

Every 48.3 days, the star's light dims steadily over 2.4 days, stays dim for about 18 days, and then brightens back to normal during another 2.4 days. Alternate dimmings progress slightly differently, suggesting that whatever obscures the star, there are two of them circling it every 96.72 days at an orbital distance of 0.3 times the Earth-sun distance, or closer than Mercury orbits the sun.

Herbst and colleagues "are really scratching our heads over this," but it appears that the inner region of this protoplanetary disk, where rocky, Earth-like planets might be forming, is behaving much as Saturn's rings do. Rather than a solid body, the obscuring matter could be the long, low crests of two pinwheel-like waves of gas and dust spiraling outward from either side of the disk, viewed edge-on. As at Saturn, such spiral density waves would be triggered by the gravitational tug of a large mass—a small star, a planet, or a denser clump of disk material—orbiting unseen, starward of the waves.

"It's fascinating," says astronomer Ray Jayawardhana of the University of California, Berkeley. "Already by 3 million years you see clumping. It's all pointing to a lot happening in the first few million years." Most satisfying for astronomers, things are visibly happening even now. A brightening in mid dimming has been weakening over the 6 years of observations, and the dimmings have been getting longer. Such changes in the silhouette of the disk might show theorists why newly formed planets—including most extrasolar planets found so far—seem to have migrated inward toward their stars and why some manage to stop just before being devoured by their parent stars.

—RICHARD A. KERR

AGBIOTECH

A Little Pollen Goes a Long Way

One of the major concerns about genetically modified (GM) crops is that they might spread their genes to nearby weeds or organic crops. Some governments have responded by recommending that GM crops be planted in isolation, or by setting limits on GM material in organic or conventional crops. But they have had few data to go on. Now a comprehensive study, described on page 2386, provides some hard numbers on the movement of pollen between fields, with implications for regulators. "These are real-world data that can be used for real-world decisions," says Paul Raymer, an agronomist at the University of Georgia, Griffin.

A team led by reproductive ecologist Mary Rieger of the Cooperative Research Center for Australian Weed Management and the University of Adelaide in Australia re-

ports that canola pollen can travel considerable distances but that the amount of gene flow is minimal. Although the findings reinforce the difficulty of growing GM-free crops, they also suggest that the levels of gene diffusion are below European standards for contamination of conventional food.

Over the last decade, a handful of small experiments has indicated that a minuscule amount of pollen from engineered crops can spread up to a few hundred meters. But what happens on real farms was unclear. To find



In the air. Pollen from canola flowers (right) moved up to 3 kilometers between fields.



out, Rieger and her colleagues at the University of Adelaide and the University of Western Australia in Nedlands took advantage of a unique opportunity. In 2000, Australian farmers for the first time planted varieties of canola with resistance to acetolactate synthase-inhibiting herbicides. (These crops are not GM varieties but instead were created by mutagenesis.) Working in three states and under various climatic conditions, Rieger's team collected seeds from 63 nearby fields planted with conventional canola.

The herbicide-resistance trait spread to 63% of the conventional fields, including some up to 3 kilometers away from the source. The percentage of resistance among seed samples ranged up to nearly 0.2%, but when averaged per field, the highest percentage was 0.07%. The harvests from the vast majority of fields contained less than 0.03%.

The good news is that this level of gene flow for canola is much lower than previous studies suggested. And Rieger says it should be applicable to GM varieties of canola. If so, the contamination of non-GM canola would be less than 1%, which is the cutoff that Australian regulators have discussed as acceptable and that their European counterparts have provisionally OK'd. Rieger says that the lower gene flow should reassure consumers that the chance of transgenes getting into non-GM crops is small.

But the study underlines a clear risk:

Once transgenes are introduced, they can't be completely controlled. That's a problem for organic farmers. "It's going to be difficult with any commodity to produce a truly GM-free crop," Raymer says. "Zero tolerance is not going to work." Because of the long distance its pollen travels, canola might not be a good plant to engineer for growing pharmaceuticals or anything else that should stay out of the food supply, notes population geneticist Norman Ellstrand of the University of California, Riverside.

Rieger and her colleagues also discovered a conflict with earlier studies of smaller fields, in which the amount of pollen declined exponentially with distance from its source. In Rieger's study, the frequency of herbicide resistance was relatively steady at various distances from the source. The reason could be that bigger fields produce more pollen, and that increases the likelihood that it will travel far. So size apparently matters: "This research indicates that pollen movement on a large scale cannot necessarily be predicated from small-scale studies," Rieger says.

With these new results in hand, however, researchers should have a better handle on gene flow when canola is modified in other ways, says herbicide physiologist Linda Hall of the University of Alberta, Edmonton. Although pollen from any crop should travel in similar ways, Hall and others note that extrapolation is tricky because crops reproduce in different ways. The flowers of wheat and barley, for example, tend to self-fertilize and are less likely to pick up foreign genes. "Canola is one of the more problematic in terms of gene flow," says plant geneticist Rikke Jørgensen of the Riso National Laboratory in Denmark. "This is a worst-case scenario."

—ERIK STOKSTAD

CANCER RESEARCH

Nanoparticles Cut Tumors' Supply Lines

Tumors hungry for sustenance need new blood vessels to deliver the goods. Cancer researchers have spent years working to starve tumors by blocking this blood vessel growth, or angiogenesis, with mixed success (*Science*, 22 March, p. 2198). Now a team has tackled the problem of choking off tumor vessels from a novel angle: The researchers packed a tiny particle with a gene that forces blood vessel cells to self-destruct,

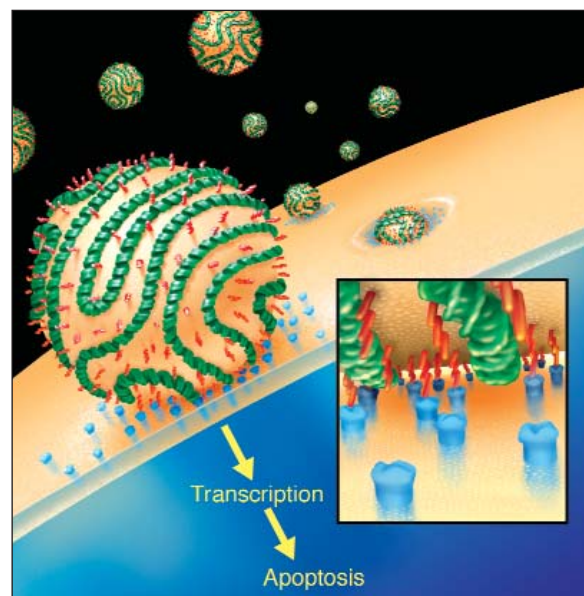
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then they “mailed” the particle to blood vessels feeding tumors in mice.

“It is a very provocative paper, which I think will become a landmark in angiogenesis research,” says antiangiogenesis pioneer Judah Folkman of Children’s Hospital in Boston. Adds Philippe Leboulch, a gene therapist at Harvard Medical School in Boston: “They achieved tumor regression—and they started with tumors [that were] quite large for mice.” Despite their enthusiasm, however, researchers are treading gingerly around the landmines in cancer treatment, where hopes have been raised and dashed many times.

The study, reported on page 2404 and led by vascular biologist David Cheresh of the Scripps Research Institute in La Jolla, California, draws on research in a number of fields. In the mid-1990s, Cheresh and others found signatures specific to different types of blood vessels that they used as target “zip codes.” One of these, belonging to a class of membrane proteins called integrins, is apparently always present on angiogenic, or newly growing, blood vessels but rarely on established ones. The integrin, $\alpha v \beta 3$, has another quality that would turn out to be convenient: It can propel viruses or other small particles into cells.

Meanwhile, various teams had become intrigued by cascades of molecular signals that seem critical to new blood vessel growth. One molecule central to several of these cascades is known as Raf. Inhibiting the *Raf-1* gene in mice prevents blood vessels from forming and halts embryonic development. “Our goal was to identify a common theme that all angiogenic pathways must pass through,” says Cheresh about this line of research. “That is *Raf-1*.”



Bull’s-eye. Nanoparticles packed with targeting molecules (red) anchor to integrins (blue) on the outside of a tumor blood vessel cell before shuttling mutant DNA (green) inside.

Cheresh’s team, assisted by organic chemist and radiologist Mark Bednarski of Stanford University, designed a lipid-based nanoparticle that would target new blood vessels. The nanoparticle’s surface is studded with molecules that bind to $\alpha v \beta 3$ and embedded with copies of a mutant form of the *Raf-1* gene that disrupts Raf’s normal activity.

The researchers infused a dose of these particles into the tails of mice that had been injected earlier with malignant cells. A single treatment erased tumors 400 cubic millimeters in size—1/40 the size of the mouse, or the equivalent of a 2-kg tumor in an 80-kg person—in about 6 days. Animals with metastases to the lungs or liver also saw most of their tumors disappear. In contrast, mutant mice without the $\alpha v \beta 3$ beacon molecules to guide the nanoparticles died after a day or two.

Examining the tumors under a microscope, the researchers saw the expected dead blood vessel cells, which self-destructed after the Raf mutant shuttled inside them. But the team also saw evidence of concentric rings of apoptosis, or programmed cell death, among tumor cells near each dead blood vessel. This illustrated a point already known, although rarely so visible: Each blood vessel cell supports 50 to 100 tumor cells, according to Folkman, and when vessel cells die, tumor cells crumble in a ring around them.

Others agree that the work has several advantages over other antiangiogenesis approaches and experimental cancer therapies. It targets the inside of the blood vessel cell rather than its surface, as other angiogenesis inhibitors appear to do. And the mutant *Raf* genes are packaged in a nanoparticle, not a virus, as is common in cancer gene therapy studies. This means there’s less chance that the body will develop antibodies to the treatment.

At the same time, “one of course has to prove it,” says Leboulch. Inder Verma, a geneticist at the Salk Institute for Biological Studies in La Jolla, wonders whether the treatment does, as predicted, leave healthy cells alone. Scripps and Stanford are both applying for patents on the technology, which is currently licensed to Merck KGaA in Darmstadt, Germany, says Cheresh. In the meantime, the researchers have their fingers crossed that this cancer cure in mice won’t be one of the many that, in Verma’s words, “never sees the light of day”—or at least the fluorescent lights of the cancer unit.

—JENNIFER COUZIN

ScienceScope

Station Plea Biologists, materials scientists, and physicists from Arizona to Japan are flocking to sign an unusual petition to NASA Administrator Sean O’Keefe. Their plea: Reverse current plans to trim the international space station’s crew and scientific instruments and end what they see as discrimination against academic science.

The letter to O’Keefe (www.desc.med.vu.nl/ISS) complains that NASA is failing to capitalize on its immense investment in the station and that cuts in crew and equipment will undermine efforts to produce good orbiting research. In particular, it warns that peer-reviewed science has “become rare, routinely taking a back seat to commercial/NASA center priorities” for the limited slots available on the space shuttle.

The plea struck a chord: Within hours of being posted on the Internet last week, more than 150 scientists had signed. Organizer Millie Hughes-Fulford, a former astronaut and biologist at the Veterans Administration Medical Center in San Francisco, says the drive allows potential station users to “speak with a global, unified voice.” Researchers have until 3 July to add their names.



A Second Blow More belt-tightening could be in store for the Consultative Group on International Agricultural Research (CGIAR) if Japan goes ahead with a planned cut in its contribution.

Last year Japan reduced its support to the 16 agricultural institutes by 48%, to about \$15 million, as part of a deficit-shrinking 10% cut in its overall foreign aid budget, called Official Development Assistance (ODA). As an optional program, CGIAR absorbed a heavier blow than most, as Japan stepped up aid to Afghanistan and honored commitments to U.N.-related agencies. “This really does not indicate any negative evaluation of CGIAR’s activities,” says an official at the Ministry of Foreign Affairs.

The Finance Ministry is contemplating another 10% cut to ODA for fiscal year 2003, which begins next April, and the foreign affairs official says, “We will have to carefully watch what that means for the optional international organizations.” The agricultural institutes last year received some \$336 million from 58 member contributors.

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