Astronomy

Galileo closes in on Jupiter

The Galileo spacecraft passed a critical milestone late last month in the final leg of its 6-year voyage to the solar system's biggest planet. On July 27, Galileo fired its main engine, setting course for a 2-year grand tour of Jupiter and its moons.

The engine firing came 2 weeks after the craft released a probe that will parachute into Jupiter's atmosphere on December 7 (SN: 7/22/95, p.54). On the same day, Galileo will begin its Jovian tour, flying past the volcanically active moon Io.

The mission now faces other hurdles. Plagued by a main communications antenna that never unfurled, Galileo must store data on tape instead of relaying it directly to Earth. Because the craft's secondary antenna transmits information more slowly, not all recorded observations will be transmitted. The craft will also make fewer observations than planned.

NASA scientists have already coped with these problems in retrieving data taken by Galileo en route to Jupiter. But the craft will lie considerably farther from Earth during its Jovian tour, making transmission more difficult. With the help of data compression, the space agency says, Galileo should accomplish 70 percent of its original mission.

Sunny findings from Ulysses and Spartan

In mid-June, Ulysses became the first spacecraft to explore the sun's north polar region. Having attained its maximum northern latitude of 80.2° on July 31, the craft will spend 2 more months exploring the pole. In the meantime, scientists continue to report findings from the craft's southern polar pass.

Ulysses found that the solar wind, the stream of charged particles blown out by the sun, exerts a higher pressure at the south pole than over the equator, says Ulysses project scientist Edward J. Smith of NASA's Jet Propulsion Laboratory in Pasadena, Calif. This suggests that the heliosphere, the region dominated by the solar wind and magnetic field, extends much farther out into space along the poles.

Scientists had previously reported Ulysses observations that the solar wind blows twice as fast, about 800 kilometers per second, at the poles than at the equator (SN: 11/19/94, p.326). That speed, however, can fluctuate by as much as 50 km per second. Researchers who recently imaged the corona, the sun's outer atmosphere, now suggest an explanation for the variation. Pictures taken with a coronagraph aboard the Spartan craft, flown several times from the space shuttle, reveal plumes of light emanating from the poles of the corona. These polar rays presumably stem from activity in slightly denser regions of the corona. The higher density produces a somewhat slower wind, possibly accounting for the fluctuations in polar wind speed detected by Ulysses.

Madhulika Guhathakurta and Richard R. Fisher of NASA's Goddard Space Flight Center in Greenbelt, Md., and Ruth Esser and Shadia Rifai Habbal of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., presented the findings in late June at a solar wind conference in Dana Point, Calif. In another Spartan study, the team reports in the June 15 GEOPHYSICAL RESEARCH LETTERS that protons streaming out of the inner corona are twice as hot as electrons. The higher temperature, which has no obvious explanation, is consistent with Ulysses' measurements.

Fisher, Guhathakurta/GEOPHYSICAL RESEARCH LETTERS

Image of coronal hole at the sun's north pole, a composite taken by the Spartan and the ground-based Mauna Loa coronagraphs, shows polar rays.

Biology

John Travis reports from the Short Course in Mammalian Genetics at Jackson Laboratory in Bar Harbor, Maine

A full-fledged Alzheimer's mouse

John D. Gearhart's latest litter of mice is just a few weeks old, but already he has high expectations for them. He hopes the mice will provide a model of Alzheimer's disease, a neurodegenerative disorder. Other investigators pursing such a model, which would help test potential therapies, have had only limited success so far.

Gearhart, of the Johns Hopkins University School of Medicine in Baltimore, and his colleagues believe they may have had better luck because of a twist they've added to the common strategy for making an Alzheimer's mouse.

To construct a mouse model of an illness, researchers add to the animal's genome a gene they believe may be responsible for the disease. In the case of Alzheimer's, attention has focused on a gene that in humans directs the synthesis of a molecule called amyloid precursor protein, or APP. The brains of people who had Alzheimer's often display dense plaques of a protein called beta-amyloid, a fragment of APP. Many investigators therefore believe that extra or abnormal APP production may cause the disorder.

The APP gene, however, is huge, much bigger than genes researchers can easily put into the mouse genome. As a result, notes Gearhart, most researchers have taken a shortcut, inserting only severely abbreviated versions of the APP gene that contain little more than the minimum amount of DNA necessary to make the protein.

But last year, Gearhart and his colleagues successfully added to the mouse genome the full version of the human APP gene and the DNA that normally surrounds it, an amount of genetic material about 60 times greater than researchers have added in previous attempts. They have now extended their work, creating new strains of mice with full-sized APP genes that include mutations found in some of the inherited forms of Alzheimer's disease.

"I think it's the only paradigm that can mimic the human system," says Gearhart, who must wait to see if the mice develop Alzheimer's as they age.

Dwarfism gene under scrutiny

Geneticists are finally finding the genes responsible for most cases of dwarfism. Last year, two groups reported that achondroplasia, the most common genetic form of dwarfism, results from a small mutation in the gene that contains the instructions for fibroblast growth factor receptor 3 (FGFR3), a protein needed for skeletal development in the embryo.

They also showed that in almost all cases of achondroplasia, the mutation consists of an alteration of exactly the same nucleotide, one of DNA's basic building blocks.

This nucleotide's mutation rate—how often it spontaneously changes—is the highest ever calculated, notes Clair A. Francomano of the National Center for Human Genome Research in Bethesda, Md. "This is a mutation that keeps occurring again and again," she says. "I can't begin to give you an explanation as to why."

This year, in the July NATURE GENETICS, Francomano and her colleagues reported that a less severe form of dwarfism, hypochondroplasia, also stems from a mutation on the *FGFR3* gene, but at a completely different nucleotide. Among the 30 hypochondroplasia patients they've studied, 29 have a mutation at that particular nucleotide, she says.

One benefit of the new data on *FGFR3*, says Francomano, is that she can offer prenatal testing to couples who are both achondroplasic. That's important, because if each parent passes on the mutated *FGFR3* gene, their child will have no normal copy. Babies with two copies of the mutated *FGFR3* gene don't survive beyond a few months because of their severe skeletal abnormalities, she explains.