

SPACE RESEARCH

Mir Gets New Lease on Its Scientific Life

After more than 7 months in mothballs, the Mir space station is once again open for business. A Netherlands-based company called Mir Corp. is helping to fix up the aging and trouble-plagued facility to make it ready for researchers—and eventually rich tourists. But it could be a short-lived venture: The Russian government, which owns the 14-year-old facility, has not decided how long to keep it in orbit, and U.S. space officials say privately they would love to see it shut down once and for all.

Mir Corp. has pledged to spend at least \$20 million on the venture. It is backed by several wealthy American investors, including Washington, D.C.-based telecommunications millionaire Walter Anderson, and the majority shareholder is the Russian space company Energia, which operates Mir for the Russian government. As a start, Mir and Energia bankrolled the launch on 4 April of a Soyuz spacecraft carrying two cosmonauts to Mir. They will check out life-support systems on Mir's collection of pressurized modules, fix a small leak, and conduct some 50 Russian science and technology experiments. Mir Corp. officials hope that a successful mission will help convince Western governments and companies to reactivate experiments they already have on board and attract new paying customers.

Jeffrey Manber, Mir Corp. president and a former Washington representative of Energia, argues that Mir offers opportunities "ranging from industrial production and scientific experimentation to space tourism and in-orbit advertising." With completion of the international space station expected to slip from its scheduled 2004 date, Manber says Mir can serve as a temporary substitute for companies and governments that have set aside money for experiments: "There is already equipment on Mir which can be used very cost effectively."

Manber admits that doing science aboard Mir won't be a big moneymaker, and the company ultimately hopes to lure wealthy and adventurous tourists to visit the station. That idea received an unexpected boost last week from U.S. Transportation Secretary

Rodney Slater, who applauded the company's efforts to create a space tourism business during a speech to aerospace industry officials in Colorado Springs, Colorado.

But many U.S. aerospace industry officials, NASA managers, and others familiar with Mir are skeptical about the company's prospects. Mir has suffered computer and power shutdowns, a fire, and a collision with a resupply vehicle that damaged one of its modules. And Russia's financial troubles

prevented significant upgrades during the 1990s. Moreover, the U.S. government in recent years has encouraged the Russians to deorbit Mir and concentrate the country's limited resources on building and launching its portion of the international station.

"There's a tough road ahead," says Manber, acknowledging the uncertain status of the facility. He is hopeful,

however, that the current mission will be followed this fall by a crew conducting experiments on behalf of Western scientists. But for now, Mir is proving that space stations can have many lives.

—ANDREW LAWLER



Back in business. Cosmonaut Alexander Kaleri waved as he entered Mir last week.

GENOME SEQUENCING

Claim and Counterclaim On the Human Genome

J. Craig Venter stole the show last week. The day before Venter appeared at a hearing of a House science subcommittee on 6 April to review research on the human genome, his company, Celera Genomics Corp. of Rockville, Maryland, issued a press release announcing that it had "completed the sequencing phase of one person's genome." The notice, which had a ring of finality about it, indicated that Celera's computers are poised to assemble the human data into a complete genome—a formidable task that Venter predicted at the hearing would take "3 to 6 weeks." Sometime later this year, he says, he will make the data available on Celera's Web site. Celera's stock, which had fallen abruptly in mid-March, soared.

It was an effective bit of propaganda: Celera released no new scientific data, but left the impression that it has bagged the human genome—just as it bagged the genome of the fruit fly in collaboration with the Berkeley *Drosophila* Genome Project earlier this year. But members of the nonprofit consortium that aims to complete

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to inactivate or "knock out" the *VR1* gene that they found that the resulting mice are impervious to capsaicin-induced pain.

For example, capsaicin injected into the hind paw of a normal mouse causes the animal to lick and shake the tender paw. However, the mutant mice barely reacted to the injection, and their paws did not swell or become inflamed as much as they do in normal mice. When researchers laced the drinking water of normal mice with capsaicin, the normal mice took one sip, rubbed their snouts, and stayed clear of the water bottle. The mutant mice, however, drank happily, says Caterina, who is now at Johns Hopkins University.

The mutant animals also tolerated high heat better, including having their tails immersed in a hot water bath and their paws put in contact with a hot plate. The animals did eventually react in both tests, showing that sensitivity was lessened, not eliminated, Caterina notes. This suggests that other heat-sensing channels play a role as well, he says.

Another type of test suggests that *VR1* plays a role in the extra sensitivity to heat usually displayed by inflamed tissues. Mustard oil painted onto the paws of normal mice causes them to become inflamed and very sensitive to heat—just as sunburned skin is seared by warm water or sunshine. But in the mice lacking *VR1*, the mustard-oil treatment did not enhance the response to heat, although the animals still displayed the hypersensitivity to touch that develops in inflamed tissues. Because touch-sensitive pain must be triggered by other neuronal responses, says Julius, the finding suggests that blocking *VR1* would not relieve a common, painful condition—extreme sensitivity to touch, such as that accompanying shingles.

The mouse work suggests, however, that such inhibitors may help combat another type of especially troubling pain, the chronic internal pain that can accompany tissue damage. Julius and his colleagues suspect that *VR1* receptors might contribute to such pain. They found, for example, that neurons carrying the receptors can be excited by the acidic environment produced by inflammation. But neurons from *VR1*-deficient mice bathed in an acidic solution did not react as vigorously as neurons from normal mice did. Thus, the researchers hope that blocking the *VR1* receptor might help relieve chronic internal pain.

The fact that the *VR1* knockout mice seem otherwise normal is encouraging for drug development, Campbell says. "It would appear that the [*VR1* receptor] molecules are specific to pain-sensing neurons," he says. That could lead to drugs with few side effects—perhaps only an inability to taste Tabasco sauce.

—GRETCHEN VOGEL