of the Berkeley team—gently colliding krypton nuclei with lead ones in the hopes that the two would fuse—had already been exhausted. "I was really surprised in May of '99," says Sigurd Hofmann, a nuclear physicist at the Institute for Heavy Ion Research (GSI) in Darmstadt, Germany. "If we had believed in fusion to make element 118, we certainly would have tried it here earlier." But in the face of the experimental data—three chains of alpha-particle decays that seemed to indicate the existence of a new superheavy element—teams across the world attempted to replicate the results.

Those attempts, at GSI, the GANIL heavy-ion research lab in France, and the Institute of Physical and Chemical Research (RIKEN) in Japan, all came to naught. But the extreme rarity of the new nuclei left it possible that a slight difference in the experimental setup or even a statistical fluke could be responsible for the failures. "Our experiment really did not disprove Berkeley's detection. There's a relatively high probability that the other experiments would see nothing," says Hofmann. So Berkeley tried, last year and this year, to repeat their own experiment.

They failed. In the wake of that failure, Berkeley researchers went back and reanalyzed their original data. "Those analyses showed that the chains reported are not there," says Kenneth Gregorich, a member of the Berkeley team. Gregorich has little idea what caused the false readings. "One of the possibilities is an analysis problem," he

ScienceScope

Indian Trial Troubles Hopkins Still reeling from a government-ordered shutdown of clinical research on its Baltimore, Maryland, campus (*Science*, 27 July, p. 587), Johns Hopkins University has run into a new furor over a project in southern India. The university announced this week that it has "directed" a faculty member "to cease all activities related to" research on an anticancer drug, after learning from news media of "serious allegations about the conduct of" a clinical trial last year at the Regional Cancer Center (RCC) in the state of Kerala.

According to media reports, RCC radiobiologist R. V. Bhattathiri raised questions about a trial led by Hopkins biologist Ru Chih Huang and RCC director Krishnan Nausing that is testing the use of tetramethyl NGDA to treat oral cancer. Bhattathiri told *Science* that he had alleged that 25 patients did not give proper informed consent, did not receive timely standard therapy, and were exposed to a potentially toxic substance. Indian officials are investigating.

Hopkins learned of the trial in March—and of the allegations on 16 July. It says its researcher reported that Indian authorities had approved the trial and that patients gave informed consent. But so far the school has found no record that the trial was approved by Hopkins officials or by the university's Institutional Review Board, which reviews clinical trials. It's not known whether the project received U.S. funding. Hopkins has appointed a three-member panel of experts "to develop the facts."

SOLEIL Protestors Prevail The French government has backed away from plans to privatize a new materials research center after protests from scientists. Earlier this year, the researchers briefly shut down two instruments to dramatize their opposition to a plan to operate the new SOLEIL synchrotron as a private nonprofit (*Science*, 23 March, p. 2293). The plan made it easier for other nations to participate in the project, but harder for the French scientists to move between jobs at government research centers.

Under a deal reached last week, the researchers—known as "Lurons" because they work at the LURE research center in Orsay—can choose between working for a public or private employer. Either way, most of LURE's 280 staff members are expected to join the SOLEIL's 350-strong payroll by the time the machine starts operations in 2005.

Contributors: Pallava Bagla and Barbara Casassus

says. "The problem we have now is that none of the possibilities look very likely."

When they bagged element 118, the Berkeley team was in a hot race with a group at the Joint Institute for Nuclear Research in Dubna, near Moscow. But Gregorich doesn't think the rivalry was responsible for the error. "In '99, things did go fairly quickly," he acknowledges, noting that researchers felt pressure to complete their work rapidly before other labs could perform similar experiments. "But we're trying to get away from the rivalry aspects of the different labs. It's pretty much a different generation of scientists from when there was a lot of rivalry in the '70s."

Hofmann says that Dubna's observations of elements 114 and 116 suffer from uncertainties similar to those of the Berkeley experiment, but their results have an internal consistency that gives him more confidence in the Dubna data. He praises the Berkeley team's candor, and, along with the rest of the heavy-ion community, hopes a fuller accounting will reveal what went wrong. Dieter Ackermann, also of GSI, says, "The problem now for me is that I need an explanation."

–CHARLES SEIFE

ENTOMOLOGY

First Light on Genetic Roots of Bt Resistance

For the last 5 years, farmers, particularly cotton growers, have been able to reduce their use of chemical pesticides by planting crops genetically engineered to make insecticidal proteins from the bacterium *Bacillus thuringiensis* (Bt). But insects can adapt to these natural toxins, just as they do to synthetic chemical pesticides. For example, some populations of diamondback moths, a devastating pest of cabbage and related crops, are no longer bothered by sprays of Bt bacteria used by organic farmers. This has raised worries that extensive use of the modified crops will lead to widespread resistance that could render both the crops and the Bt sprays useless. Now scientists have taken a big step toward understanding how Bt resistance arises—a key to predicting the occurrence of such resistance.

In work reported in this issue of *Science*, two teams, one led by Linda Gahan of Clemson University in South Carolina and David Heckel of the University of Melbourne in Australia and the other by Raffi Aroian of the University of California, San Diego, have identified the first resistance genes for Bt. "It's a huge leap forward," says Bruce Tabashnik, an entomologist at the University of Arizona, Tucson. The most practical payoff may be an easy DNA test for detecting resistance in insect pests; this could help alert farmers to burgeoning resistance in time to stop planting Bt crops and switch to chemical pesticides for a while.

For their experiments, which are described on page 857, Gahan and Heckel used a lab strain of the tobacco budworm that was developed by Fred Gould of North Carolina State University in Raleigh. This strain, known as YHD2, resists the Bt toxin designated Cry1Ac, which is present in a genetically modified cotton produced by Monsanto Corp. of St. Louis.

In 1997, the Gahan-Heckel team, working with Gould, obtained evidence indicating that the gene responsible for the budworm's Bt resistance is located on chromosome 9. After narrowing the location of the putative gene, which they called *BtR-4*, the team checked that stretch of the chromosome for known genes that code for proteins that bind the Bt toxin. Resistance might reside in one of those genes, they thought, because of the way Bt toxins kill—by binding to cells in the midguts of insects that eat them, causing the cells to burst. A mutation that could prevent that binding, either directly or indirectly, could thus confer Bt resistance.

Lab studies have identified two classes of proteins that bind to Bt: the aminopeptidases, enzymes used by insects to help digest proteins in their gut, and cadherins, some of which are located on cell surfaces and are involved in cell adhesion. Heckel and Gahan quickly ruled out two aminopeptidase genes, as they weren't located on the same chromosome as *BtR-4*.

So the researchers turned to the cadherins. They used the polymerase chain reaction to isolate a fragment of a cadherin gene that mapped to the same location as *BtR-4*. The fact that the cadherin gene maps to the same area as *BtR-4* provides "almost irrefutable evidence" that it's the Bt resistance gene, Tabashnik says. "The odds of that being a coincidence are essentially nil."

The pair went on to show that this cadherin is made in the right place to confer resistance—the budworm's midgut. What's more, the researchers have evidence that the gene has been inactivated in the resistant YHD2 budworm strain. In that lab strain, but not in nonresistant budworms, the gene's coding sequence was interrupted by the insertion of a retrotransposon—a bit of movable DNA that can jump from place to place in the genome. Such an insertion would likely disable the gene, presumably preventing the Bt toxin from latching onto —and killing—the cells of the budworm's midgut. Finding such a disabling mutation was "totally unexpected," says Heckel, as insecticide targets are usually very important to the insect and can't tolerate such large changes.

Because large mutations such as retrotransposon insertions are easy to detect, researchers should be able to develop a rapid test for this type of resistance to Bt. But a single test won't suffice. "Insects can have more than one mechanism of resistance," explains Ian Denholm of the Institute of Arable Crops Research's Rothamsted Experimental Station in Harpenden, United Kingdom. Indeed, that message is brought home by the Aroian team's paper, which appears on page 860.

Aroian and his colleagues study Bt resistance in the roundworm *Caenorhabditis elegans*, which like insects suffers intestinal damage from Bt toxins. Last year the group located five genes, dubbed *bre* for Bt resistance, that when mutated confer resistance to a Bt toxin called Cry5B. Now they have cloned one of those genes, *bre-5*, and confirmed that blocking its activity, as a mutation might do, does in fact make the worm resistant to Cry5B and also to Cry14A.

The BRE-5 protein turned out to be an enzyme called β-1,3-galactosyltransferase, which adds carbohydrates to lipids and proteins. Aroian's team has evidence suggesting that such carbohydrate addition to the Bt protein receptor is needed for toxin binding in the gut. The researchers also showed that losing the enzyme creates resistance. "It's an important mechanism to understand," Aroian says, because losing the enzyme could be an effective way to gain resistance to many Bt toxins at once. If it works this way in insects, a mutation in the enzyme might help insect pests defeat the next generation of genetically modified crops, which are being endowed with multiple Bt toxins to help prevent resistance.

–ERIK STOKSTAD

Nipped in the bud. DNA tests could help detect genes that allow the tobacco budworm and other insect pests to resist Bt toxins.

CREDIT: F. GOULD/NORTH CAROLINA STATE UNIVERSITY