Astronomy

Two stars, two planetary embryos

In the search for planets outside the solar system, astronomers have focused on stars that go it alone in the cosmos. Theorists have reasoned that stars that occur in pairs are less likely to be surrounded by disks of gas and dust, the embryonic material from which planets arise. The gravitational tug-of-war between the two stars could prevent disks from forming or from lasting long enough to make planets.

New observations reveal that, at least in one case, a close stellar partnership hasn't prevented the formation of disks. Indeed, the orbiting duo may form planets faster than single stars do.

Studies with the Very Large Array radio telescope near Socorro, N.M., confirm that what had seemed a single star in the L1551 molecular cloud—a star-birth region 450 light-years from Earth—is in fact two stars. The stars are just slightly farther apart than the distance from the sun to Pluto.

Despite their closeness, each star has its own disk of gas and dust, report Luis F. Rodríguez of the National Autonomous University in Mexico City and his colleagues in the Sept. 24 NATURE.

The proximity of the two stars does seem to have affected the size of their dust disks. Each has a radius similar to the distance between the sun and Saturn—about one-tenth the typical size of disks encircling single stars. "Large outer planets like Neptune and Pluto will not form in these disks because the disks are simply not big enough," says Rodríguez.

Because paired stars are believed to be far more common than single stars, "the finding that the stars in this run-of-the-mill binary [have the potential] to form Earthlike planets suggests that planet formation is a very robust process that may be going on around most stars in the universe," Rodríguez notes.

The interaction of the two stars may downsize each other's disks, but it also seems to have made the disks more massive, notes theorist Alan P. Boss of the Carnegie Institution of Washington (D.C.). The observations suggest that each disk contains about 5 percent as much mass as the sun, more than double the amount in disks surrounding young, single stars.

According to Boss, when a disk is massive enough and cold enough, it becomes unstable and breaks up into large clumps. Such clumps may represent the rapid, wholesale formation of giant planets (SN: 8/8/98, p. 88). That's in contrast to the standard picture, in which planets build up little by little as material in the disk slowly packs together.

Boss adds that if a giant planet forms near the edge of one of the disks, it might be pulled out of orbit by the gravity of the other star. Such a scenario could bolster support for the controversial claim that the puzzling object TMR-1C is a wayward planet ejected by a pair of stars that lie nearby (SN: 6/6/98, p. 357). —*R.C.*

More water on the moon

Researchers analyzing data from the Lunar Prospector, the tiny robot that has been orbiting the moon since January, have drastically upped their estimate of the amount of frozen water buried beneath the lunar poles. In March, the Prospector team suggested that craters in permanent shadow at the north and south poles could hide 300 million metric tons of ice (SN: 3/14/98, p. 166). After refining models of how water might be delivered to the surface, the team now says that the poles may contain as much as 600 billion metric tons, with the north pole harboring perhaps 15 percent more of the precious resource than the south.

William C. Feldman of the Los Alamos (N.M.) National Laboratory and his colleagues report the new findings in the Sept. 4 SCIENCE. The evidence is indirect. Prospector's measurements indicate that the poles have a higher abundance of hydrogen than elsewhere on the moon, and the team asserts that the hydrogen is almost certainly tied up in water. —*R.C.*

Biomedicine

Gene interplay may govern spread of cancer

Cancer is like a deadly infection. The body can often handle the problem, or physicians can treat it, if it confines itself to a small area. If it spreads, however, disaster usually follows. Scientists now suggest that a gene, *KAI1,* that can suppress the dispersal of tumor cells is under the control of *p53*, a gene often mutated in cancers.

In 1995, scientists observed that adding *KAI1* to rat prostate tumor cells prevented the cancer from spreading to the animals' lungs, as it normally does. Since then, they have also found that as human prostate, breast, bladder, pancreatic, and lung cancers grow, tumor cells frequently make less of *KAI1*'s protein—even though the gene isn't mutated.

Kounosuke Watabe of Southern Illinois University School of Medicine in Springfield and his colleagues recently identified a DNA sequence called a promoter region around *KAI1*. Proteins that attach to this region control the gene's activity. Unexpectedly, *KAI1*'s promoter resembles promoters that *p53*'s protein binds.

Normally turned on by DNA damage, *p53* safeguards the body against cancer in several ways. Its protein can temporarily halt the division of cells, giving them time to fix mutations. The protein can also induce the suicide of cells damaged beyond repair. Given *p53*'s protective powers, it's not surprising that it is the gene most frequently found mutated in tumors. More than half of all cancers have a disabled *p53*.

Watabe and his colleagues demonstrated that *p53's* protein does indeed bind to *KAI1*'s promoter region, suggesting an unanticipated role for *p53* in suppressing the spread of cancer. In the Sept. 15 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, they also report that adding a working copy of *p53* to cells significantly increases production of *KAI1*'s protein.

They propose that mutations in *p53* explain why cancer cells stop producing *KAI1*'s protein. The function of that cell-surface protein "is not well-understood. It is probably involved in cell adhesion, but that's about all we know," notes Watabe. —*J.T.*

The human genome race heats up

Only a few months after a private company revealed its intent to decipher the full human genome by the year 2005 (SN: 5/23/98, p. 334), leaders of the federally funded Human Genome Project have announced an acceleration of their own timetable. Instead of wrapping up in 2005, the project plans to completely finish sequencing the human genome by 2003 and to create a "working draft" of it by 2001. —*J.T.*

War on drugs enlists an antibody

Hoping to combat cocaine overdoses or make it difficult for a rehabilitating addict to get high, scientists have enlisted the immune system to generate antibodies that bind cocaine and clear it from a person's system. One concern, however, is that a person could respond by simply taking more cocaine than the antibodies could mop up.

An antibody that chews up a cocaine molecule and comes back for more has now been proven to protect mice from becoming addicted to and overdosing on cocaine, Donald W. Landry of Columbia University and his colleagues say in the Aug. 18 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

The drug-busting protein, known as a catalytic antibody, splits a molecule of cocaine into two harmless fragments without destroying itself (SN: 3/27/93, p. 199). In trials on volunteers, researchers have already started tests of cocaine antibodies that simply bind to the drug, as well as a vaccine that induces such antibodies, notes Frank Vocci of the National Institute on Drug Abuse in Bethesda, Md. "The [immune] approach is alive and well. It's laboring along, and we should have an answer in a year or two about how viable this approach is," he says. —*J.T.*