from lavas elsewhere around the world, Gordon concedes. Tarduno and Kent see that as a serious problem, suggesting a solely regional phenomenon like a redirecting of Pacific Ocean plate motions, but Gordon suspects the data from beyond the Pacific may just not be good enough yet.

If so, he, like Sager, looks to Earth's interior for the driver of true polar wander. Perhaps a pile of ocean plates that had sunk 700 kilometers down finally broke through into the lower mantle, abruptly shifting the planet's mass distribution and triggering true polar wander. Then plate tectonics would lie behind Earth's tipsiness.

–RICHARD A. KERR

REMOTE SENSING SATELLITES

The Promise of All Weather, All the Time

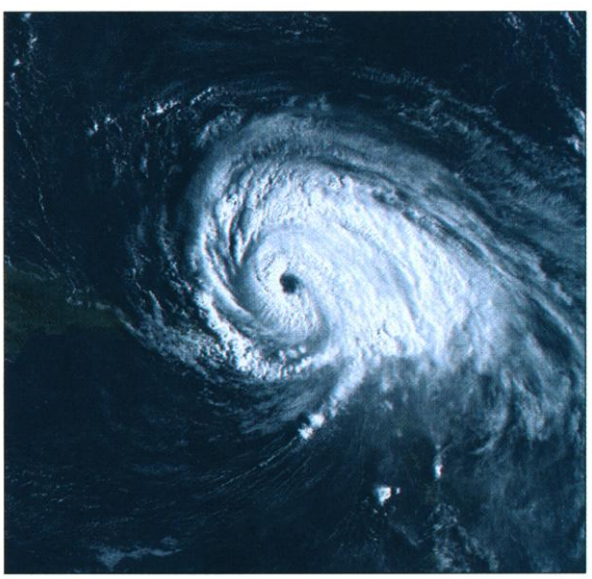
Want to watch the weather unfold on your home computer or TV? Next year a Mississippi company called Astro Vision Inc. hopes to launch the first in a series of small satellites that would provide live, high-quality color videos from space of storm fronts, hurricanes, forest fires, and other natural disasters. The satellites have piqued the interest of NASA officials, who hope that the data will be useful to researchers, and they have caught the attention of venture capitalists, who see an opportunity to feed the Internet's insatiable demand for material.

This week company officials said they have almost finished raising nearly $65 million in private funding for the first phase of their business plan, which calls for the launch of Avstar 1 in October 2001 and a twin 6 months later. The fund raising was aided by the prospect of some real income: Under a 1998 contract, NASA will buy $9.4 million worth of data on how tornadoes form. The company has a broader market in sight, however—the millions of people who watch The Weather Channel and monitor the Web site of the National Oceanic and Atmospheric Administration (NOAA). "We're going to change the way we view Earth's environment and weather," boasts Michael Hewins, the company's chief executive officer. NASA officials aren't willing to go that far, but they are complimentary. "It's a neat little project, and they've got a good shot at making it happen," says David Brannon, chief of commercial remote sensing at NASA's Stennis Space Center in Pearl River, Mississippi, who has worked closely with Astro Vision officials for the past 3 years.

Each Avstar satellite will cost between $20 million and $25 million to build and launch, says Hewins. It would carry both a wide-angle camera with a resolution of 7 kilometers and a second camera with a narrower field to spot objects as small as a half-kilometer across. Although its resolution is poor compared with military spy satellites, it is similar to the 1-kilometer resolution achieved by NOAA's Geostationary Operational Environmental Satellites (GOES) and well-suited for weather watching.

Astro Vision managers will be able to provide users with an image per minute, rather than the single image a government weather satellite typically produces every 15 minutes to a half-hour. That will enable Astro Vision to stitch together videos of hurricanes evolving, thunderstorms growing, and forest fires spreading. A customer interested in volcanoes could be alerted to an eruption, for example, or forest rangers could monitor the spread of fires. The second phase of Astro Vision's business plan calls for launching a satellite every 6 months or so, with a resolution down to 100 meters. In time, Hewins says, the company will operate a fleet of spacecraft "that can watch everything at once—eventually globally."

Fritz Hasler, a research meteorologist at NASA's Goddard Space Flight Center (GSFC) in Greenbelt, Maryland, says the Astro Vision approach could provide researchers greater flexibility than the GOES system, which must choose between viewing Earth from a wide angle or doing rapid scans of a smaller area. "Basically, we can only do one or the other," says Raymond



CREDIT: NASA/GSFC

Eyeful. Astro Vision satellites would provide clear view of storms like Hurricane Luis that struck the Caribbean in 1995.

ScienceScope

Academic Freedom Japan's leading university has cautiously endorsed a government proposal to cut loose the country's 98 national universities (*Science*, 13 August 1999, p. 997). A University of Tokyo panel has declared that "denationalization ... could help invigorate research and education" by freeing universities from regulations on administrative matters. However, it says, serious questions remain about whether funding cuts are the price of freedom—or if the government really can let go.

The panel's stance is expected to influence the academic community during negotiations. The education ministry hopes to submit a plan to the Diet by late spring, for implementation no earlier than 2002.

Chimp Deaths The Coulston Foundation is in hot water again. According to allegations made by In Defense of Animals (IDA), six chimpanzees have died at the primate lab since last August, when Coulston and the U.S. Department of Agriculture (USDA) announced an agreement that resolved charges of animal-care infractions (*Science*, 10 September 1999, p. 1649). In its latest broadside, IDA asserts that a chimp named Donna died of an infection after carrying a dead fetus in her womb for at least 2 weeks. A USDA report from December notes that the causes of several other deaths were "not fully determined."

Coulston's Don McKinney says the foundation is formulating its response to the USDA report. As for Donna, he says, Coulston vets had delayed surgery to remove the fetus in order to allow her to gain strength after failed attempts to induce delivery. The NIH and USDA are investigating. Coulston could face sanctions if found negligent.

Numbers Crunched Indian Prime Minister A. B. Vajpayee's promise to boost research spending sharply by tying it to the country's GDP—initially 1% and rising to 2% over 5 years—assumed that the current spending level is 0.86% of GDP (*Science*, 14 January, p. 209). But the actual figure is 0.66%, Indian officials now admit.

The revision means the government must come up with an additional $500 million—and a total increase of $1.25 billion over a current $2.5 billion budget—to meet the PM's pledge in the 2000 budget. Although a sudden rise of such proportions is unlikely, the head of the Department of Science and Technology says it should be possible to reach the 2% figure in 5 years.

Contributors: Michael Baker, Andrew Lawler, Laura Helmuth, Dennis Normile, Gretchen Vogel, Pallava Bagla

Steiner, a GOES operations specialist. And because GOES serves a broad community, it has fewer opportunities to do rapid scans for individual researchers. Although NOAA would like an additional GOES satellite that can be devoted to performing rapid scans of storm fronts and hurricanes, there is no budget for it.

In addition, Brannon says Astro Vision's narrow-angle camera will give researchers a better view—at half-kilometer resolution versus GOES's 1-kilometer resolution—of the tops of storm cells that can produce funnel clouds, and potentially provide up to 15 minutes of additional warning of an impending tornado. Ghassem Asrar, chief of NASA's earth science office, predicts that the agency may be able to do some of its research more cheaply "by purchasing data upon delivery from the private sector instead of developing, building, and launching new satellites."

Not everyone is so impressed, however. Dennis McCarthy, the meteorologist in charge of the National Weather Service's Norman, Oklahoma, office, says that a combination of Doppler radar and GOES imagery can, in some cases, already provide forecasters with plenty of warning of a tornado. And he adds that rapid scans—about one a minute—by GOES "already let us see where the storms are developing."

Hewins insists that Astro Vision is not merely dedicated to providing data to the government. "We're a video-coverage company, providing entertainment," he says, noting that television executives "go wild over this." He anticipates that media, combined with Internet companies such as America Online and Microsoft, will account for roughly two-thirds of their business, with government agencies providing the rest. If so, it could prove a case of science leading to an entertainment spin-off.

–ANDREW LAWLER

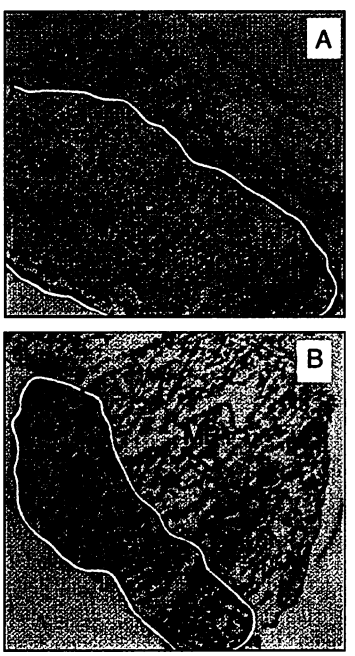
IMMUNOLOGY

Embryos Attacked by Mom's Natural Defenses

Ever since the 1950s when Nobel Prize–winning immunologist Peter Medawar first likened the mammalian embryo to a tissue transplant received from a genetically different individual, immunologists have been puzzled by a basic conundrum: How do we manage to procreate? Why doesn't the mother's immune system regard the prospective Dad's share of the embryo as foreign tissue and reject it? Work reported in this issue may help answer that question.

Immunologists trying to tackle this paradox have focused on identifying factors that might suppress the so-called acquired branch of the body's immune system, which produces T and B cells directed at very specific target antigens. Although they have found some clues, a unifying picture is still elusive. The new findings, which come from rheumatologist Hector Molina and his colleagues at Washington University in St. Louis, Missouri, and appear on page 498, for the first time point to suppression of the immune system's innate branch, which is evolutionarily older and far less specific, as key to embryonic survival.

The Molina team has found that Crry, a cell surface protein that suppresses a key part of innate immunity called the complement system, is necessary for the embryo to survive pregnancy, at least in mice. The complement system is a group of proteins usually activated during an inflammatory response triggered by foreign invaders. "Although the bulk of immunologists still believe in acquired immunity as playing a major role during pregnancy, this is beginning to confirm the importance of the innate immune system," says reproductive immunologist Charles Loke of the University of Cambridge in the United Kingdom.



Complement attack. Compared to the normal placenta *(top)*, the fetal placenta (FP) of a *Crry* knockout *(bottom)* is reduced, and both the FP and maternal placenta (MP) show brown staining for complement.

It remains to be seen whether human embryos use a similar trick to evade a complement attack, but Loke and others think that that is highly likely. Indeed, Christopher Holmes of the University of Bristol in the United Kingdom, who proposed a complement-mediated mechanism for fetal survival in humans about 10 years ago, says it may be interesting to see "if complement inhibition could be a way of preventing miscarriages and spontaneous abortions," which may end as many as 50% of all conceptions.

Molina did not set out to study the role of complement in embryonic survival. Instead, he wanted to test whether Crry helps protect the body's own cells against damage during inflammation. He and his colleagues inactivated the *Crry* gene in mice, expecting to see little effect as there are numerous complement regulators that might help out if one of them is missing. But to Molina's surprise, knocking out the *Crry* gene had drastic consequences: None of the *Crry*-deficient embryos made it through pregnancy. They all died in the uterus about 10 days after conception.

In healthy mouse embryos, Crry is the only complement regulator the team found in abundance on fetal cells called trophoblasts that form part of the placenta, the interface between mother and embryo. So Molina hypothesized that "in the absence of Crry there may be an excessive complement deposition on these cells." That turned out to be the case: The researchers found activated complement on trophoblasts of *Crry*-deficient embryos, but not on those from normal embryos. What's more, the knockout embryos showed a massive invasion of inflammatory cells, again in striking contrast to normal embryos. If the mothers were complement-deficient, however, they gave birth to essentially normal pups, further confirming that abnormal complement activation killed the *Crry*-deficient embryos.

The picture that emerges, says Molina, is that "a lack of Crry on the trophoblasts leads to complement activation that, in turn, attracts and activates inflammatory cells. Because these cells lack specificity, they destroy the trophoblasts and, ultimately, the embryo."

That makes sense to immunologist Polly Matzinger of the National Institute of Allergy and Infectious Diseases in Bethesda, Maryland. "An internally grown fetus is no different from any other tissue [in the body] in that it has to protect itself from complement, which is amazingly dangerous stuff. And it does this by expressing a potent complement regulator," she says.

The big question now, though, is whether something similar happens in humans. "There are several examples of fetomaternal tolerance mechanisms that are very different between humans and mice, because a human pregnancy is quite a bit more complicated," cautions Susan Fisher, a reproductive biologist at the University of California, San Francisco.

One difference is that humans don't have a *Crry* gene. But, explains Holmes, two other complement regulators "are present in huge quantities at the fetal-maternal interface to form a barrier of sorts" and could perform *Crry*'s vital job in humans. In fact the issue is next on Molina's agenda; he plans to tackle it by looking at complement regulators in women suffering from spontaneous miscarriages.

–MICHAEL HAGMANN

CREDIT: XU ET AL.