lines, accounting for the magnetic and particle oscillations detected beyond Earth. Lending support to the idea that motions at the sun's surface can set up long-range oscillations in the solar wind, Jokipii has just reported that the Ulysses spacecraft, during its pass over the southern pole of the sun, seems to have picked up long-period magnetic oscillations. What drives them, he thinks, is the jiggling of magnetic field lines by gas rising and sinking in the polar regions of the sun.

But even though the Thomson group's claim has stood up to all scrutiny so far, many solar physicists are cautiously waiting for independent confirmation. "Thomson apparently has a very good reputation as an analyzer of time series," explains Jokipii, "but most of us don't have the background to understand really how he did it. That's where the skepticism comes from." Researchers are also wary because the implications of the claims are so large.

If the oscillations are real, for example, it would alter the way solar wind specialists calculate the migration of charged particles through the solar system, says Lanzerotti. And if the g modes are real, it will be a boon to solar physicists. Coming from the sun's energy-producing core, g modes could carry clues to the apparent neutrino shortage. Many physicists suspect that the missing neutrinos might somehow be evading detection. But by studying the strength and timing of the modes, solar physicists could test an alternative idea: that the problem lies in researchers' understanding of the solar core, leading them to expect more neutrinos from fusion there than are actually produced. The exact nature of the g modes might be part of the explanation: By mixing the hydrogen fuel and the helium ash of fusion, they might change the workings of the core in a way that could account for the shortage.

The solar wind may soon have some competition as a potential source of these clues. The Global Oscillation Network Group, a global network of sophisticated telescopes dedicated to helioseismology, already has three of its six sites up and running. They should get better-than-ever data on p modes and intensify the effort to detect g modes directly. And the Solar and Heliospheric Observatory is due for launch in November with three helioseismology instruments on board. But solar wind data are already there for the taking, notes Gough. "There may be a lot of people around the world," he says, "who have the information on their computers and don't know it."

–Richard A. Kerr

IMMUNOLOGY

How the T_H2 Response Is Marshaled

Different enemies sometimes require different weapons. That lesson applies to the immune system as well as to warfare. While the immune system faces some pathogens that have invaded cells, it must also combat others that have yet to enter a cell or that do their damage strictly from the outside. To attack internal pathogens, the immune system relies heavily on a battalion of cells known collectively as "cell-mediated immunity," which can identify infected cells and clear them. On the other hand, to fight invaders that are, say, floating in the bloodstream, the weapon of choice is antibodies.

While this general picture has been understood for some time, recently it has been refined by the remarkable finding that, in this war, two sets of immune cells are the equivalent of field marshals—and are powerful enough to lock each other in the barracks if they so choose, keeping one of the two classes of immunological weaponry off the field of battle. While the immune system typically calls out both battalions, this cross-regulatory system allows for a finely tuned attack, and in some extreme cases, for unknown reasons, it favors one weapon exclusively. Now researchers at Columbia University and DNAX Research Institute of Palo Alto, California, have teamed up to elucidate how it is decided which field marshal gets to write the battle plan.

As described in a report on page 245, Columbia University's Paul Rothman, Alessandra Pernis, and co-workers investigated the molecular workings of these two field marshals—white blood cells known as types 1 and 2 T helpers, or T_H1 and T_H2. The researchers found that the absence of a specific molecule on the surface of T_H1 cells helps them gain the upper hand over T_H2 cells. This finding may ultimately help to explain why the troops that make up cell-mediated immunity get called into action rather than antibodies. "It's a very nicely done piece of work that will have substantial implications," says National Institutes of Health (NIH) immunologist William Paul. Paul adds that this work fits in nicely with a similar finding about the molecular workings of T_H2 dominance published by Kenneth Murphy of the Washington University School of Medicine and colleagues in the June issue of *Immunity*.

The current study focuses on the fact that T_H1 cells secrete a chemical messenger called interferon γ (IFN-γ), which in turn stimulates production of cell-mediated immunity. IFN-γ also inhibits T_H2 cells, which direct antibody production. The researchers explored the molecular basis for this and asked why IFN-γ does not shut down proliferation of T_H1 cells.

"It's a very nicely done piece of work that will have substantial implications."

—William Paul

They focused on a cascade of events triggered by IFN-γ that allows T_H1 cells to emerge as supreme commander. The cascade includes several "signal transducing factors" (STFs), chemicals that pass a signal received at the cell surface to the cell nucleus. In this case, the STFs shut down the ability of T_H2 cells—but not of T_H1 cells—to proliferate. What the Rothman group found was that IFN-γ induced T_H2 clones (derived from mice) to produce a protein known as STF-IFNγ, the final messenger in a signal's journey to the nucleus. IFN-γ, however, had no such effect on T_H1 clones.

Through a complicated series of experiments on the roles of different STFs, the researchers deduced that the key difference between the T_H1 and T_H2 responses lay in the receptors for IFN-γ on the surfaces of the two different cell types. The full IFN-γ receptor consists of two chains, designated IFN-γRα and AF-1; the Rothman team discovered that while both clones have the IFN-γRα chain, only the T_H2 cells have the AF-1 chain. They further showed that genetically engineering the AF-1 chain into T_H1 clones led them to produce STF-IFNγ. This system apparently allows T_H1 cells to shut down reproduction of their T_H2 rivals while remaining unaffected themselves.

Although some researchers wonder what relevance this in vitro finding will have to living organisms, Rothman thinks the finding may well have clinical applications. He suggests it could help researchers determine whether a person afflicted by an autoimmune or infectious disease has mounted primarily a T_H1 or T_H2 immune response. And that could be a critical diagnostic tool for, say, leishmaniasis, a devastating skin disease that can be controlled by a T_H1 response. Currently, distinguishing between T_H1 and T_H2 is a cumbersome process that involves growing clones of different cell populations.

Whether the work yields practical applications likely will depend on whether Rothman, Pernis, and others can further illuminate the workings of T_H1 and T_H2 cells. Still, this study is shining a light on the immune system's military strategy—a strategy that a mere decade ago was a black box.

–Jon Cohen