the fuel rods and into the distant environment over millennia. The panel agrees that DOE has done a good job assessing such possible disruptions as earthquakes, volcanic eruptions, and nuclear reactions suddenly taking off, but notes that other assumptions "may be unduly optimistic." For example, the cladding that encases the enriched uranium rods and provides the first line of defense may not hold up as well as assumed. More lab work on the cladding's behavior under repository conditions is needed, says the report.

The behavior of the radioactive material once it leaks out, as it eventually must, is also unclear, says the report. More exploratory holes should be drilled into aquifers far from Yucca Mountain, where the radioactivity will ultimately spread, it suggests. The panel is especially concerned about the assumptions behind the repository's "hot" design, in which heat from the waste is supposed to keep temperatures well above boiling and thus initially keep out moisture that could corrode the rods. "We don't think anybody can model that convincingly," says Whipple. Such stubborn problems might be handled by making some conservative, simplifying assumptions, says Whipple, an approach DOE has yet to accept.

Van Luik says he's "a tad surprised at the amount of material they think we need to do." Some of the suggested work is already under way, he notes, and project staff are still debating the merits of a hot design. "This is not our final design, nor our final understanding of the site," he explains. But he's concerned by the fact that "the panel recommends that we do additional work that would extend us beyond our current schedule."

Kevin Crowley, staff director of the National Research Council's Board of Radioactive Waste Management in Washington, D.C., says DOE would be wise to take the panel's advice because its current schedule is unrealistic. The panel's emphasis on gathering more data and dealing with the intractable complexities, he adds, could be key to resolving the technical issues. "The DOE has some real challenges ahead," he warns.

–RICHARD A. KERR

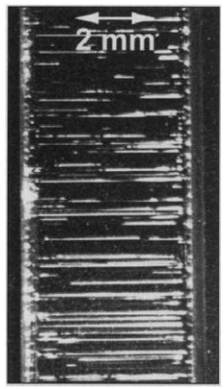
MATERIALS

Stress Profiling Gets The Best Out of Glass

Try to bend a piece of window glass, and you'll get a vivid demonstration of glass's brittle behavior. When stressed, it shatters without warning into thousands of shards. Now an international team of researchers has developed a clever way to make glass a little more pliant and predictable. On page 1295, materials scientist David Green of Pennsylvania State University in University Park and his team describe a chemical toughening process that resulted in glass that both resists fracture better and delivers a warning before it finally fails, in the form of small cracks on its surface.

"Usually, when a piece of glass starts to break, that's the end of the story. In this glass you can arrest the cracks and you get some warning before the final failure," says Green. "The fact that multiple cracking can be observed in glass is indeed remarkable," agrees William Tredway, advanced ceramics group manager at United Technologies Research Center in East Hartford, Connecticut.

The traditional method for making glass more resistant to fracture is called tempering. Manufacturers use either heat or chemicals to increase the "residual stress"—the compressive forces between atoms—at its surface. Before an external stress forms a crack, it must overcome not only the normal strength of the material, but also this extra residual stress. Tempered glass is more resistant to fracture, but when a crack does form at the surface it quickly moves deeper where the stress is lower, and the material fails catastrophically.



Flexiglass. Cracks form on a piece of treated glass as it is flexed, but it does not break.

In 1991, Green and Rajan Tandon, now at Caterpillar Inc. Technical Center, a construction machinery manufacturer in Peoria, Illinois, did theoretical studies that pointed to a better way to strengthen glass. The studies showed that a compressive stress "profile," with relatively weak stress at the surface increasing to a maximum at a depth of 20 to 30 micrometers, would stop cracks because they would face increasing compressive stress as they moved deeper into the material. "The idea went against the current dogma of what you are supposed to do," says Green. "Usually people try to get the maximum compression at the surface."

To create the required stress profile, the researchers developed a two-stage chemical tempering process. The main skeleton structure of glass is composed of silicon and oxygen atoms, interspersed with sodium atoms. The researchers immersed a glass sample in a bath of molten potassium nitrate at high temperature, allowing some of the potassium ions in the bath to swap places with sodium ions in the glass—a process called "ion exchange." Potassium atoms have a radius that is 25% larger than sodium, says team member Vincenzo Sglavo of the Uni-

CREDITS: (TOP) GREEN *ET AL.*; (BOTTOM) IOWA STATE UNIVERSITY

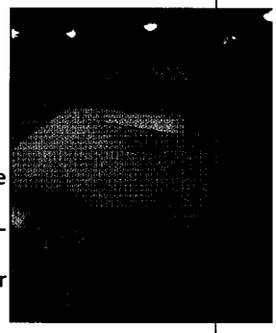
ScienceScope

Food Fight Fed up with the ongoing media feeding frenzy surrounding genetically modified (GM) food, 19 of Britain's most eminent scientists, all Fellows of the Royal Society, have called for the use of peer review rather than public opinion to judge scientific results. Writing in the 23 February issue of London's *Daily Telegraph* and *Guardian* newspapers, they say that "it is a dangerous mistake ... to assume that all statements claiming to be scientific can be taken at face value."

The letter follows a public statement from 21 scientists in defense of protein chemist Arpad Pusztai (*Science*, 19 February, p. 1094). Last summer, Pusztai was suspended from his post at the Rowett Research Institute near Aberdeen after declaring in a TV documentary that his unpublished research indicated that potatoes genetically altered to resist pests stunted growth and suppressed immunity in rats.

One signatory of the new letter, botanist Ghillean Prance, director of the Royal Botanical Gardens at Kew, says that the Fellows are concerned about several recent incidents in which unpublished results have been promoted in the media. "So much bad science is going into the press," he says. The society is convening an expert panel to review Pusztai's results and broader issues related to GM foods.

Meltdown Nuclear research reactors continue to disappear from U.S. university campuses as the field loses ground to other disciplines. Last month, Iowa State University in Ames finalized plans to dismantle by next year its 10-kilowatt minireactor, whose $200,000-a-year budget became an unjustifiable expense after the school closed its nuclear engineering program a few years ago.



The 40-year-old reactor (above) becomes the 43rd campus-based atomic plant to close since 1975, leaving about 30 operating academic reactors. Up to a third of the remaining plants could close within the next decade, experts say, due to operating cost concerns and relicensing expenses. Several advisory panels have called on the Department of Energy to stem the decline, which they say threatens a wide range of engineering, materials, and physics research.

Contributors: Daniel Clery and David Malakoff

versity of Trento in Italy. "This causes compressive stress in the material."

This first stage of the procedure is much like traditional chemical tempering. But in a new twist, says Sglavo, the researchers then reversed the direction of ion exchange. They briefly immersed the glass in a mixture of molten sodium and potassium nitrate. Some of the potassium ions migrated from the glass back out into the bath. The result was glass with a very thin surface layer containing sodium atoms and deeper layers richer in the potassium deposited by the first treatment. "Right at the surface some of the compressive stress is released," says Sglavo.

When the researchers tested their glass samples under increasing loads, they found that their strength was up to 5 times that of typical window glass. Sglavo reports that they could flex a 10-centimeter piece of glass by more than 1 centimeter in its center. "Usually you break it," he says. And when they flexed it, they observed small cracks forming on the convex surface. "This is the indication of a critical condition like you see in plastic or in metals," says Sglavo. Although glass tempered with such a stress profile would cost more than normal window glass, the researchers believe such a "safer" glass would be very valuable for certain purposes, such as car windscreens. The computer industry would also welcome thinner, stronger glass for light-weight displays, says Green.

–ALEXANDER HELLEMANS

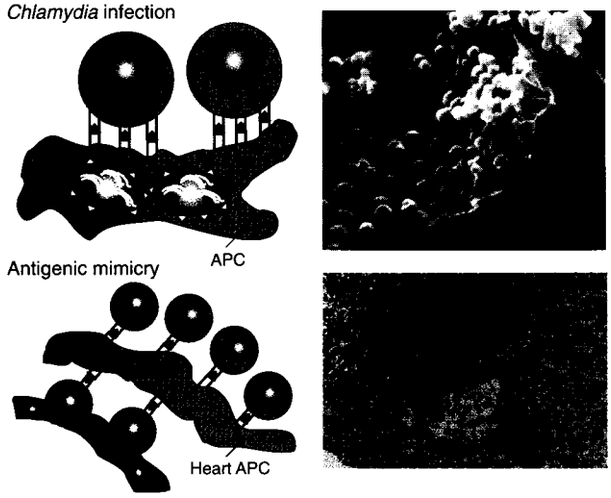
Alexander Hellemans is a writer in Naples, Italy.

IMMUNOLOGY

Chlamydia Protein Linked to Heart Disease

Nature has its share of copycats, which rely on deceit to escape predators: insects that look like the sticks they walk on, frogs disguised as leaves, harmless butterflies that model themselves after their poisonous cousins. Even microbes disguise themselves with proteins that mirror those of their host as a way of evading detection by the immune system. But such molecular mimicry may harm the host as well as protect the microbe by causing the immune defenders to mistakenly turn on the body's own tissue. Over the past year, investigators have implicated molecular mimicry in an eye disease and chronic Lyme arthritis, and now in one of the most common serious illnesses: heart disease.

On page 1335, a team led by immunologist Josef Penninger of the Ontario Cancer Institute and the Amgen Institute at the University of Toronto reports that the bacterial pathogen *Chlamydia* makes a peptide that mimics a portion of a heart muscle protein. In mice, the bacterial peptide can cause immune sentries known as T cells to attack the heart muscle, triggering a severe inflammation. If something similar occurs in human beings and the inflammation also plays a role in the formation of the artery-clogging plaques of atherosclerosis—two big ifs—the work may provide a molecular explanation for a long-suspected link between infections and heart disease.



Copycat. *Chlamydia* bacteria, seen upper right emerging from an infected cell, carry a peptide resembling one in heart myosin. As shown in the diagram, this peptide, when displayed by antigen-presenting cells (APC), can trigger T cells (pink) that attack both *Chlamydia* and heart cells, thus causing heart muscle inflammation *(lower right)*.

So far, the evidence for that link has been circumstantial: a stream of studies associating cardiovascular disease with infection by agents including *Chlamydia* (*Science*, 3 July 1998, p. 35), plus a report in the 3 February 1999 issue of the *Journal of the American Medical Association* that antibiotic use reduces the risk of heart attack. But researchers have had little idea about how infections might lead to heart problems. The new study, says epidemiologist Hershel Jick at Boston University, who co-authored the *JAMA* report, could "give us an important piece of the puzzle in the story of infection and heart disease."

Penninger and Kurt Bachmaier, a postdoc in his lab, had previously shown that injecting a fragment of the heart muscle protein myosin into mice causes severe inflammatory responses in the animals' hearts. To Penninger, this suggested that the immune system was mistaking the heart peptide for something foreign—perhaps a peptide in a microbe to which the mice had previously been exposed. To come up with a likely suspect, the team plugged the sequence for the offending peptide into sequence databases.

The researchers expected to find a related peptide in something like the Coxsackie B3 virus, long known for infecting heart muscle. But to their surprise, the sequence of the myosin fragment closely matched that of a peptide found in three strains of *C. trachomatis*, the culprit in sexually transmitted diseases. They also found similar, but not identical, peptides in *C. pneumoniae* and *C. psittaci*, known to cause respiratory infections.

The researchers soon confirmed that injecting the *Chlamydia* peptides, together with an immune booster called Freund's adjuvant or with the microbe's own DNA, into mice provokes heart inflammation, caused by T cells infiltrating the heart muscle. The vigilant immune cells, when activated in a mouse injected with the *Chlamydia* proteins and then transferred to another mouse, could also cause the same heart-destroying response in that animal.

But perhaps the most crucial evidence of all was the finding that live bacteria pumped by catheter and syringe into the noses or genital tracts of mice caused subsequent heart inflammation. The researchers also found that the mice made antibodies to the bacterial and heart peptides, and also to a third peptide from another heart protein—a telltale sign of an overzealous immune response, the researchers say. "We have proven that a bacterial infection in the genital tract or lungs can lead to cardiac inflammation," Penninger says. "Our paper takes this out of the realm of epidemiology and really says this is a causal link of how *Chlamydia* could work to cause heart disease." It also implies that preventive treatment with antibiotics might thwart some cases.

Other researchers are cautious, however. Cardiologist Brent Muhelstein at the University of Utah in Salt Lake City and others note that the inflammation resulting from the mimicry seems confined to the heart muscle itself rather than extending to the arteries, where it could trigger the plaques characteristic of atherosclerosis. In addition, *C. trachomatis*, which evokes the strongest response in Penninger's study, hasn't yet turned up in atherosclerotic plaques.

And even if *Chlamydia* infections are involved in human heart disease, researchers will want to know why many people escape the problem, even though *Chlamydia* infections are very common. One possibility, of course, is that such well-established risk factors as smoking and high blood cholesterol concentrations also influence how the body responds to *Chlamydia*.

CREDITS: (TOP RIGHT) LUIS M. DE LA MAZA, (ALL OTHERS) J. PENNINGER *ET AL.*