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

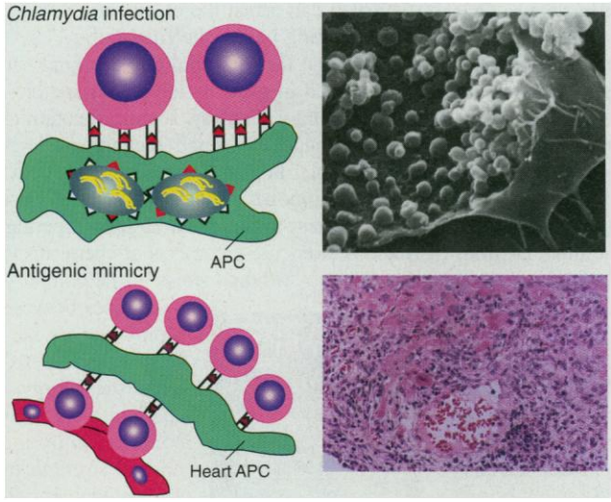
Alexander Hellemans is a writer in Naples, Italy.

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.*

The only way to tell if the microbe is triggering heart disease through molecular mimicry, Penninger says, is to do epidemiological studies to see if people who have antibodies against the bacterial peptide have a higher rate of the disease. Boston University's Jick agrees. "One of the obvious limitations is that, so far, the effect has been shown only to occur in mice," he says. **–TRISHA GURA**

Trisha Gura is a science writer in Cleveland, Ohio.

NEUROBIOLOGY

Fruit Fly Odor Receptors Found

Although researchers identified the receptors mammals use to detect odors almost a decade ago, they've been unable to sniff out those of any insect. Now, the impasse has been broken. Two teams, one led by John Carlson of Yale University and the other by Richard Axel of Columbia University, have independently discovered the first odor receptors in the fruit fly, *Drosophila melanogaster*.

The work, described in the February *Neuron* by Carlson and his colleagues and in the 5 March issue of *Cell* by Axel and his, has so far pulled out a total of 17 genes encoding *Drosophila* odor receptors. Given that these came out of the 15% of the *Drosophila* genome that has been sequenced, the insect may have 100 to 200 odor receptor genes in all.

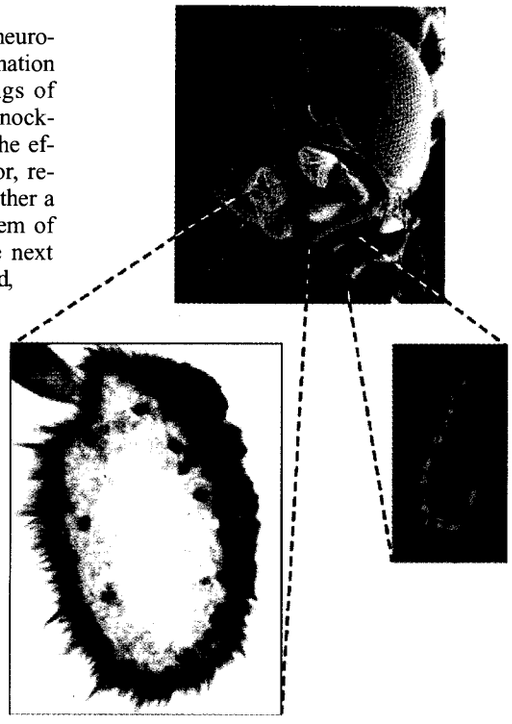
Their discovery will be a boon to neurobiologists, who hope to use the information to probe the more complex workings of mammal brains. By systematically knocking out the fly genes and observing the effects on odor sensitivity and behavior, researchers should be able to piece together a wiring diagram of the olfactory system of the fruit fly. "One can expect in the next few years that a lot will be discovered, providing important new insights into olfaction and probably into sensory coding," predicts Harvard University neurobiologist Catherine Dulac.

The first payoff, however, may be explaining how other insects behave. Already, researchers are using the sequences of the newfound *Drosophila* genes to track down odor receptors in insects that damage crops or transmit human diseases. Having these receptors in hand will make it much easier to find specific compounds that interfere with the insects' ability to detect odors. Because insects depend on smell to find mates and food, such substances could "really enhance our ability to control insect pests," notes Tim McClintock, a neurobiologist at the University of Kentucky College of Medicine, Lexington.

The key to success for both the Yale and Columbia groups was finding the first olfactory receptor gene. For years, others had tried to find these genes by looking for fruit fly genes whose sequences resemble those of known mammalian odor receptor genes. But those searches all came up empty. "These guys came up with a better way," says neurobiologist Dean Smith of the University of Texas Southwestern Medical Center, Dallas. They used a new method to search a growing fly DNA data set: the sequences accumulated by the Berkeley *Drosophila* Genome Project.

Aware that the odor receptor proteins would have to be embedded in the membranes of olfactory nerve endings, Yale's Peter Clyne, Junhyong Kim, and Coral Warr first looked for DNA sequences in the Berkeley data that might encode transmembrane domains, strings of hydrophobic amino acids that can tolerate insertion into fatty membranes. They then eliminated the nonsense DNA and the known genes. This got them down to 34 candidates. Two turned out to be elusive odor receptor genes, as evidenced by their location in the olfactory neurons.

At Columbia, Leslie Vosshall and her colleagues found their first *Drosophila* odor receptor gene by searching for genes that are active only in the fruit fly olfactory organs, the antennae, and a rod-shaped projection on the head called a maxillary palp. The researchers did this by comparing messenger RNAs (mRNAs), which indicate active genes, from the olfactory organs with mRNAs from the whole body and the head. Vosshall gradually homed in on a small set of genes, which she could begin sequencing and testing whether they are active only in the olfactory sensory nerve cells. She found one such gene, and like the Carlson team, used it to find related genes in the sequence database.



CREDIT: CARLSON ET AL.

Odor code. The dark staining *(lower left)* shows that an odor receptor gene is active in just a subset of *Drosophila* olfactory nerve cells, and the light blue staining *(lower right)* shows that the same is true for a gene regulating odor receptor expression.

Both groups now have clues about how the fruit fly brain perceives odors. They've shown that the genes are expressed differently in the various olfactory nerve cells. These data suggest that fruit flies, like vertebrates, may discriminate odors by decoding patterns of nerve activation that reflect the responses of many individual cells, each attuned to a single sensation.

Carlson's team also learned something from flies with a damaged sense of smell. In a separate study, they found a defective gene in the flies that codes for a protein that regulates gene expression. The defect appears to turn off certain receptor genes in some olfactory nerve cells. "The fact that some receptors are gone is pretty cool," Smith says, as it suggests this DNA regulatory protein helps set up the pattern of gene activity—and odor sensitivity—in the fly. In addition, as in vertebrates, Carlson notes, various fly odor receptor genes appear to be active at different times during development and may help organize the olfactory system.

He and his team plan to continue to look for more odor receptor genes and try to understand how these genes are regulated. "I feel like a kid in a candy store," Carlson says. "There's a million things we can now do."

–ELIZABETH PENNISI

ARCHAEOLOGY

Kennewick Man Gets His Day in the Lab

More than 30 months after a 9000-year-old skeleton was found on the banks of Washington's Columbia River, a government-appointed team of scientists has begun an examination to decide, once and for all, whether Kennewick Man qualifies as a Native American. Scientists are happy that the skeleton has made it into the lab, but they are worried that the government could put a crimp on the way the work is done and reported.

On 17 February, the Interior Department announced that five scientists have been appointed to help Frank McManamon, chief archaeologist of the National Park Service, perform a systematic analysis of Kennewick's 300-plus bone fragments. The work is being done at Seattle's Burke Museum,