Although the U.S. support is welcome, some say it falls short of the $20 million that scientists recommended last year as a minimum contribution (*Science*, 23 October 1998, p. 653). "The U.S. [financial] input is disproportionately small," says Benjamin Burr, a plant geneticist at Brookhaven National Laboratory in New York. "But it could have a disproportionate impact because the labs picked have high sequencing capacities." In addition, two other U.S. groups intend to continue sequencing efforts on their own. The University of Wisconsin, Madison, a finalist in the competition for the new grants, hopes to sequence portions of chromosome 11, in part to demonstrate the effectiveness of its optical mapping technique. A group at Rutgers University's Waksman Institute in Piscataway, New Jersey, will link up with other U.S. labs working on chromosome 10.

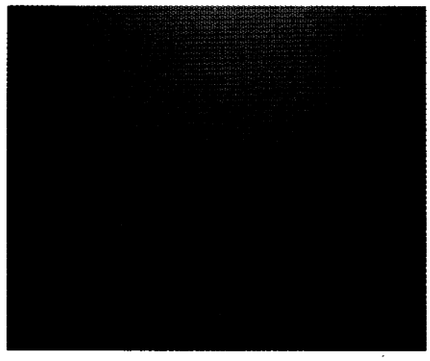
Even with the additional resources, though, Sasaki says "[he] can't promise" to complete the sequencing by 2004. For one, although groups in Canada and the United Kingdom have indicated an interest in sequencing, their governments have not yet committed money. And outside China and Japan, the other Asian groups are expected to contribute minimal amounts of sequence data because their genomics efforts are just getting off the ground. Even China's effort comes with a proviso: Its scientists are sequencing a different rice cultivar from the Nipponbare used by the rest of the international collaboration. **–DENNIS NORMILE**

PLANETARY SCIENCE

Neptune May Crush Methane Into Diamonds

Diamonds might become as cheap as coal if miners could ever plumb the hellish interiors of Neptune and Uranus. Laboratory researchers are now creating tiny bits of those interiors, where heat and pressure can be far more intense than in the depths of Earth. They are finding, among other surprises, tiny flecks of diamond.

On page 100 of this issue of *Science*, mineral physicist Robin Benedetti of the University of California, Berkeley, and her colleagues report that methane—a major constituent of Neptune and Uranus's deep interiors—decomposes far more easily than predicted when it is heated and squeezed in the laboratory. That decomposition, which produces diamonds and complex organic matter, could have altered the chemical composition and internal churning of those planets. "This is an exciting piece of work," says mineral physicist Russell Hemley of the Carnegie Institution of Washington's Geophysical Laboratory, "because it shows the promise of this sort of experiment in studying planetary interiors."



Diamonds in the sky. Neptune's heat and pressure may forge them.

Experimentalists have only recently started exploring the highly fluid interiors of the gas giants—Jupiter, Saturn, Uranus, and Neptune. They first squeezed the hydrogen that makes up the bulk of such bodies to see when it might turn into a liquid metal (*Science*, 22 March 1996, p. 1667). Now they're working on methane, which becomes a prominent constituent of Neptune deeper than 4000 kilometers below the planet's visible cloud tops. Benedetti and her colleagues sealed liquid methane between the tips of two gem-quality diamond "anvils" and squeezed them together to raise the pressure as high as 50 gigapascals (GPa, equal to 500,000 atmospheres). Then they shot a laser through the diamonds and the sample until the temperature of the methane rose as high as 3000 kelvin. Under such extreme conditions, equivalent to those as deep as 7000 kilometers below Neptune's cloud tops, the methane decomposed into two identifiable forms of carbon—diamond crystals about 10 micrometers in size and complex, polymerized organic matter.

Theorists had suggested that diamonds might form in Uranus and Neptune, but only toward the center of the planets, above a pressure of 300 GPa. The shallower level for diamond formation is a surprise, says Hemley, and it means that far more of the interior could be producing a girl's best friend, with proportionately greater effects on the planet as a whole. Being denser than the fluid from which they formed, diamonds would sink, releasing heat from their store of potential energy. That heat would help churn the interior, perhaps boosting Neptune's magnetic field, which is driven by such convection. It might also add to the heat seen escaping the planet.

Methane might also be breaking down at depths even shallower than those at which diamond forms, producing byproducts such as light hydrocarbons that telescopes and spacecraft might detect. In other diamond-anvil experiments, mineral physicist Thomas Schindelbeck and his Geophysical Laboratory colleagues found that methane is unstable at just 7 GPa and 2000 kelvin. From such shallow depths, decomposition products such as ethane could waft up to the visible cloud tops—fumes from the hell a few thousand kilometers down.

The new diamond-anvil results are reminding researchers to take a critical look at the textbook picture of gas giants as being neatly subdivided into layers of unchanging composition. "One needs to take into account high-pressure chemistry in understanding the icy planets like Uranus and Neptune," says Hemley—not that the experiments so far give a complete chemical picture of the planets' innards. "The real Neptune is a more complicated soup of chemical molecules" than experimentalists have cooked up in their first tentative forays, says planetary scientist William Hubbard of the University of Arizona, Tucson. There's water mixed in with the methane, he notes, as well as hydrogen. Either one might affect reactions in the planet's interior. So recreating the depths of hell on Neptune and other planets will take a while longer. **–RICHARD A. KERR**

NEUROSCIENCE

India Creates Novel Brain Research Center

NEW DELHI—India is hoping to break into the front ranks of neuroscience with a new National Brain Research Center (NBRC) that opens here this week. The venture hopes to capitalize on India's large population and on a pool of talent now scattered around the world: Indian researchers now working abroad are expected to fill most of the 12 new scientific slots, working in areas ranging from developmental and computational neurobiology to the effects of malnutrition on the brain.

Smart move. India's Manju Sharma announces "state-of-the-art" facility for brain science.

The center, funded by the Department of Biotechnology, will be devoted to basic research. "It will be a state-of-the-art institute ... and will have no clinical facilities attached to it," says Manju Sharma, a botanist and secretary of the biotechnology department, adding that the center will serve "as a national apex for brain research." In another unusual twist, half of its $4 million budget over the next 3 years will be earmarked for extramural research, including scientists at labs funded by other

CREDITS: (TOP TO BOTTOM) JPL/NASA; P. BAGLA

ministries, such as the well-regarded National Institute of Mental Health and Neurosciences in Bangalore.

India has a special opportunity to contribute to the field of neural imaging, says Vijayalakshmi Ravindranath, a neurochemist in line to be director of the center, by carrying out large-scale functional mapping studies on so-called "drug-naïve patients," those with neurological disorders who have not yet received treatment. Supporters acknowledge that it will be a while before the center can hope to enter the front ranks of global science, however. "Catching up is a Herculean task, and it may take another 20 years," says Prakash Narain Tandon, a neurosurgeon at the All India Institute of Medical Sciences in New Delhi. But they argue that creating the center is an important, and necessary, step.

India is already looking for international collaborators. About 250 scientists from five countries are participating in a Colloquium on Brain Research here this weekend to showcase the new center, and Richard Nakamura, deputy director of the U.S. National Institute of Mental Health, is heading a delegation that expects to sign a memorandum of understanding for future collaborations and scientific exchanges with NBRC. The center also hopes to link up with Japan's Brain Science Institute at the Institute of Physical and Chemical Research (RIKEN), outside Tokyo. "India is not without promise in the neurosciences," says Nakamura. "Indian scientists have always done very well in the U.S. because they are well trained and do not face a major language barrier. By setting up strong centers within India, this brain drain can be slowed, and talented scientists can help develop the economy of India and work to improve the health of its people."

The center is currently housed in temporary quarters at the International Center for Genetic Engineering and Biotechnology. Work is under way on a new home in Gurgaon, about 35 kilometers outside Delhi, where an unused vaccine laboratory built several years ago is being renovated.

–PALLAVA BAGLA

HUMAN GENETICS

Gene Defect Linked to Rett Syndrome

As the parent of a child with Rett syndrome, Patty Campo describes the genetic disease as "a horrific nightmare." Second only to Down syndrome as a cause of female retardation, Rett syndrome left Campo's daughter, who appeared normal at first, unable to stand, talk, or use her hands by age 5. Now, a team led by pediatric neurologist and geneticist Huda Zoghbi of Baylor College of Medicine in Houston may have tracked down the gene at fault in Rett syndrome, which afflicts at least one in 10,000 girls.

In the October issue of *Nature Genetics*, Zoghbi, Uta Francke at Stanford University in California, and their colleagues report that mutations in a gene called *MeCP2* cause nearly a third of the Rett syndrome cases they studied. The protein encoded by the gene is known to help "silence," or shut down, other genes that have been tagged with a methyl group during development. The group's work is the first to link a human disease to a defect in this process, says geneticist Brian Hendrich, who co-authored a News and Views editorial on the work.

Early sign. Excessive hand-wringing is one of the first symptoms displayed by girls with Rett syndrome.

CREDIT: HHMI

Exactly how the defect leads to the neurological decline of the afflicted girls has yet to be deciphered. Still, geneticists say they welcome the discovery. "*MePC2* silencing, which underlies the defect, is not only fascinating in its own right," says Huntington Willard of Case Western Reserve University in Cleveland, Ohio, "but the discovery of its link to Rett syndrome also opens all kinds of doors to new [research] avenues" that might lead to a treatment.

Although at least a half-dozen labs pursued the Rett syndrome gene, the hunt stretched out into decades. Geneticists normally locate disease genes by studying how the disease is inherited in afflicted families. But Rett syndrome rarely seems to run in families, and even when the mutation is passed down in a family, it is often hidden.

One problem is that males who inherit the mutation, which affects a gene on the X chromosome, usually die before or shortly after birth, presumably because they do not have a second copy of the X chromosome that might compensate for the defective one. In contrast, females carry two X chromosomes. But their cells randomly inactivate one to avoid overproducing proteins encoded by X chromosome genes, and so a female may not have symptoms unless more than half her cells express the bad gene.

Despite these hindrances, Zoghbi's group found two families with more than one affected member and Stanford's Francke found another. They combined their samples to help Zoghbi narrow down the location of the Rett syndrome gene to a region around the q28 segment of the X chromosome. In addition, Eric Hoffman of the University of Pittsburgh also had evidence that the gene is located in Xq28. To pinpoint the gene, Zoghbi then turned to what she terms "a brute-force way."

She scrolled through databases to identify all the known genes in Xq28—at least 100 candidates, she notes—and then sequenced the versions carried by the patients and compared their DNA to that of unaffected individuals. After more than a dozen failed tries, her team hit pay dirt with the *MeCP2* gene, which had been cloned in 1992 by geneticist Adrian Bird's team at the University of Edinburgh in the U.K.

Still, many questions remain. The gene was mutated in only three of eight affected family members and in five of 21 spontaneous cases, raising the possibility that mutations in other X chromosome genes might also cause the disease. Zoghbi offers another possible explanation: Her team only scoured *MeCP2*'s protein-coding regions and so might have missed mutations in regulatory regions that affect the gene's expression.

Even more mysterious is how mutations in one gene involved in the silencing of other genes could cause the myriad of Rett syndrome defects. "We really don't know the answer to that," says Zoghbi, although she and others have several ideas. One is that a failure of *MeCP2* to prevent excess gene expression results in genetic "noise" that harms the brain, in particular.

Studying *MeCP2* function in patients' cells and analyzing the defects in mice that have had their *MeCP2* genes knocked out could yield some answers. Bird and his colleagues have already made knockout mice and found, consistent with what's already known about Rett syndrome, that male mice without a functional *MeCP2* gene die before birth, while females that have one bad copy of the gene may develop symptoms similar to Rett's. Only after learning the targets of the *MeCP2* defect, Zoghbi says, "can one start thinking about treatments."

–TRISHA GURA

Trisha Gura is a writer in Cleveland, Ohio.