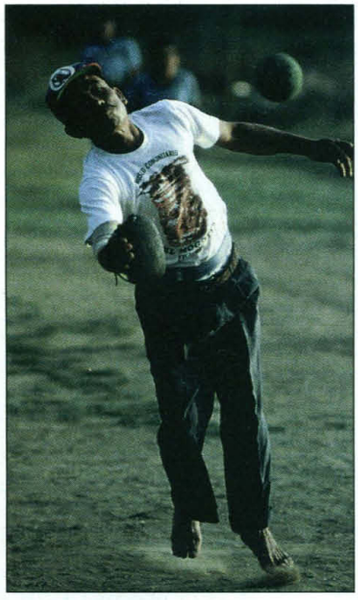
ingly magical material, Martyr wrote, by collecting sap from lowland trees and mixing in juice from a vine. Four centuries later, this crude recipe has finally given up some of its secrets. On page 1988, researchers describe how the Olmec, Maya, and other ancient Mexican and Central American cultures turned raw latex into rubber. This feat of chemistry, which converts the slippery polymers in raw latex to a resilient structure, was not duplicated until the mid-19th century. "It's a marvelous example of technology demonstrated at an incredibly early stage," says Frank Bates, a polymer chemist at the University of Minnesota, Minneapolis.



Lay techs. Re-creation of chaah, played with crude rubber ball.

The ball game, invented at least 3400 years ago, was an important ritual for many Mesoamerican societies. To the Maya, for instance, the game—called chaah—reenacted portions of their creation story. By the 5th century A.D., many towns had central stone courts, some of which could hold thousands of spectators. Leaders tested prophecies through tournaments, rival cities took out their aggressions on the court, and the rich placed huge wagers. According to a 16th century codex, the Aztec capital Tenochtitlan demanded 16,000 rubber balls each year as tribute from one province. The ballmakers "were the ancient equivalent of Rawlings," the sporting goods manufacturer, says Warren Hill, an archaeologist at the New World Archaeological Foundation of Brigham Young University in Provo, Utah. These societies also used rubber for a host of other products, including religious figurines, incense, and even lip balm.

Last summer, Massachusetts Institute of Technology (MIT) archaeologist Dorothy Hosler and undergrad Michael Tarkanian traveled to Chiapas, Mexico, to gather the raw materials for rubbermaking mentioned in ancient documents. To their surprise, they saw farmers collecting latex by slashing the bark of *Castilla elastica* trees, then mixing in juice from pulverized morning glory vines that wrap around the trees—just as the 400-year-old texts described. "It was amazing," recalls Tarkanian. "After about 10 minutes, a mass of rubber rose to the surface. We formed it into a ball that would easily bounce over your head."

The pair brought the ball, as well as raw latex and vine juice, back to their lab. A battery of tests showed that the homemade rubber was about twice as elastic as dried latex, which cracks when handled. With MIT materials scientist Sandra Burkett, the researchers probed the material with nuclear magnetic resonance spectroscopy, finding unidentified organic compounds in the latex that were absent from the rubber.

The team speculates that some of these mysterious compounds might be plasticizers, which would keep the latex runny by preventing its polymer molecules from linking to each other. (Modern rubber is made by cross-linking polymers.) If the vine juice dissolves the plasticizers, the researchers thought, polymer molecules would be more likely to entangle and form a rubbery mass. Although they failed to find direct evidence for cross-linking, they did discover vine juice components—traces of sulfonyl chlorides and sulfonic acids—that can react with polymers, stiffening segments and making them more likely to interact. The team says that only a few such entanglements would be enough to give the rubber its spring.

Understanding ancient rubbermaking "teaches us how conscious these people were of their environment and how they were able to manipulate it," Hosler says. She and her colleagues next plan to test rubber made with varying amounts of vine juice to see whether the Olmec, Maya, and Aztec could have engineered rubber with specific elasticities. No matter what they find, the Mesoamericans have earned the respect of modern chemists. "To discover [the process] and refine it to make those products is impressive," says Bates. "They probably had a pretty good R&D team."

–ERIK STOKSTAD

CREDITS: (LEFT) KENNETH GARRETT/*NATIONAL GEOGRAPHIC*; (RIGHT) ARCHIVE PHOTOS

NEUROBIOLOGY

Mutant Fruit Flies Respond to Lorenzo's Oil

The 1993 movie *Lorenzo's Oil* raised the profile of adrenoleukodystrophy (ALD), a fatal hereditary brain disease that strikes one in every 20,000 boys. The film told the true story of how one patient's parents set out to find a cure. Their brew of fatty acids, now known even among researchers as "Lorenzo's oil," didn't become the cure they had hoped for. But on page 1985, Kyung-Tai Min and Seymour Benzer of the California Institute of Technology in Pasadena report that a component of the oil prevents neural decay in fruit flies with a similar condition. The finding might spark new research on the oil, and it already has researchers enthused about the potential of the mutant flies for studying what causes ALD and how it might be treated.

Concerned parents. In the 1993 film *Lorenzo's Oil*, an afflicted boy's parents, portrayed by Susan Sarandon and Nick Nolte, looked for an ALD treatment.

"Using the fruit fly as a model … is ex-

tremely exciting," says neurologist Hugo Moser, who studies ALD at the Kennedy Krieger Institute at Johns Hopkins University School of Medicine in Baltimore. Neurologist Dennis Choi, of Washington University School of Medicine in St. Louis, agrees. Benzer and Min "didn't just simply create the model"; they also showed that it could identify "something that is of interest for human treatment," he says. "That is a validation."

Benzer and postdoc Min made their discovery as part of a project to find fruit fly mutations that mimic those that cause human neurodegenerative conditions. First they mutated flies with P elements, little bits of DNA that jump around the fly's genome, inactivating genes. Min then looked for mutants with shortened lifespans and examined them for signs of dying neurons. In one such mutant, one of the neural layers in the flies' eyes had an abnormal bubbly appearance, prompting the researchers—who had already named other neurodegenerative mutants after foods—to call it *bubblegum*.

When Min cloned the mutated gene, it turned out to encode a protein whose sequence suggests it is an acyl coenzyme A (CoA) synthetase, a type of enzyme that helps break down fatty acids. The involvement of that enzyme brought ALD to mind, Min recalls, because a similar enzyme is impaired in ALD. In the human disease, which is passed from mother to sons on the X chromosome, the impairment is indirect, because the primary mutation is in a gene encoding one of a class of proteins that transport substances across membranes. Researchers do not know how the transporter affects the synthetase, but the synthetase's decreased activity results in high blood levels of very long chain fatty acids (VLCFAs) in ALD patients and—also for poorly understood reasons—progressive degeneration of brain neurons. The ALD link prompted Min to see whether VLCFA levels are also high in the mutant flies, and sure enough, they were.

> **"Using the fruit fly as a model ... is extremely exciting."**
>
> —Hugo Moser

The researchers then tested whether the flies respond to Lorenzo's oil, which is a mixture of two fatty acids. In humans, the oil lowers VLCFA levels by slowing their synthesis, although so far in clinical trials it has shown little or no effect on disease progression. Benzer and Min saw something similar when they treated adult mutant flies with glyceryl trioleate oil, one of the components of Lorenzo's oil. The flies' VLCFA levels dropped, but their brains still had dying neurons. Min tried another approach, beginning treatment when the flies were larvae. When those flies grew to adulthood, says Min, "the pathology was not there."

On the face of it, that finding suggests that starting oil treatment early might ward off the neuron death. But giving the oil to humans before symptoms begin doesn't seem to have increased its effectiveness, says Moser. One reason may be that humans have a barrier that prevents easy transfer of many substances—including Lorenzo's oil—from the blood to the brain. Fruit flies lack such a barrier, and Moser says the fly results may rekindle efforts to alter Lorenzo's oil to enable it to enter the brain.

ALD and the fly condition differ in other ways as well. Not only are the mutations in different genes, but flies lack an inflammatory response, which may contribute to the human disease. And the sick fly neurons look different from the dying neurons in ALD. Although flies may be an imperfect model, there are enough similarities to expect that "we can learn a lot" from them, says biochemist Paul Watkins, who studies ALD at the Kennedy Krieger Institute.

The flies should allow researchers to quickly screen potential ALD treatments for their effects on diseased brain cells, says Choi. Compounds identified this way would not be a sure bet against the human disease, he cautions, but they "would be good starting points" for further study. Watkins adds that the mutant flies may provide insights into the molecular basis for ALD. His lab is searching for the acyl CoA synthetase that is key in ALD, and although they have found six human versions of the enzyme so far, none is abundant enough in the brain to fit the bill. The defective fly enzyme belongs to a different subfamily than the known human enzymes, and because its mutation causes neuropathology, he says, it "could be the one we're missing."

If any of these applications proves fruitful, Benzer will no doubt be pleased. He says his goal was to establish flies as a disease model that, although not perfect, would provide "hints on physiology which may or may not extrapolate to humans." And he has his sights set beyond ALD; *bubblegum*, he says, is just one of a "whole zoo" of mutant fly strains developed by his lab that suffer neural degeneration and might turn out to be useful models of other human diseases.

–MARCIA BARINAGA

ScienceScope

Down to the Summit Marine scientists are cruising toward their goal of building an observatory at the tip of a massive underwater volcano. More than two dozen researchers this week boarded a University of Washington research vessel bound for the Axial volcano, 400 kilometers off the Oregon coast. Once there, they hope to complete the second phase of the year-old U.S.–Canadian observatory project, which plans to assemble a suite of instruments focused on the geology and biology of the Juan de Fuca Ridge, where microbes and other sea life thrive around sea-floor vents of superheated water.

Last year, researchers installed the first instruments—including water chemistry and earthquake monitors—at the New Millennium Observatory (NeMO), which sits more than 1000 meters above the sea floor and 1400 meters under the surface. On this year's cruise they plan to drill rock cores and deploy new equipment at the observatory, which has no firm completion date.

Hopes for neat science are high, as NeMO rests on "the most volcanically active site" in the region, says oceanographer Stephen Hammond of the Marine Environmental Laboratory in Newport, Oregon. To follow the action, check out newport.pmel.noaa.gov/nemo.

Gene Bonanza? Germany's biggest basic research funder says the country's genome studies are lagging behind and need a big cash infusion to catch up to the United States, France, and the U.K. In a report last week, the DFG argued that the government should spend an additional $570 million over the next 5 years to put German genome research on the world map.

The report suggests that about 40% of the new funds go to studying genes identified in the human genome project, the genomes of model organisms such as the fruit fly, and ethical and legal issues. Plant and microbe studies would split another 40%, with the rest to be spent on bioinformatics, overhead, and other categories. The DFG also made a pitch for a new national committee to coordinate Germany's genome efforts.

Such ideas are likely to get a serious hearing from research minister Edelgard Bulmahn, who wants more genome research. Last month, she said her ministry will ask Parliament to double funding for Germany's part of the Human Genome Project, to about $45 million a year by 2002.