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

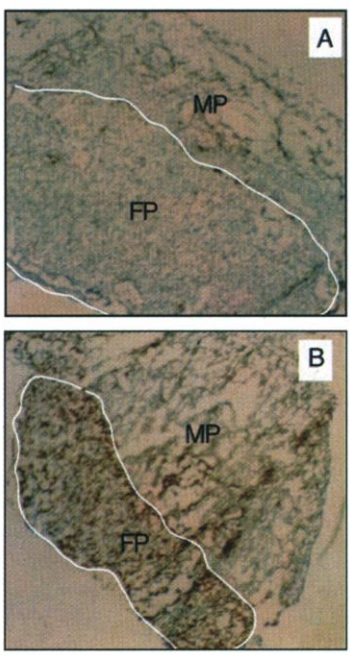
IMMUNOLOGY

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

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