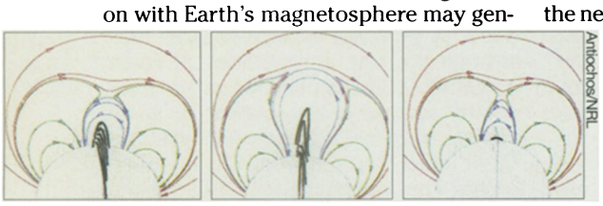
Deepening insight into solar outbursts

Imagine billions of tons of gas erupting from the sun's outer atmosphere at speeds greater than 1,250 kilometers per second. These huge upheavals, which are becoming more frequent as the sun enters the most active period of its 11-year cycle, can wreak havoc on Earth.

This type of solar outburst, known as a coronal mass ejection (CME), accelerates interplanetary protons to speeds that enable them to penetrate spacecraft and cripple electronic equipment. A cloud of coronal material colliding head-on with Earth's magnetosphere may generate geomagnetic storms that disrupt communication systems and create large-scale power outages.



From left to right, simulation depicts the emergence of a coronal mass ejection. Blue lines represent a magnetic field that hasn't yet broken loose from the sun's lower corona; black lines show an associated blob of gas. Red and green depict the overlying fields that restrain the gas and field. As energy builds up in the agitating field, it pushes upward. After the restraining fields vanish, the field and gas blob erupt.

Relying on computer simulations and new data from the SOHO spacecraft, researchers report that they have developed a deeper understanding of the magnetic forces within the sun that create the fastest, most damaging upheavals. That knowledge, says SOHO investigator Spiro K. Antiochos of the Naval Research Laboratory in Washington, D.C., could lead to better predictions of these catastrophic events.

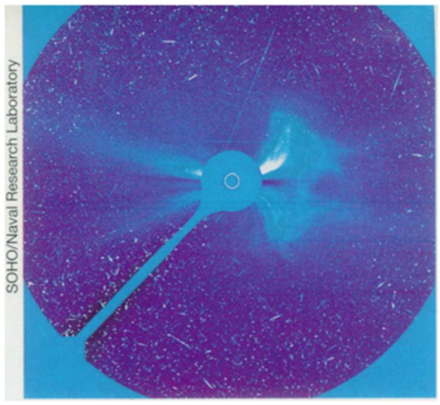
He described the findings last week at the fall meeting of the American Geophysical Union in San Francisco.

Antiochos began his investigation after viewing SOHO images of high-speed outbursts. In addition to showing magnetized parcels of electrically charged gas emerging from the sun's outer atmosphere, or corona, these images also reveal smaller, neighboring gas blobs threaded by magnetic fields. The smaller blobs remain stationary. Antiochos' computer simulations suggest that the neighbors are not innocent bystanders, as researchers had supposed, but signposts of a magnetic interaction that forces tremendous amounts of energy into some eruptions.

Comparing a coronal ejection to an inflating balloon, Antiochos finds evidence that the magnetic architecture lying above this rising package of gas and intense magnetic field weighs it down. While it remains trapped, the magnetic field soaks up huge amounts of energy and begins expanding upward. Eventually, the overlying fields can no longer contain the swelling gas. The whole kit and caboodle—the magnetized gas and the fields above it—typically moves slowly outward, carried by the sun's wind of charged particles, but under some circumstances, says Antiochos, something more dramatic occurs.

If the magnetic fields associated with the neighboring parcels of gas have an orientation opposing that of the overlying fields, they can merge and cancel each other out in a process known as magnetic reconnection. Then, all restraints on the rising blob of gas abruptly disappear. In this model, coronal outbursts can reach enormous speeds, exemplified by an eruption seen by SOHO on Nov. 6.

The model is important, says SOHO science operations coordinator Piet C. Martens, based at NASA's Goddard Space Flight Center in Greenbelt, Md., because "if you can recognize the magnetic structure that will soon be broken up and will give rise to a CME, you have a means of predicting roughly when it's going to happen." —*R. Cowen*



The sun's corona on Nov. 6, after a mass ejection. Traveling 1,585 kilometers per second, this eruption is the fastest detected to date in this solar cycle. White dots and streaks are protons jazzed by the outburst. Circle indicates the sun's disk.

Let's repeat: Mutation gums up brain cells

In most genetic disorders, the function of the gene that's mutated determines the symptoms of the disease. For the unusual kind of mutation that causes Huntington's disease and several similar brain disorders, however, the identity of the gene affected may not matter much.

That's the message emerging from studies of mutant mice created by Peter J. Detloff of the University of Alabama at Birmingham and his colleagues.

The researchers investigate disorders caused when a brief DNA sequence known as a CAG repeat occurs an abnormally large number of times in a gene's usual sequence. A half dozen or so neurodegenerative diseases stem from an excess of CAG repeats. Huntington's disease, for example, results when a certain gene harbors about 40 or more CAG repeats (SN: 6/10/95, p. 360).

Excess CAG repeats in a given gene add extra copies of the amino acid glutamine to the protein encoded by the gene. Detloff and other investigators have wondered whether almost any protein burdened with additional glutamines would cause a neurodegenerative disease or whether CAG repeats do their damage only in the context of specific genes.

The scientists engineered a strain of mice that has 146 extra CAG repeats in a gene that encodes hypoxanthine phosphoribosyltransferase (HPRT), an enzyme used by all cells. "You produce a full-length HPRT protein plus the added glutamines," says Detloff. The gene is active in brain cells, but the scientists had no reason to suspect that mutations in it would cause any problems. Mice with a disabled copy of the gene are essentially normal, says Detloff.

The addition of CAG repeats produced a neurological disorder in the mice that resembles aspects of CAG repeat diseases in humans, the investigators report in the Dec. 12 CELL. As the altered mice age, they begin to suffer more seizures than normal mice. Moreover, while mice generally live 2 years or more, none of the mutant mice survived more than 53 weeks.

Finally, Detloff and his colleagues found that the glutamine-laden HPRT proteins accumulate abnormally, forming clumps in the nuclei of the animals' brain cells. Similar deposits occur in human CAG repeat disorders and may trigger brain cell dysfunction and eventual death (SN: 8/16/97, p. 102).

Curiously, the researchers did not find evidence of abnormal brain cell death in their mice. "You can get the symptoms of the disease before the cells are dying," says Richard M. Myers of Stanford University School of Medicine.

These findings bolster the belief that the illnesses caused by extra CAG repeats stem primarily from a mutated protein's additional glutamines, says Myers. Scientists suspect that the long glutamine stretch of one protein may stick to that of another, causing a gradual buildup into the clumps seen inside nuclei.

If this feature is common to all CAG repeat disorders, it raises the possibility that a single drug that inhibits the binding of glutamine stretches would offer a treatment for the illnesses, says Detloff. —*J. Travis*