CHEMISTRY

New Polymer May Rev Up the Output Of Fuel Cells Used to Power Cars

ATLANTA, GEORGIA—Most technologies must keep constantly improving to stay on top. But in the world of low-temperature fuel cells—the sort used to power cars—a polymer membrane made by DuPont, called Nafion, has been the gold standard for decades. Last week, however, at a meeting here of the American Chemical Society, researchers from North Carolina unveiled an upstart that might finally dethrone Nafion and markedly improve the performance of automotive fuel cells.

Fuel cells work by converting chemical fuel directly into electricity without burning it. The standard approach requires reacting hydrogen and oxygen at two different electrodes separated by a thin plastic membrane. At one electrode, hydrogen molecules are stripped of their electrons, which are then sent through an external circuit to do work. The leftover protons are channeled through the polymer membrane to another electrode, where they meet up with oxygen and the circulating electrons to produce water.

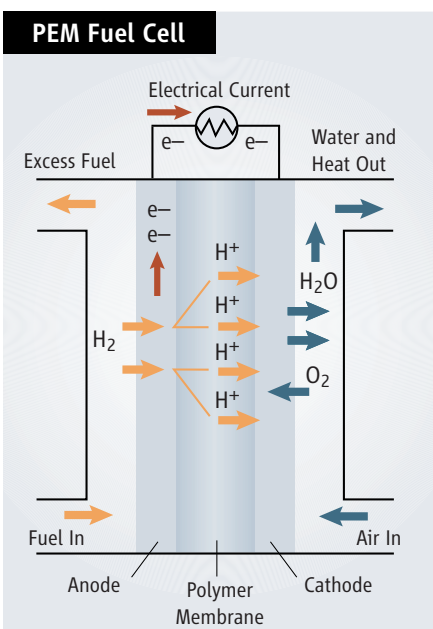
But making good proton conductors from polymers isn't easy. Nafion's strategy is to link acid groups to the end of fluoropolymer chains. Because acids hold on to their protons only loosely, they are good proton conductors. But acids also readily dissolve in water, which is needed for standard proton exchange membrane (PEM) fuel cells to operate. So if you put too many acids on your polymer, it falls apart when you run your fuel cell.

In hopes of boosting the acid content of their polymers, Joseph DeSimone, a chemist at the University of North Carolina, Chapel Hill, and his graduate student Zhilian Zhou decided to create a polymer with extra links between the chains so that it wouldn't dissolve in water. They started with a heavily fluorinated polymer called perfluoropolyether, which they copolymerized with a derivative of an acid-rich compound called styrene sulfonic acid. The researchers mixed the compounds as liquids and then polymerized them using ultraviolet (UV) light. The UV light knitted the two compounds together into chains and forged links between the chains, creating an extended network of polymers that doesn't dissolve when the water content climbs.

Because the polymer contained more acid groups, it conducted protons nearly three times as well as Nafion. "This sounds very nice and could set a new gold standard," says Robert Hockaday, a fuel cell expert who runs Energy Related Devices Inc., a fuel cell company in Los Alamos, New Mexico.

Unlike Nafion, which comes only in thin sheets, the new polymer also can be cured from its liquid precursors in essentially any shape. Zhou and DeSimone, for example, patterned their polymer using a standard stamping technique into a form with a much higher surface area than a flat film. That surface area is key to fuel cells because engineers pattern the two sides of their films with the metals, such as platinum, that make up the electrodes that carry out the needed chemical reactions. An increased surface area allows for a more widespread platinum coating, leading to increased power. In this case, the patterning doubled the power output of their fuel cells.

The new polymers could bring another advantage as well. Because the crosslinked polymers are likely to be far more robust than Nafion, they should withstand higher operating temperatures. That's key, because raising a fuel cell's operating temperature from the standard 80°C to about 120°C ought to prevent contaminants such as carbon monoxide from glomming onto the platinum catalyst and sapping the cell's performance. DeSimone says he expects that their new crosslinked polymer membrane will hold up far better than Nafion at higher operating temperatures, but he and Zhou haven't had a chance to run the experiment. Their continued success could herald a new king of the hill among polymer fuel cells. **–ROBERT F. SERVICE**



CREDIT: ADAPTED FROM U.S. DEPARTMENT OF ENERGY

Power plastic. A better proton (H^+) conductor bids to unseat the longtime champion at the heart of large fuel cells.

Maryland Goes for Stem Cells

Maryland is about to become the fourth state—after New Jersey, California, and Connecticut—to create its own human embryonic stem cell research program after Republican Governor Robert Ehrlich pledged to sign a bill passed last week by state lawmakers.

The bill, a 5-year authorization that sets up a commission to oversee the work, is the result of some fancy footwork by the Democrat-controlled legislature that avoids any mention of "human embryos" and substitutes the term "material." But it defines stem cells as cells that "divide indefinitely" and give rise to "many" cell types, thus excluding most adult stem cells. The bill also outlaws reproductive cloning. Although it doesn't forbid research cloning, it authorizes funds only for research on embryos that would otherwise be discarded by fertility clinics. A separate spending bill included $15 million for the work next year.

Curt Civin, a stem cell researcher at Johns Hopkins University in Baltimore, welcomes the law, saying Maryland's investment might eventually be "comparable" to the $300-million-a-year California program on a per capita basis. Supporters say they expect additional state funding if Ehrlich loses his reelection bid in November, as Republican lawmakers whittled down an initial plan to spend $25 million. **–CONSTANCE HOLDEN**

Collateral Damage

TOKYO—A genetics center is the latest victim of a scandal roiling the Japanese scientific community. Last week, the National Institute of Advanced Industrial Science and Technology in Tsukuba announced it will close a 3-year-old gene function research center led by Kazunari Taira, a University of Tokyo chemist under fire over his failure to substantiate findings in a series of papers published in prominent journals (*Science*, 3 February, p. 595).

In an action that an institute official says is not a disciplinary measure, the institute has opted not to extend the contracts of Taira and a key associate, transferring the 51 other center staff members to other labs. "The misconduct problem has made it very difficult to administer the research center," says institute director Masanori Yoshikai. Last week, a University of Tokyo investigative committee said it found no solid evidence of deliberate fraud in the original work but that Hiroaki Kawasaki, a research associate in Taira's lab, fabricated data during attempts to reproduce the experiments. Taira intends to retract four papers; a disciplinary panel will weigh in next week.

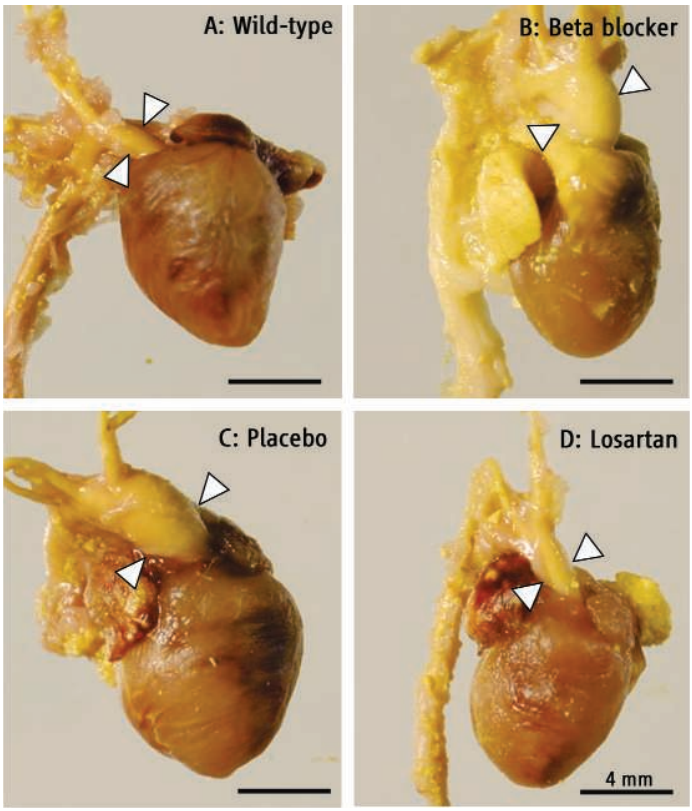
–HIROMI YOKOYAMA

Published by AAAS

such a defect was to restore the missing fibrillin-1, yet gene therapy and other potential replacement solutions seemed like distant prospects.

Almost from the outset, Dietz had doubts about this explanation for Marfan syndrome. "We began to recognize that selective features couldn't be explained by disease models that relied on weakness of tissues," the Howard Hughes Medical Institute investigator says—bone overgrowth, for example, was difficult to reconcile.

In 2001, Dietz's team reported creating mice that make low amounts of fibrillin-1 and develop emphysemalike lung problems similar to those seen in about 10% of Marfan patients. But a close look at the mouse lungs showed that the defect began early in development, challenging the notion that the emphysema was due to the deterioration of lung tissue over time. The scientists instead fingered overactivity of a protein called transforming growth factor–β (TGF-β); they also found that administering antibodies to it prevented the lung damage in the mice.



Heart of the matter. The aorta (arrows) of a normal mouse (A) and a losartan-treated mouse with a fibrillin-1 mutation (D) are indistinguishable, but those of mutant mice treated with a beta blocker (B) or placebo (C) have aneurysms.

Dietz now believes that fibrillin-1 binds TGF-β in the extracellular matrix, keeping it inactive. In people with Marfan, the growth factor is unleashed, he hypothesizes. Indeed, Dietz and his group have also shown that antibodies to TGF-β can prevent heart valve problems and aortic aneurysms in their Marfan-like mice.

Seeking a more practical way than antibodies to block TGF-β signaling, the researchers hit upon losartan, which was known to somehow thwart the growth factor. It seemed a natural drug to explore because many physicians treating Marfan syndrome already prescribe blood pressure–lowering drugs called beta blockers to ease stress on the aorta and slow its growth.

For their losartan study, Dietz's group worked with mice engineered to have a fibrillin-1 mutation analogous to those seen in Marfan syndrome. They began administering the drug to 2-month-old mice, which already have obvious aortic changes. After 6 months, the aortic aneurysms had worsened in mice given either a placebo or a beta blocker, but normal mice and losartan-treated Marfan ones were indistinguishable, indicating that the drug had reversed the early aorta damage. "It was truly a jaw-dropping moment. It was beyond anything I could have anticipated or hoped," says Dietz.

Because the drug has a good safety record in people, Dietz and his colleagues are already giving losartan to a few children. But he cautions that there could be unanticipated side effects in those with Marfan syndrome. These initial tests on the children with rapidly progressing aneurysms are ethically defensible, says medical geneticist Peter Byers of the University in Washington, Seattle, because the children will otherwise need multiple, risky surgeries as they outgrow initial aortic replacements.

Dietz says NIH is also finalizing plans to enroll 700 to 1000 people with Marfan syndrome, ranging from 6 months to 25 years old, in a trial coordinated by a multicenter group called the Pediatric Heart Network. Patients will receive either losartan or a beta blocker. Byers notes that the "striking" mouse results could make it difficult to recruit patients into a trial in which they may not receive losartan.

Meanwhile, Dietz and his colleagues are looking into whether losartan can treat additional conditions associated with aortic aneurysms. "I think you are going to see this [drug] extended across diseases that are TGF-β–related," predicts Chien. **–JOHN TRAVIS**

CREDITS (TOP TO BOTTOM): J. HABASHI *ET AL.*, *SCIENCE*; U.S. DOE

Italian Voters Ponder Science

A hot topic in Italy's election on 9 and 10 April is whether research has thrived under Silvio Berlusconi's conservative government. His research minister, Letizia Moratti, has installed a commission for evaluating research, centralized academic recruitment, and secured more private funding for national institutes, aligning them with national goals (*Science*, 1 April 2005, p. 35). Berlusconi insists the moves will raise competitiveness, but the left-wing opposition and many in the research community say the policies have weakened Italian science.

At a rally in Rome last week, the left-wing coalition led by Romano Prodi announced that, if elected, it would more than double Italy's rate of research spending from the current 1.1% to 3% of gross domestic product by 2010.

–SUSAN BIGGIN

Updates

■ After a slow start, a U.S. advisory panel set up to prevent the results of federally funded biology research from being used by terrorists has put out two draft proposals. The National Science Advisory Board for Biosecurity has aired a report on so-called dual-use experiments and drafted guidelines to help journals screen papers.

■ India has joined the government steering committee of a U.S. project to build a $1 billion advanced coal plant that sequesters carbon dioxide and produces hydrogen (design below). Meanwhile, 22 sites in nine states have said they plan to compete for the plant, called FutureGen. Proposals are due 4 May.

■ Last week, the U.S. Supreme Court heard the second set of arguments in 2 weeks on patents. The cases involve patenting a scientific concept and the power of a patent to halt a competitor (*Science*, 17 February, p. 946). The biotech community is closely awaiting the decisions, expected by June.

■ Ending a 2-year battle, France's National Assembly this week gave final passage to a research reform bill. Researchers are disappointed that the bill offers no guarantees that science budgets will be indexed for inflation until 2010 (*Science*, 24 March, p. 1693).