Stress Plagues the Fastest Chips

It first came to light about five years ago. An insidious plague was killing integrated-circuit chips as they sat in storage. Defects — some as serious as the severing of metal circuit lines — were developing under normal storage conditions, sometimes without a chip ever having been used. Last week, Cornell University materials scientists published the first complete theory that explains on an atomic scale what is causing these chips to fail. The silent killer, the researchers say, is unrelieved stress.

Computer chips are fabricated at temperatures of about 400°C. The current chip-making process bonds aluminum-based, current-carrying circuit lines onto a substrate, usually silicon. As the finished chip cools to room temperature, its metal circuit lines attempt to contract. However, because they are anchored to a more massive base with a smaller thermal expansion/contraction rate, the lines never fully contract. Their unrelieved "desire" to shrink further creates a materials stress in the metal lines, according to the theory.

The first sign of this stress is the formation of voids — a cluster of several empty atomic sites — in the lattice structure of the lines' metal. They develop in the "boundary region" between metal grains (single crystals). As the metal attempts to further relieve its stress, layers of atoms begin peeling off around these voids and redepositing nearby. As more and more atoms leave, the microscopic voids at the grain boundaries grow into cavities.

"This is a thermally activated phenomenon," explains Che-Yu Li, who heads the research team. "At higher temperatures, it can happen within a few hundred hours. At room temperature, it can take a year or two." The important thing, he emphasizes, is that this type of failure can occur without currents passing through the chip.

In today's fastest chips, the metal lines are only about 1 micron wide. They also are about one grain wide, with single grains aligned in a row. It doesn't take long for small voids in the boundaries between the grains to grow enough that line-severing cracks form. And that is one reason why the highest-performance chips — those having the narrowest current-carrying lines — are most vulnerable, Li says.

Though this passive-cracking phenomenon has been linked since 1984 to stresses introduced during chip fabrication, the Cornell theory, published in the July 4 Applied Physics Letters, is the first to model the atomic behavior of chip lines under stress. Li says it also is the first to "predict its width dependence." Specifically, his data indicate that for each 10-fold reduction in circuit-line widths, the risk of stress-cracking failure will increase 1,000-fold.

Though their smaller size will make the next generation of chips significantly more vulnerable, current chips are already being affected. Notes Billy Livesay, a chip-reliability engineer at the Georgia Institute of Technology in Atlanta, "A particular microprocessor has had some failures in the past year that are bugging the industry: These things are cracking — either in service or on the shelf" — from the type of stress, he says, that Li has modeled.

In fact, the potential for stress cracking "is a well-recognized problem in the [chip] industry," says William Nix, a Stanford University materials scientist. Though engineers have been quietly tinkering with their chip-fabrication processes to solve the problem, "rarely if ever have they dealt with what the microscopic causes are," he says. That's why Nix believes the Cornell theory "is a good, solid, fundamental contribution." And its predictive capabilities, he adds, could guide engineers toward solutions to the problem.

What types of solutions? Working at Cornell's National Nanofabrication Facility, Li and his colleagues will be exploring the idea of altering metals used in the lines and developing more flexible bonds between the metal and its substrate.

— J. Raloff

Two for Phobos

An elaborate Soviet mission to photograph, land on, drill into and analyze the tiny Martian moon Phobos (SN: 6/18/88, p.392) got underway with the launching of two unmanned spacecraft on July 7 and 12. The multipurpose vehicles are expected to reach their Mars-circling orbits next Jan. 29 and Feb. 5 respectively, after which they are to send landers down to the satellite's surface, tentatively on April 7 and May 5.

The venture calls for each orbiter to deploy a "hopper" to make surface measurements at several locations, using spring-loaded legs to jump across the terrain from site to site. In addition, each orbiter will spend about 20 minutes moving low across the surface while zapping it with lasers and charged-particle beams, using instruments such as mass spectrometers to study the surface composition.

Several U.S. and European scientists are taking part in the mission, and the U.S. Deep Space Network will provide tracking data to help the Soviets navigate their craft during the tricky maneuvers near Phobos. The space vehicles are the first sent toward Mars since the U.S. Viking project in 1975, and the first from the Soviet Union since the Mars 7 craft took off in 1973. □

MS and monoclonals

Scientists have completed preliminary studies of an experimental, immune-suppressing therapy they hope may someday assist victims of autoimmune disorders. The researchers injected mouse monoclonal antibodies — highly purified, identical antibodies mass-produced inside engineered mouse cells — into eight patients with multiple sclerosis (MS). The antibodies interfered with the activity of two specific types of white blood cells that may play a role in the disease.

Studies suggest MS is an autoimmune disease in which certain of a person's own T-lymphocytes attack and damage the protective coating of nerve cells in the brain and spinal cord. But immune-suppressing drugs currently used to treat the disease indiscriminately affect "innocent" T-cells and other useful white blood cells called B-lymphocytes.

The new approach, by David A. Hafler and Howard L. Weiner at Brigham and Women's Hospital and their colleagues at the Dana-Farber Cancer Institute in Boston, seeks to attack T-4 and T-11 lymphocytes without damaging other immune-system cells.

The treatment produced no major side effects, the scientists report in the July 1 Journal of Immunology. It remains to be shown, however, that monoclonal antibodies can help MS sufferers. The disease runs an unpredictable course and has so far proved resistant to cure (SN: 8/22/87, p.120; 10/10/87, p.234).

Byron H. Waksman, vice president of research and medical programs at the New York City-based National Multiple Sclerosis Society, says he regards the results as "encouraging" although this specific approach "won't work in a durable way" because patients produce antibodies against the mouse antibody, thus wiping out further response to the treatment.

But evidence that the mouse antibodies elicited a genuine, specific suppressive effect, says Waksman, suggests that similar antibodies mass-produced in human cells might prove effective without prompting an anti-antibody response. □