Astronomy

From a meeting in Baltimore of the American Geophysical Union

Leonids: The coming storm

Earth is poised to plunge headlong into the most intense interplanetary dust storm it has encountered in 33 years.

A souped-up version of the annual Leonid meteor shower, this storm won't harm Earthlings when it strikes in late 1998 and again in 1999. However, satellites could suffer significant, possibly fatal, electrical damage, says Peter Brown of the University of Western Ontario in London, Ontario.

Leonid meteor showers occur each November, when Earth plows through a broad, tenuous band of dusty debris, or meteoroids, expelled by Comet Temple-Tuttle during centuries of passages near the sun. As the debris burns up in Earth's atmosphere, some 100 meteors an hour may grace the skies.

A Leonid storm occurs once every 33 years, when Earth passes through the meteoroid stream shortly after Temple-Tuttle has neared the sun and spewed fresh particles. On Nov. 17, 1998, Earth will hit the Leonid stream just 9 months after the comet has passed closest to the sun. In that short interval, the torrent of new meteoroids won't have had time to spread out. Our planet will encounter a dense swath of debris, creating a veritable tempest.

At the storm's peak, which may last for just a few hours, observers in the right location—the Far East—should be treated to a terrific light show, with as many as 100,000 meteors an hour streaking through the sky. One year later, an encore performance should thrill skywatchers in Western Europe.

The dust particles are tiny, so chance collisions with spacecraft aren't the prime worry of scientists. Rather, researchers express concern about the potential of these particles to create localized clouds of electric charge, or plasma, that can penetrate satellites and short-circuit equipment.

The high speed of a Leonid meteoroid—about 72 kilometers per second, more than three times that of an average meteoroid—favors the production of clouds of charged material, notes Brown. These can generate lightninglike discharges inside satellites, zapping fragile electric components.

Another meteor storm, this one associated with a swath of cometary debris known as the Perseids, is credited with taking a satellite out of commission in 1993 (SN: 10/2/93, p. 217). However, the potential for damage is highly uncertain, notes Brown. Come 1998, "everyone is going to go through this test, whether they like it or not."

To study the Leonid storm and its parent comet at wavelengths including the ultraviolet, which are undetectable from the ground, Peter Jenniskens of NASA's Ames Research Center in Mountain View, Calif., is proposing to fly a pair of spacecraft during the 1998 and 1999 events. By flying in tandem, the craft should obtain the complete, three-dimensional orbit of the debris. They may also provide a view of a special class of clouds, predicted to condense high in the atmosphere around electrified dust grains from the storm.

Set to take place on a moonless night, the 1998 storm is likely to generate a spectacular light show. The 1999 event, however, promises to be a windfall for lunar astronomers. Encountering Earth during a full moon, the storm will provide researchers with a unique opportunity to observe what happens when meteoroids penetrate the thin lunar atmosphere and pummel the moon's surface. Astronomers have conjectured that meteoroids striking the lunar surface generate the sodium in the moon's atmosphere. —*R.C.*

Leonid meteor storm as seen in 1799.

Biology

HIV may spare cells—for a short time

While the AIDS virus is best known for destroying the body's immune system by killing its cells, HIV may also deliberately delay the death of immune cells, according to a new report.

Like all viruses, HIV depends on host cells to make its offspring. If it kills infected cells too quickly, HIV may not have time to replicate. Indeed, part of the body's defensive strategy may be to sacrifice infected cells. "We have billions of T cells [the cells targeted by HIV]. Losing a few hundred thousand doesn't mean too much," says David Kaplan of Case Western Reserve University in Cleveland.

Kaplan suspects that HIV can buy itself some time, however. The body has a system for regularly ridding itself of unwanted immune cells: When Fas, a protein found on the surface of immune cells, binds to Fas ligand (FasL), another cell surface protein, the Fas-bearing cell commits suicide. It's as if FasL were the finger pushing a cell's self-destruct button.

Kaplan's group has found that immune cells from the blood of HIV-infected people have a FasL deficiency, compared to similar cells from uninfected people. Indeed, cells from three of six HIV-positive individuals showed no FasL activity at all in a test-tube study, the team reports in the May 27 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

This was a surprise. Earlier studies had shown that Fas was made more abundantly by HIV-infected people, leading some scientists to speculate that HIV killed immune cells via the Fas-FasL system. Instead, HIV seems to interfere with this cell suicide mechanism, presumably to keep infected cells alive long enough for the virus to replicate, says Kaplan.

In studies of immune cells from HIV-positive people, antibodies that trigger Fas-induced suicide decreased HIV replication by 90 percent, the scientists report. Compounds similar to FasL might therefore prove medically useful, though Kaplan and other researchers caution that such drugs could also damage nonimmune Fas-bearing cells, such as those in the liver. "This approach has some distant therapeutic potential, but nothing immediate," says Warner Greene of the Gladstone Institute of Virology and Immunology in San Francisco. —*J.T.*

Stopping cellular traffic in pregnancy

The cell-death-inducing Fas ligand (FasL) has also emerged in another arena. Cells in the uterus and placenta of pregnant mice make FasL, report Joan S. Hunt of the University of Kansas Medical Center in Kansas City and her colleagues in the May 1 JOURNAL OF IMMUNOLOGY. They don't know why yet, but FasL may prevent potentially dangerous maternal immune cells from reaching a fetus and vice versa. "It looks like it's a general prohibiter of trafficking cells," says Hunt.

Her research group has also found that mice with mutant FasL genes seem to have reduced fertility. —*J.T.*

A bacterium that munches on solvents

Just as few kids like to eat vegetables, not many bacteria favor a diet of chlorinated solvents like perchloroethylene (PCE) or trichloroethylene (TCE). Such solvents, used to clean clothes, machines, electronic parts, and more, are major groundwater contaminants. Scientists have long hunted for bacteria that could digest these suspected carcinogens.

Stephen H. Zinder of Cornell University and his colleagues have now isolated a bacterium that seems to thrive on PCE and TCE. While previously identified microbes can degrade these solvents to less dangerous compounds, the new bacterium converts them to the nontoxic gas ethene, the researchers report in the June 6 SCIENCE. A genetic analysis of the solvent-loving bacterium reveals that it's a novel microbe. "It doesn't seem related to much of anything, and we know little about the distribution of this organism," says Zinder —*J.T.*