what he called "a blow" to those disciplines and Walker explaining that it was simply "proper management" in a time of fiscal austerity. The House did reject, by a vote of 339 to 59, a Science Committee–passed measure to change NSF's name to the National Science and Engineering Foundation.

Walker's bill also proposed a whopping $373 million cut in NASA's EOS effort, eliminating the Chem spacecraft, reducing funding for two others, and cutting the budget for the data system in half. The proposed cut was fought unsuccessfully by Democrats. Leading the attack on this and other measures was Representative George Brown (D–CA), who said the bill was "antiscience." However, in a rare show of bipartisan support, members of both parties voted 287 to 126 to continue authorizing $2.1 billion a year for the space station.

The noise level during the debate over this reauthorization bill may, however, be in inverse proportion to its chances of becoming law. Although Walker and the Republicans had enough votes to win House approval for the bill, the Senate is unlikely to consider 1997 authorizations for science agencies. The crush of other business will probably shunt the bill into a legislative siding, and in any case, many senators prefer to work through the Appropriations Committee. Moreover, Vice President Al Gore, who attacked the bill as "extreme legislation that would make unnecessary and unwise cuts," said he would recommend that President Clinton veto the measure if it reaches his desk.

All of this may foil Walker's wish to leave behind a new blueprint for science when he retires in January. But come what may, Congress is certain to approve a 1997 budget, and this first glimpse suggests that lawmakers, despite the occasional heated rhetoric, are searching for a middle ground.

–Andrew Lawler

With reporting by Jeffrey Mervis and Jocelyn Kaiser.

INFECTIOUS DISEASES

Malaria Hideout Found in New Mothers

Malaria has many dangerous and lethal tricks, but one is especially grim: attacking first-time mothers. Although repeated exposure to the disease brings some measure of immunity to people who live in malaria-infested areas, women often lose part of those defenses at the worst possible moment, during their first pregnancy. And while it's the mothers who come down with the disease, the fetus bears the brunt of the infection. In sub-Saharan Africa, it's linked to maternal anemia and low-birth-weight babies, who have an increased risk of subsequent disease and death. "Malaria in pregnancy is a problem of enormous importance," says Thomas Wellems, head of malarial genetics at the U.S. National Institute of Allergy and Infectious Diseases (NIAID). And until recently, it's been a puzzle without an answer in sight.

Now, however, a possible solution is emerging in a tissue that only pregnant women have: the placenta. Red blood cells infected by the malaria parasite produce surface proteins that allow them to bind to particular tissues in the body. And on page 1502, scientists report finding a specialized subgroup of infected blood cells that can hide out on the placental walls. Because cells bearing the receptor protein needed to bind to the placenta are an uncommon phenotype that occurs only in large numbers during pregnancy, even women who are normally immune to malaria lack the immune defenses needed to attack them.

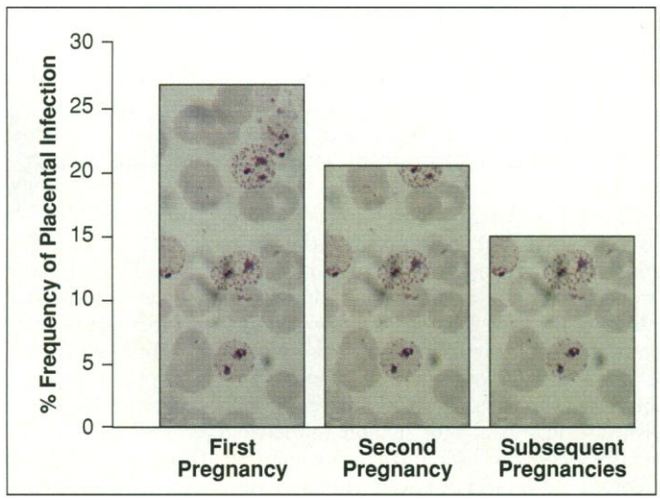
Malaria researchers welcome the new work, which was carried out by Michal Fried and Patrick Duffy at the U.S. Army Medical Research Unit–Kisumu, Kenya, and the Kenya Medical Research Institute, Kisumu. "It's the first fresh idea on the problem we've had in ages," says Louis Miller, chief of the Laboratory of Parasitic Diseases at the NIAID. Not only does it offer an explanation for this immunological mystery, but Miller and others say that the discovery that infected red blood cells bind to a particular receptor on the placental cells—a protein known as chondroitin sulfate A (CSA)—opens up new therapeutic approaches. A treatment that blocked the infected cells' ability to bind to CSA, for instance, could be an effective check on the disease, although scientists caution that they need to learn much more about the molecular interactions involved before such a check is developed.

Malaria parasites—the most serious form that infects humans is called *Plasmodium falciparum*—are transmitted by the bite of blood-feeding mosquitoes. The parasites grow and reproduce in red blood cells until the cells are filled to the bursting point. When the cells rupture, the parasites flood out and infect new cells. If the body has been exposed to the parasites before, it can counterattack: Antibodies detect affected cells that are circulating in the bloodstream and mark them for subsequent destruction in the spleen.

This scenario, however, couldn't explain the presence of infected red blood cells in the placentas of pregnant women who had already built up immunity to malaria, says malaria researcher Kevin Marsh of the Kenya Medical Research Institute in Kilifi, near Mombasa. "No one has understood why the parasites are there," he says. "Many researchers have thought it may be the result of changes in the immune status of mothers which occur during pregnancy," he adds. But because mothers actually become much less susceptible to malaria after their first or second pregnancy (see chart), pregnancy itself seemed unlikely to have lowered the mothers' defenses.

The groundwork for a different answer was laid last year. Three groups reported that malaria parasites have between 50 and 150 genes from a large family, known as *var* genes, that encode proteins that bind to receptors on various host cell surface molecules (*Cell*, vol. 82, p. 77, 1995). This diversity of *var* genes suggested that the parasites had an enormous repertoire of potential host binding sites, says Wellems, enabling them to hide out in a variety of tissues and present a variety of antigens in an effort to beat the immune system. Just such a "sequestration" mechanism, in fact, has long been thought to be behind cerebral malaria, where infected cells bind to blood-vessel walls within the brain.

Fried and Duffy, who were working with women from the malaria-endemic area of western Kenya, wondered if parasite-infected cells might have some particular mechanism for binding to the placenta. This could explain infection in newly pregnant women: Cells capable of



Vulnerable victims. Malaria parasites (shown in graph) most often infect the placenta during first pregnancies of Kenyan mothers.

anchoring to the placenta would have novel surface proteins that the woman's immune system had never "seen" before, and hence could not attack. By hiding out in the placenta, these cells could provide a persistent source of infection.

To find out whether infected cells really can home in on the placenta, the duo measured the ability of infected cells to bind to frozen sections of placentas from uninfected women. "We found that the infected cells bound to the sections in a pattern similar to that found with parasites bound in naturally infected placentas," says Duffy. Then the researchers tried to block this binding by adding different proteins to the infected cells. The only one that could do so was CSA, a well-known extracellular matrix protein. Last year, groups at the Pasteur Institute in France and the Walter and Eliza Hall Institute in Australia discovered that it acts as a receptor for parasite-infected cells. And in the current experiments, Fried and Duffy's work suggested, CSA apparently preempted the highly specific parasitic receptor proteins on the surface of the infected cells, suggesting that it was the target protein on placental cells.

And that specificity made these placental cells rather different from parasite-infected blood cells in nonpregnant women. In spite of the diversity of *var* genes, previous studies have found that most circulating infected cells bind to an endothelial cell surface molecule called CD36. Duffy and Fried found that, outside the placenta, the blood of infected pregnant women contained a mixture of parasitized cells that could bind either CD36 or CSA. But in nonpregnant women, they found that parasitized cells only bound to CD36. The results suggest that the parasites are able to exploit the opportunities presented by pregnancy. "It appears that the placental cells are a distinct subpopulation with a distinct binding phenotype," says Duffy. After these cells appear during a first pregnancy, the immune system may gradually learn to recognize them, so that it mounts a stronger response in subsequent pregnancies, he says.

The puzzle isn't completely solved, however: CSA is found widely in the body, so it's odd that infected cells bind to it only in the placenta. Duffy and Fried suggest that the tissue may be the only site where the protein can interact most effectively—through a presently unknown process—with red blood cells.

Nonetheless, researchers think these findings add to the potential new targets for therapeutic approaches to serious manifestations of the disease, says Wellems. In cerebral malaria, for instance, the infected cells may bind to a molecule called ICAM-1, which is expressed on blood-vessel walls within the brain. Blocking that target could help modify the course of the disease. Similarly, armed with knowledge about new targets such as CSA and the *var* genes that encode parasite receptor-binding molecules, researchers think it might be feasible to develop a drug treatment for pregnant women that would block the ability of infected red blood cells to grow in the placenta; this would lessen the damage to the fetus. First, however, they'll need to learn more about the interactions between CSA and the parasites.

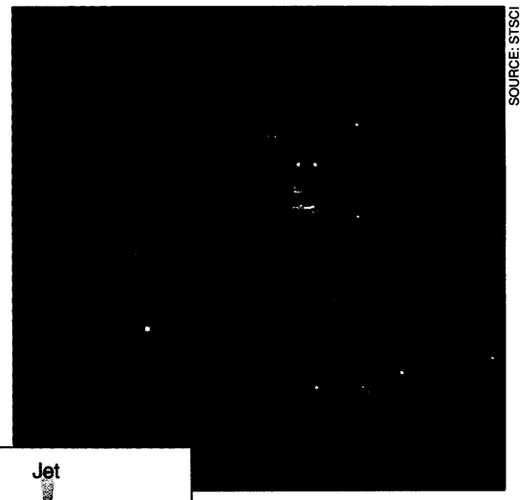
Although a treatment along these lines wouldn't cure pregnant women (who would still have to cope with the larger population of parasites which have other types of binding molecules), it could have a major impact on the healthy development of babies in malaria-prone countries. "Up to 40% of recorded low birth weight in these areas may result from maternal malaria," says Bernard Brabin of the Liverpool School of Tropical Medicine. "It's a high priority for control." And with a better idea of where and how the parasites are hiding, scientists now have a better chance of flushing them out.

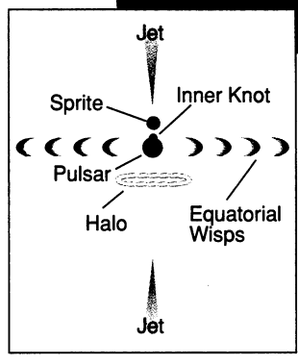
–Nigel Williams

ASTRONOMY

Movie Captures Dance of the Crab

Although nearly 1000 years have passed since Chinese astronomers recorded the supernova explosion that created the Crab Nebula, this cosmic dynamo still holds surprises. Over the past year, astronomers used NASA's Hubble Space Telescope to make time-lapse images of the Crab, creating a movie that reveals week-by-week changes in the glowing gases. The Crab shows "an awful lot more [going on] than anyone imagined," says Arizona State University (ASU) astronomer Jeff Hester, the team leader. The movie also poses a challenge to previous ideas about how the nebula is energized by the neutron star at its heart. "Ten years down the pike this will be the thing that sparked a lot of research," says Anne Kinney of the Space Telescope Science Institute in Baltimore.





Scene from a movie. Waves of gas ripple outward along the equatorial plane of the Crab pulsar *(above)*. The complete image series revealed other features *(left)*.

The nebula, nearly 7000 light-years away, is a thin cloud of debris from the exploded star's outer layers; the spinning, magnetized neutron star, or pulsar, at its center is what remains of the star's core. The pulsar, a Manhattan-sized lump of matter with a density of about a billion tons per teaspoon, flings electrons and positrons outward along its whirling magnetic lines of force. Astronomers had thought that these particles would be cast outward in all directions. But by combining images made every few weeks over a period of months, the Hubble researchers found that the emission is confined to two regions. At the pulsar's equatorial plane, waves of ionized gas ripple outward at half the speed of light, while its south pole emits a wisplike jet of gas and dust. (Another jet probably streams from the north pole, which is blocked from view.) A bright, shifting shock wave forms where the polar jet runs into quieter parts of the nebula.

The Crab watchers themselves don't have a good explanation for these antics. One early hypothesis for the equatorial waves is that the charged particles clinging to the magnetic field lines only break free when they reach the speed of light, which happens most often at the equatorial plane, says ASU astronomer Paul Scowen. At the poles, the field lines get twisted up as the star spins, creating a funnel that shoots out particles. Scowen likens the effect to the behavior of an overwound clock spring, which snaps violently, relieving the tension.

But that's as far as the team's explanations go; the observers hopes that after watching the movie, theorists will take the next step. Says Hester, "From here on out, we've thrown down the gauntlet."

–Kim Peterson