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Cover A giant, Jupiterlike planet closely orbits its fiery star in this illustration. When such planets pass in front of their stars, astronomers can not only detect the planet but also measure its size and composition. The method is revolutionizing exoplanet astronomy. (T. Pyle, JPL-Caltech/NASA) Page 24

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SCIENCE NEWS This Week

Tumor Suicide Gene therapy

Gene therapy makes cancer cells self-destruct

More than 37,000 people in the United States will be diagnosed with pancreatic cancer this year, and nearly all of those cases will be untreatable. Now, scientists have developed a gene therapy that kills pancreatic tumors in mice by causing the tumor cells to commit suicide.

Gene therapies often use crippled viruses to deliver therapeutic genes into a patient's cells, but injecting these modified viruses into people can be risky. For example, a patient in a 1999 gene therapy trial died from a severe immune reaction that scientists suspect was caused by the delivery virus.

To avoid such problems, Mien-Chie Hung and his colleagues at the M.D. Anderson Cancer Center in Houston packaged a self-destruct gene inside microscopic bubbles called liposomes. These bubbles, measuring 100 to 200 nanometers across, are roughly the size of viruses and have surfaces made of fat molecules similar to those in cell membranes. When liposomes touch cells in a patient's body, they can easily fuse with a cell's membranes and dump their genetic cargo inside. There, the gene triggers the cells' natural self-destruct mechanisms, which normally swing into action to remove severely damaged cells.

To avoid adverse side effects, the scientists had to ensure that the gene would exert its deadly influence only in cancerous cells. The team incorporated the self-destruct gene into a ring-shaped DNA molecule in such a way that the gene would become active only in the presence of a certain protein found exclusively in pancreatic-tumor cells. Although the liposome delivered this ring of DNA to cells throughout the animals' bodies, the killer gene affected only tumor cells.

"In terms of gene therapies for pancreatic cancer, this is by far the most impressive data because we designed the therapeutic gene to be active only in the tumor cells," Hung says. In experiments on human cells in culture, the liposomes killed pancreatic cancer cells but spared healthy cells. The scientists then implanted human-pancreatic cancer cells into mice and allowed tumors to develop. Treatment with the gene therapy shrank the tumors, and 50 percent of the treated mice survived at least 120 days with no detectable cancer. All the untreated mice died in less than 45 days. The researchers found no evidence of toxic side effects from the treatment, they report in the July *Cancer Cell*.

"I think this [study's] approach is unique in achieving both a strong effect and good specificity" for pancreatic cancer cells, comments Scott E. Kern, a pancreatic oncologist at Johns Hopkins University School of Medicine in Baltimore. However, Kern notes that only a small fraction of liposomes injected into the bloodstream will randomly find their way to the tumor cells, as occurred in the Hung team's mouse experiments.

Kern suggests that scaling up the therapy for people might require an additional way to target the liposomes to enter only tumor cells. Hung, however, believes that the current delivery system will work in people. —P. BARRY

Sea Change People have affected what penguins eat

The eating habits of Adélie penguins in Antarctica changed significantly about 200 years ago, according to chemical analyses of the birds' eggshells. Scientists attribute the shift in diet to whaling and other hunting in the region.

The ratios of carbon and nitrogen isotopes in an animal's tissues—including bones and eggshells—can provide a wealth of information about its eating habits, says Steven D. Emslie, a paleontologist at the University of North Carolina in Wilmington. Recently, he and William P. Patterson, a geochemist at the University of Saskatchewan in Saskatoon, looked at the chemical composition of Adélie penguin eggshells laid during the past 38,000 years to see whether the birds' dietary habits had changed.

Surprisingly, says Emslie, climate change 10,000 years ago, at the end of the latest ice age, didn't significantly affect the birds' diet. In the past 200 years, however, the chemical composition of the penguins' eggshells made a dramatic shift to lighter isotopes of carbon and nitrogen. Because animals higher in a food chain hold greater concentrations of heavy isotopes, the change is clear evidence that the penguins' diet shifted from primarily fish to prey such as krill.

The dietary change boils down to the availability of prey, Emslie and Patterson speculate in the July 10 *Proceedings of the National Academy of Sciences*. During the 19th century, the population of krill in southern seas exploded after Antarctic fur seals, prodigious consumers of krill, were hunted nearly to extinction. That slaughter, followed by widespread killing of krill-eating whales during the 20th century, enabled the tiny crustaceans to proliferate nearly unchecked, says Emslie.

"It's rare to see such catastrophic changes [in diet] in such a short period," says Keith A. Hobson, an ecologist at Environment Canada in Saskatoon. The changes "point to a large shift in the ecosystem," he notes. Even so, Hobson adds, it's not clear why abundant krill would cause penguins to suddenly shift from fish to what had previously been a secondary food source.

The team's results are "very compelling evidence of a terrific change" in penguin



DIET CHRONICLE Shifts in the chemical composition of Adélie penguins' eggshell fragments suggest that the birds' diet has changed significantly in the past 200 years.

SCIENCE NEWS This Week

diet, says Charles H. Peterson, a marine ecologist at the University of North Carolina's Institute of Marine Sciences at Morehead City. "It's a cosmic irony of foodweb ecology that a rare species is only rare because it's kept in check by predators," he adds. "Maybe krill was one of [the penguins'] favorite foods all along."

Modern-day fishing around Antarctica has depleted fish stocks there. Meanwhile, krill populations have declined as much as 80 percent in the past 2 decades. Understanding why penguin diets changed 2 centuries ago may be vital for their future survival, says Emslie. —S. PERKINS

E-Waste Hazards

Chinese gear recyclers absorb toxic chemicals

Residents of a Chinese region where 80 percent of families include workers who dismantle and recycle electronic devices have high concentrations of flame-retardant chemicals in their blood, researchers report. Inhabitants of a fishing village not far away also carried elevated amounts of the chemicals, called polybrominated diphenyl ethers (PBDEs).

Much of the world's electronic waste ends up in China, where most handlers of the materials work without protective gear. They smash the components and strip out metals, releasing dust laden with deca-BDE, a flame retardant commonly added to plastic components.

In this first study of PBDE occupational exposure in China, researchers at the Chinese Academy of Sciences in Guangzhou and Lancaster University in England analyzed blood samples from individuals at two sites in southern China. One group of people lived in Guiyu, an electronic-waste-dismantling area in southern China. People in a comparison group lived in Haojiang, a fishing village 50 kilometers away.

PBDEs come in 209 forms that include different arrangements of up to 10 bromine atoms. Studies in mice and rats have shown that PBDEs with 5 or 8 bromine atoms harm brain development (*SN: 10/13/01, p. 238; 10/25/03, p. 266*). Growing evidence suggests that deca-BDE, which contains 10 bromine atoms, can cause the same developmental problems either on its own or



STRIKINGLY UNPROTECTED Electronics recyclers in Guiyu, China, often work outside and without safety equipment, exposing themselves and the environment to contaminated dust.

when it breaks down into PBDEs with fewer bromines, says Linda Birnbaum, director of the Environmental Protection Agency's experimental toxicology division.

Deca-BDE is widely used in electronics and upholstery. The Guiyu residents had a median concentration of deca-BDE up to 200 times as high as were typically seen in two Swedish studies of industrial workers.

Total PBDE concentrations among individuals in Guiyu had a median value three times as high as did the individuals in Haojiang, the researchers report in an upcoming *Environmental Science & Technology*. The elevated concentrations of PBDEs in villagers in Haojiang indicate that airborne dust particles might have carried the chemicals to the village, says Gareth Thomas of Lancaster University, a coauthor of the study. The highest deca-BDE contamination ever reported was recorded in a 32-year-old Guiyu man whose blood contained 3,100 parts per billion (ppb) lipid. Lipid molecules, or fat, accumulate these chemicals.

The astronomical concentrations of deca-BDE, a median of 310 ppb lipid in Guiyu, indicate regular, heavy exposure to the chemical, comments Åke Bergman of Stockholm University. That's because deca-BDE has a half-life in the body of just 15 days. "In order to keep up these very high concentrations, the people need to be continuously exposed," he says.

The overall PBDE concentrations seen in the Guiyu residents are in "a risk region" for exposing a woman's fetus to amounts of the compounds that could damage a developing brain, Bergman adds. He notes that electronic-waste recycling is done in other countries by workers who may be no better protected than the Guiyu workers are. "We may have a few more areas in the world where we have [elevated] exposure to humans and also to the environment," he says. —S. WEBB

Smoke This

Parkinson's is rarer among tobacco users

Call it a flimsy silver lining to a noxious blue cloud: Long-term smokers have half the risk of Parkinson's disease that nonsmokers do, according to a new report.

In 12,000 people studied, those who smoked the most—the equivalent of at least a pack a day for 60 years—had the lowest risk. And after smokers stubbed out their last butts, the protective effect faded.

Cigarette, cigar, and pipe smoking appear to offer similar anti-Parkinson's benefits, according to the report in the July *Archives* of *Neurology*.

Author Beate Ritz of the University of California, Los Angeles characterizes the amount of Parkinson's protection provided by smoking as moderate. "Never-smokers have about a twofold higher risk of Parkinson's disease than ever-smokers," she says.

However, because Parkinson's disease is fairly rare—only about 60,000 new cases are diagnosed each year in the United States—and because smoking causes cancer and heart disease, "nobody would ever recommend smoking in order to prevent Parkinson's," Ritz emphasizes.

Ritz and her colleagues compiled data from 11 epidemiological studies dating back to 1960. The studies included about 3,000 Parkinson's patients and 9,000 healthy people. Ritz' team recorded the smoking history and Parkinson's status of each subject.

For 2 decades, researchers have speculated that tobacco prevents Parkinson's. The new report, though, "is extremely convincing," says Maryka Quik of the Parkinson's Institute in Sunnyvale, Calif.

The protective effect was seen in every age group except those diagnosed at age 75 or later. Ritz speculates that the difficulty of diagnosing Parkinson's in older patients may explain that result.

As for how smoking may prevent the disease, "nicotine is the likely suspect," says study coauthor Harvey Checkoway of the University of Washington in Seattle.

Robert L. Copeland Jr. of the Howard University College of Medicine in Washington, D.C., agrees. He points to studies in his lab and elsewhere showing that nicotine protects neurons that generate dopamine, a key signaling molecule in the brain.

Parkinson's symptoms appear after patients lose 70 to 80 percent of their dopamine-making neurons. Several small studies have tested nicotine patches and gum for symptom relief in people who already have the disease. But by then, it may be too late for nicotine to do much good, says Quik. Such trial results have been equivocal.

Ritz offers other possible explanations. Some 4,000 chemicals pollute tobacco smoke, and any of those alone or in combination with nicotine may contribute to the anti-Parkinson's effect, she says.

Some aspects of the data lead Ritz to speculate that the explanation isn't chemical at all. Instead, there may be some fundamental difference in susceptibility to nicotine addiction between people who develop Parkinson's and those who don't. "It could be a difference in the dopamine system in brain," Ritz says. Dopamine is important both in Parkinson's disease and in addiction. —B. VASTAG

Shattering Find?

Comet fragments show surprising uniformity

When comets pass close to the sun, solar radiation can bake and chemically alter their outer layers. Yet new observations of fragments of a comet that broke apart almost in front of astronomers' eyes suggest that its interior was remarkably similar to its exterior.

For comets, breaking up isn't hard to do. Relics of the solar system's formation, these fragile amalgams of ice, rock, and dust can burst into fragments when the sun's heat vaporizes some of their icy material.

By observing such fragments, astronomers can compare material disgorged from a comet's core with the presumably sun-altered material at its surface. Theory suggests that these changes ought to be significant, especially for comets that frequently pass near the sun. That's why scientists were surprised to find that two recently separated chunks have highly similar compositions.



COMET CHIP Fragment B of comet 73P/Schwassmann-Wachmann 3, observed last spring with the Hubble Space Telescope. The fragment is casting off boulder-size chunks.

If that observation is representative of comets in general, it suggests that at least some of these frozen bodies may preserve much more of their primordial composition than astronomers have generally believed, says Neil Dello Russo of the Johns Hopkins University's Applied Physics Laboratory in Laurel, Md. He and his colleagues describe their study of two large fragments of a comet called 73P/Schwassmann-Wachmann 3 (SW3) in the July 12 *Nature*.

The comet, which orbits the sun every 5.34 years, split into at least five chunks in 1995. In June 2006, it passed within 11.7 million kilometers of Earth, just as it further disintegrated into 68 identifiable pieces (*SN*: 5/6/06, p. 277).

The two largest chunks, dubbed B and C, are each several hundred meters in diameter. Russo are his colleagues took spectra of these fragments with NASA's Infrared Telescope Facility and the Keck II telescope, both on Hawaii's Mauna Kea. The measurements, the most accurate ever obtained for a disintegrating comet, reveal that B and C have virtually identical relative abundances of many simple compounds, including water, the hydroxyl molecule, and carbon dioxide.

"We were really lucky" that the comet came close enough for astronomers to make observations soon after a breakup, says Russo. Because such compositional information hasn't been obtained for other fragmenting comets, it's difficult to determine how broadly the findings apply, he cautions.

Previous measurements of other fragmenting comets had hinted at a uniform composition, says comet scientist Michael F. A'Hearn of the University of Maryland at College Park. Researchers are trying to determine whether the diversity of dust particles observed after NASA's Deep Impact spacecraft blasted a hole in Comet Tempel-1 is the result of recent exposure to the sun or represent primordial variations in the comet's composition (*SN: 9/10/05, p. 168*).

The bottom line, says A'Hearn, is that while the new findings on SW3 "may [indeed] be suggesting that comets might be homogeneous ... I'm not yet ready to tilt in favor of that hypothesis." —R. COWEN

Forget About It How the brain suppresses unwanted memories

Not only can people intentionally forget disturbing memories, but they do so thanks to a pair of previously unreported neural processes, a new study finds.

Researchers have long argued about the existence of memory suppression and especially the ability to wipe out mental traces of traumatic events. A team led by psychologist Brendan E. Depue of the University of Colorado in Boulder has now found that a one-two neural punch fosters volunteers' ability to forget upsetting scenes.

In early stages of intentional forgetting, part of the brain's prefrontal cortex dampens activity in neural areas involved in visual and other sensory aspects of memory, Depue and his colleagues report in the July 13 *Science*. As the process of forgetting continues, a different prefrontal area quells the activity of structures implicated in conscious recall of information and emotions such as fear.

"The brain acquires multiple [ways] to reduce the likelihood of retrieving unwanted memories," Depue says.

His team obtained functional magnetic resonance images of brain activity in 16 women during a forgetting experiment. Volunteers first learned to associate each of 40 strangers' faces with a disturbing scene, such as a car crash. While viewing the same faces in a dozen ensuing trials, participants were instructed either to think of the previously associated images or to keep those images from entering their consciousnesses.

The researchers then tested the women's

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ability to describe images that they had linked to each face. Those who had tried to remember the linked scenes recalled an average of 71 percent of the images, as opposed to 53 percent among volunteers who had tried to forget linked scenes.

Brain images indicated that blood flow, a marker of neural activity, increased in a specific prefrontal area early during volunteers' attempts at forgetting. At the same time, blood flow declined in the visual cortex and the thalamus, areas that handle sensory components of memory. The prefrontal cortex regulates these and other structures through networks of anatomical connections.

A different neural pattern eventually appeared among memory suppressors. Pronounced activity in another prefrontal area accompanied sparse blood flow in the hippocampus and amygdala, areas responsible for consciously recalled memories and fear conditioning, respectively.

The new results elaborate on a 2004 study, directed by psychologist Michael C. Anderson of the University of Oregon in Eugene, that implicated prefrontal areas in memory suppression. Anderson investigates intentional forgetting of disturbing words (*SN: 3/17/01, p. 164*).

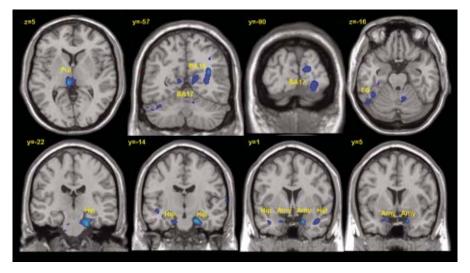
Depue's work shows for the first time that a neural emotion regulator, the amygdala, contributes to suppressing upsetting memories and that two neural mechanisms promote this process, Anderson says.

He regards memory suppression as a voluntary form of the defense mechanism known as repression. "These findings run counter to the general idea that traumatic memories are difficult, if not impossible, to suppress," Anderson remarks.

Other new evidence indicates that the prefrontal region activated early in the attempt to forget upsetting scenes also participates in suppressing memory for information irrelevant to a current task, adds neuroscientist Anthony D. Wagner of Stanford University. His team's findings appear in the July *Nature Neuroscience.* —B. BOWER

Pulling Strings Stretching proteins can reveal how they fold

Proteins, long strings of amino acids, spontaneously fold into intricate shapes that enable them to perform a cell's dazzling



SLIPPED MY MIND Colored areas of brain scans show visual regions deactivated in early stages of memory suppression (top row), and emotion and memory structures disengaged later during intentional forgetting (bottom row).

variety of functions. To better understand the forces that determine these shapes, scientists have developed a technique for stretching a protein to follow in reverse the path it took when folding.

"The basic idea is to pull the molecule at both ends to stretch it and see what happens," says Ching-Hwa Kiang, a biological physicist at Rice University in Houston.

When a cell builds a protein, it links amino acids that pivot around each other and interlock. These movements are dictated by electrostatic forces between the amino acids and by their tendency to hide their water-repelling sides while leaving their water-loving sides exposed.

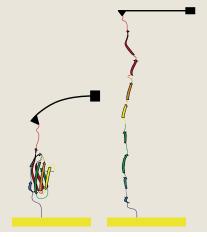
A fully folded protein is in a state of minimum energy because force must be applied to pull it apart. Kiang and her Rice collaborators devised a technique to measure that force. They placed water droplets containing proteins on a movable surface below a microcantilever akin to a tiny diving board. The researchers fished for proteins by varying the distance between the surface and the cantilever. When the cantilever snagged one end of a protein, the scientists could pull back the surface, slowly unfolding the protein.

The bending of the cantilever indicated the force required to stretch the protein. The researchers tested their technique on a synthetic version of the muscle protein titin, consisting of a chain of eight identical amino acid strings. As the researchers stretched the protein, the strings unfolded one after the other, generating the same sequence of force measurements each time. The team reports its findings in an upcoming issue of *Physical Review Letters*.

Unfolding a protein requires energy to overcome friction between molecules in addition to the energy needed to counter molecular forces. To tease apart these effects, the team used a mathematical technique invented in 1997 by Christopher Jarzynski, now at the University of Maryland at College Park. That analysis took into account the reductions in measured force due to random molecular jiggling that sometimes kicked the protein into an unfolded state.

The researchers plan to apply their technique to other proteins. They also hope to measure the energy required to unzip the double helix of DNA. Kiang says that researchers could also use the technique to test whether environmental conditions such as acidity or temperature affect folding. Scientists believe that misfolded proteins may cause certain diseases, including Alzheimer's.

Kevin Plaxco of the University of California, Santa Barbara says that scientists are eager to find methods for mapping the energy of proteins. While the new technique traces only one possible way that a protein unfolds, as opposed to the full range of a protein's possible states, "it's the most concrete example I've seen," of such a measurement, he says. —D. CASTELVECCHI



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PASSAGES Revealing the nature of exoplanets

BY RON COWEN

leven years ago, David Charbonneau was a new graduate student at Harvard University's astronomy department, eager to explore the birth of the universe. "Then I learned of the incredible first discoveries that had just been announced in exoplanets," he recalls. Those objects, the first planets found outside the solar system, prompted Charbonneau to drop the Big Bang like a hot potato. He's been hunting for exoplanets ever since.

Yet the orbs that piqued the imagination of Charbonneau and so many other astronomers in the mid-1990s were then little more than phantoms. Too small to be seen, each planet revealed its presence only because its gravitational pull made its parent star wobble a little. Astronomers could ascertain just two basic properties of each of these elusive planets: a minimum value for its mass and the time it takes to orbit its star.

Seven years ago, Charbonneau and his colleagues brought the first of these alien worlds out of the shadows. They measured how much light the planet blocked when it passed in front of, or transited, its parent star. Such minieclipses reveal a planet's true

size and mass, while the filtering of starlight by its atmosphere shows what gases cling to the alien world.

Scientists have now observed the transits of 20 planets. Astronomers have been able to measure the heat emitted by four of the planets and in one case have gone even further, constructing the first temperature map of an exoplanet.

Now, a mother lode of new transit observations is within reach. Two space missions, one launched late last year and the other scheduled for takeoff in late 2008, are likely to find hundreds of planets showing transits. Next year's mission offers the prospect of finding the first exoplanet that's the same size as Earth.

"Transits ... offer insights that cannot be gained by [the wobble method] alone," says theorist Alan Boss of the Carnegie Observatories of Washington (D.C.).

"We want to know what planets are made of—whether they're made of rock or [are] giant balls of gas," says Charbonneau. Transits provide that information.

PLANETARY PASSAGES In the standard technique for finding an exosolar planet, astronomers analyze the light from stars, searching for periodic shifts in wavelength that result from the star wobbling ever so slightly as it moves through space. Those wobbles betray the presence of an unseen planet that's pulling the star to and fro. The method has a natural bias for finding massive planets that lie close to their stars. Nevertheless, in the late 1990s, astronomers were astonished to find dozens of planets nearly as heavy as Jupiter whipping around their stars in orbits less than onetenth the size of Mercury's orbit around the sun.

Such close-in planets, lost in the glare of their parent stars, can't be photographed. But a massive, tightly orbiting planet makes an ideal candidate for transit studies, realized astronomer Tim Brown, now director of the Las Cumbres Observatory Global Telescope Network in Goleta, Calif.

To create a minieclipse, a planet must pass in front of its star, from the viewpoint of an observer on or near Earth. The closer a

planet lies to its star, the less precise that alignment needs to be. Moreover, a fatter planet will block more starlight than a smaller one, making a transit easier to spot. Still, the effect is tiny: A large, closein planet might blot out about 1 percent of a star's light, producing an effect comparable to a mosquito flying in front of a flashlight a few thousand kilometers away.

> With that in mind, Brown and a colleague cobbled together a tiny, 3.5-inch telescope from spare parts and installed the device in a converted chicken coop on a friend's farm just north of Boulder, Colo. By the time graduate student Charbonneau joined him in 1999, Brown had built a new 4-inch tele-

scope, this one set up in a parking lot at the National Center for Atmospheric Research in Boulder.

Charbonneau came not only with enthusiasm but also with some intriguing information: An international team of astronomers had just used the wobble method to find tentative evidence of a giant, Jupiterlike planet circling close to a near-Earth star called HD 209458. Since Brown's telescope needed to be tested anyway, why not point it at the star and hope that its planet might transit?

Brown and Charbonneau monitored the star for 2 months but were too busy with another project to immediately analyze the data. When they did, they found that their telescope had indeed found a transiting planet. It blocked about 1 percent of HD 209458's light.

"We were really startled," says Brown. "The first time you use a telescope, you wind up a winner—that just doesn't happen." Information from the transit, combined with the data from the wobble method, revealed that the planet around HD 209458 is two-thirds as massive as Jupiter and that it has a diameter 32 percent larger.

Next, Charbonneau used a spectrograph aboard the Hubble Space Telescope to record the wavelengths of starlight absorbed when the planet passed in front of HD 209458. The observations showed that while some starlight was entirely blocked by the planet, another portion was merely attenuated. That proved what astronomers had always suspected: The planet wasn't a solid body, but, like Jupiter, was a puffy ball of gas with an extended atmosphere.

MAP QUEST — The first map of an exoplanet shows temperature variations across the cloud tops of a hot, Jupiterlike orb called HD 189733b. The map reveals a hot spot on the side of the planet that always faces its star.

Starlight filtering through the planet's atmosphere indicated a small population of sodium atoms. It was the first time that anyone had detected a planetary atmosphere beyond the solar system and gotten a whiff of its composition (*SN: 12/1/01, p. 340*).

"Everyone had assumed that if you wanted to [detect] the atmosphere of an extrasolar planet, you'd have to image it," says Charbonneau. But the transit method provided that information without the need of a snapshot.

It took several years for astronomers to detect another transiting planet. There was no dearth of intriguing signals, notes Brown, but most of them proved to be spurious—eclipses of one star by another star, not by a planet. Now, with several arrays of telescopes set up across the globe—the Trans-Atlantic Exoplanet Survey in Arizona, California, and the Canary Islands; the Optical Gravitational Lensing Experiment in Chile; the Hungarian Automated Telescope Network in Arizona and atop Mauna Kea in Hawaii; the Wide Angle Search Experiment in the Canary Islands; and the XO project in Maui—transit information is flooding in.

These small, automated telescopes stare at large fields of stars over as many consecutive nights as possible, usually for about 2 months at a time. Because the devices pre-

2 months at a time. Because the devi cisely measure a star's brightness, astronomers can detect the telltale, periodic dimming that's due to a transiting planet. The surveys have now discovered 16 transits among stars not previously known to have planets orbiting them.

HOT TOPICS The 2003 launch of NASA's infrared Spitzer Space Telescope dramatically increased the information that astronomers can glean from transits. Planets emit most of their light in the infrared part of the spectrum. When detectable, that radiation provides a measure of a planet's surface temperature.

The Spitzer telescope can't directly image a planet, so researchers employed a trick to

separate the planet's infrared emissions from those of the star. First, they used Spitzer to measure emissions from the star and planet when the two were side by side. They measured again when the planet went behind the star. By subtracting the second measurement from the first, researchers determined how much infrared light was emitted by the planet alone.

Having used an elaboration of this technique, Heather Knutson of Harvard University and her colleagues report in the May 10 *Nature* the first temperature map of the atmosphere of an exoplanet.

The astronomers focused their attention on a Jupiterlike gas giant that circles the star HD 189733 every 2.2 days. During 33 hours of Spitzer telescope observations, Knutson and her colleagues recorded the infrared light emitted by the planet just after it emerged from behind the star. Infrared brightness varied slightly because the planet rotates, Knutson explains. The team used those variations to define a series of longitudinal strips, from the orb's north pole to south pole, depicting the uneven infrared brightness of the planet's upper atmosphere.

Among the cloud-top features, the map reveals a hot spot about 30,000 kilometers wide, or roughly 1.5 times the diameter of Earth. Intriguingly, the hot spot is offset by about 30° in longitude from the region of the planet closest to the parent star, the spot that would receive the highest intensity of radiation.

Knutson and her colleague suggest that the offset has come about because strong winds redistribute the planet's heat. Blowing with speeds up to 10,000 kilometers per hour, the proposed winds would be about 30 times as strong as any wind on Earth.

Other measurements, reported by Giovanna Tinetti of the Insti-

PLANETARY FINGERPRINTS — When an exoplanet passes in front of its parent star, starlight filters through the planet's atmosphere (small halo in illustration) and reveals the gases there. The change in infrared brightness of the planet-star system when the planet dives behind the star and then reappears indicates the planet's heat.

tute of Astrophysics in Paris and her colleagues in the July 12 *Nature*, suggest that the planet's atmosphere contains water vapor.

In the spring of 2009, the Spitzer telescope will run out of coolant and will no longer be able to make observations at the longest infrared wavelengths, where emissions from planets are brightest. However, the telescope will still register radiation from transiting planets at shorter infrared wavelengths.

Spitzer can examine only worlds that emit copious infrared radiation and are far too hot to sustain liquid water or life. However, the proposed successor to Hubble, the James Webb Space Telescope, scheduled for launch in 2013, will have the capability to record the much fainter infrared emissions from Earthlike worlds.

GETTING SMALL Astronomers have now found 13 exoplanets that, according to wobble measurements, may be no more massive than Neptune. For those small orbs that happen to produce transits, astronomers hope to determine whether the bodies are composed mostly of gas like Jupiter, contain mostly ice like Neptune, or are rocky, giant versions of our own planet.

Indeed, Swiss researchers announced this spring that they had discovered the smallest transiting planet

yet, a body only 22 times as heavy as Earth, similar in mass to Neptune. Every 2.6 days, the planet whips about GJ 436, a dwarf star considerably dimmer than the sun. These transit observations prove that there are exoplan-

ets similar in structure to Neptune but warmer, Charbonneau says. The planet isn't habitable: Water would be steam at its surface and a compressed solid below (*SN: 5/19/07, p. 308*).

But the parent star's dimness also means that the close-in planet isn't as blisteringly hot as it would be around a sunlike star. Searching for transiting planets around dwarf stars could reveal a truly habitable planet, says theorist Sara Seager of the te of Technology

Massachusetts Institute of Technology.

Two new missions are likely to aid in that search. Last December, the European Space Agency launched a spacecraft called COROT, which will survey 120,000 stars to look for transiting planets with sizes down to twice that of Earth.

Scheduled for launch in early 2009, NASA's Kepler mission will feature a larger telescope, just under a meter in diameter, that will have the capability to detect and study hundreds of transiting planets that are Earth's size or even smaller. With a large field of view and an orbit around the sun rather than Earth, Kepler will have an unobstructed view of the heavens, enabling it to repeatedly monitor the brightness of 100,000 stars during its 4-year mission.

Kepler scientists calculate that the telescope should be able to detect the transit of 50 planets about the size of Earth. If most of these star-eclipsing bodies have a diameter 30 percent larger than Earth's, the number of detections could rise to 185 planets. The latter number would more than triple if the typical transiting planet had a diameter about double that of Earth.

The researchers base their predictions on the assumption that most stars have planets but that intrinsic variability in the brightness of some stars would make transits difficult to detect. To have confidence in a detection, the team will require that a planet make at least four transits during Kepler's 4-year mission.

"We may be witnessing the start of a cosmic real estate boom," says Jill Tarter of the SETI Institute in Mountain View, Calif. "Hopefully, Kepler will be able to tell us about the frequency of truly Earth-size planets around stars of varying types within the next few years." ■

BRAIN ATTACK

Progress is slow in finding better ischemic-stroke therapies

BY ANDREAS VON BUBNOFF

bout a year ago, E. Gail Anderson Holness was in church when she suddenly felt lightheaded. At first, the then-49-year-old minister, motivational speaker, and Washington, D.C.-based writer didn't think that the episode was serious, even though she'd had

headaches for several days. She was healthy, after all, and she ran 4 miles almost every day.

"Because I am an athlete, I could not understand what was happening to me," she says. "I said, 'Just let me go home." But when her speech became slurred and the left side of her face started to droop, a friend convinced her to go to the hospital.

Anderson Holness had had an ischemic stroke, the type caused by a blocked blood vessel in the brain. The stroke killed some of her brain tissue by cutting it off from its oxygen and nutrient supplies.

About 600,000 people suffer ischemic strokes each year in the United States. The other kind of stroke, which occurs when blood spills into the brain, affects 100,000 people a year. With more than a million survivors facing varying degrees of permanent damage, strokes are the leading cause of serious, long-term disability in the country.

Still, there is currently only one drug treatment approved by the Food and

Drug Administration for use at the time an ischemic stroke occurs. That drug is tissue plasminogen activator (tPA), given intravenously to dissolve blood clots. FDA approved the famous "clot buster" in 1996.

Treatment with tPA saves many patients—as it did Anderson Holness—by opening obstructed blood vessels in their brains. But the drug has severe limitations. Doctors have to first do a computerized tomography scan to make sure that the person isn't experiencing a bleeding stroke, which tPA would make worse. And the clot-busting drug must be administered within 3 hours of the onset of a stroke—which isn't always possible because many delays occur on the way to emergency treatment. After 3 hours, the risk of bleeding outweighs the potential benefit of tPA, at least according to current medical judgment. The potential complications have made emergency room physicians reluctant to give tPA, says Costantino Iadecola, chief of the division of neurobiology at Weill Cornell Medical College in New York City.

As a result, less than 5 percent of ischemic-stroke patients in the United States get treated with tPA. "Very few patients can benefit from the only treatment we have available for stroke," Iadecola says. "There is a tremendous interest to develop new treatments."

Indeed, trials of many treatments are under way. Some test alternative clot busters that may be less likely than tPA to provoke bleeding. Other studies focus on drugs that keep brain cells from

> dying once a stroke has occurred. Some researchers are using genetics to look for yet more such neuroprotective compounds to test.

> Other scientists are trying combinations of drugs because each drug typically targets just one or a few of the many processes that lead to brain-cell death after a stroke.

> All in all, researchers have come up with a number of approaches that have shown promise in animal studies. Even so, making such treatments work in people has proved to be a stubborn problem.

> **BUSTING OUT** Given that clot busting is the only approved and effective treatment for ischemic stroke, some researchers are trying to expand on it. For example, Anand Vaishnav of the University of Kentucky Medical Center in Lexington and his stroke team advise doctors at rural hospitals on whether tPA will help or hurt a particular stroke patient.

> Many doctors are insufficiently trained or too conservative to use the drug, Vaishnav explains. "We'll give them the green signal to give tPA" when it's

appropriate, he says. After a preliminary study, Vaishnav concludes that a national telemedicine system, which would provide instant expert advice to any location, could increase the proportion of ischemic-stroke patients getting tPA to 10 or even 20 percent.

Other doctors are starting to extend the time following a stroke during which tPA can be used, using brain imaging to show that a person still has salvageable tissue in the damaged area. Last year, a team led by researchers at Stanford University published a study suggesting that such patients can benefit from tPA treatment up to 6 hours after a stroke.

Still others are using ultrasound to make tPA more effective. In a small clinical study published in 2004, Andrei Alexandrov, now at the University of Alabama at Birmingham, found that applying ultrasound through the skull triples the effectiveness of tPA in dissolving ischemic-stroke clots. The sound waves agitate



area at right in this computerized tomography brain scan was damaged by an ischemic stroke that occurred about 6 hours earlier. Smaller dark area, circled at left, represents damage from an older stroke.

the stagnant blood in the blood vessels affected by the stroke, Alexandrov says, allowing tPA to better reach and penetrate the blood clot. "It's like a spoon that stirs sugar in a cup of tea," he says.

More recently, Alexandrov has been working with ImaRx Therapeutics of Tucson, Ariz., which makes tiny, gas-filled bubbles by enclosing minute quantities of perfluoropropane in fatty shells that are similar to cell membranes in humans. Administered intravenously, the bubbles aren't dangerous, he says, because perfluo-

ropropane is chemically inert and present in only small amounts. But when the bubbles reach the site of stroke damage, ultrasound vibration makes them oscillate or even explode. This further agitates the stagnant blood around a clot, Alexandrov says. In February, he told the International Stroke Conference in San Francisco of early success combining tPA, ultrasound, and gas bubbles to dissolve clots in stroke patients.

Until recently, many doctors had high hopes that a drug called desmoteplase, derived from the anticlotting saliva of vampire bats, would prove to be an effective clotbusting alternative to tPA. Last year, a study funded by the biopharmaceutical company PAION, based in Aachen, Germany, reported in *Stroke* that desmoteplase appeared to clear brain blood clots in patients up to 9 hours after a stroke, without causing excessive bleeding.

In late May of this year, however, PAION announced that the drug didn't show significant benefits in a larger trial. Success might have positioned desmoteplase for FDA approval within a few years, says Marc Fisher of the University of Massachusetts Medical School



VICIOUS BLOCK — Foreground illustration shows a blood clot lodged in a cerebral artery, the location of which is diagrammed in the brain at rear. Such an event, an ischemic stroke, starves tissue of oxygen and kills brain cells unless treatment can quickly clear the blockage.

in Worcester. "It's very disappointing that it didn't show anything," Fisher says. "That was the only promising thing. Now there is nothing."

FOR PROTECTION Since therapies that restore blood flow in the brain have limitations, scientists have for decades also searched for means to protect neural cells while they're starved of blood and oxygen. Ideal neuroprotective drugs would interfere with the stroke-related events that cause those cells to die.

By now, researchers have a pretty complete understanding of these events, Iadecola says. As soon as blood flow is interrupted, nerve cells in the immediate area of the blockage start dying for lack of oxygen and energy-providing glucose. Most of the cells at the core of a stroke can't be rescued unless blood flow is reestablished within a few minutes.

The area around the core still has some blood flow and remains salvageable for hours, Iadecola says. But dying cells in the core make matters worse by releasing glutamate, a chemical that normally transmits nerve signals.

In this case, glutamate is dangerous because it triggers brain cells outside the stroke core to open critical pores to an influx of calcium ions. That starts a wave of inflammation, free radical production, and DNA and protein destruction that leads, about a day after the stroke, to the activation of a cell-suicide program called apoptosis.

Researchers have been looking for compounds that target these

processes by soaking up free radicals, for example, inhibiting the effects of calcium and glutamate, or fighting inflammation. In March of last year, David Howells of the University of Melbourne in Australia and his colleagues reviewed in the *Annals of Neurology* about 1,000 animal studies and 100 human trials that have tested such compounds, as well as clot busters, since 1957.

But so far, none of the neuroprotective compounds that worked in animals or lab-cultured cells has benefited stroke patients, says

> Ashfaq Shuaib of the University of Alberta in Edmonton. "Everything was negative," he says. "The record is pretty bad."

> Shuaib says that researchers tried to improve the situation by establishing quality criteria for testing stroke drugs in 1999. The Stroke Therapy Academic Industry Roundtable (STAIR) standards declared, for example, that a drug should be tested in animals in several labs, including ones other than that of the company developing the drug; that it should be tested in several animal species, including primates; and that the drug's physical and behavioral effects should be monitored for weeks after an animal undergoes an induced stroke.

One current study that follows most of the STAIR criteria is testing infusions of magnesium sulfate. Magnesium can keep calcium from rushing into cells. The drug can be given even before a patient reaches the hospital because it carries no known risk of increased bleeding. A team at the University of California, Los Angeles School of Medicine is studying the effects of magnesium sulfate given within 2 hours after a stroke. So far, 300 patients have received the infusions, says Anna

Yanes, the chief nurse coordinator of the trial. Another 1,000 patients will be recruited, and indications of the long-term effects of the treatment should emerge in 2 to 4 years.

But the STAIR criteria don't seem to be enough. That became clear last October, when the drugmaker AstraZeneca announced that the first trial in people of a drug that satisfied all STAIR criteria had failed. The trial found no significant benefit to stroke patients from an antioxidant called NXY-059, which was intended to inhibit damage by free radicals. As a result, AstraZeneca abandoned its efforts to seek FDA approval for the drug.

"We were devastated, really disappointed," says Shuaib, who was the lead investigator of the trial.

Meanwhile, researchers are trying cutting-edge approaches to finding new neuroprotective-drug candidates. The drug company Wyeth, for example, is analyzing dying nerve cells to determine what genes and proteins are active in them. The company's scientists plan to screen libraries of chemicals in search of compounds that modulate these genes or proteins and so might keep cells from dying, says Giora Feuerstein, who heads Wyeth's department of discovery and translational medicine in Collegeville, Pa.

Seong-Seng Tan of the Howard Florey Institute for brain research in Melbourne, Australia, and his colleagues are also looking for proteins that help cells survive. Tan's group has identified about a dozen proteins that become more active in brain cells that survive experimental strokes in animals.

One of them is a mouse protein called BP5. It's 10 times as abun-

dant in brain cells that survive a stroke as in other cells in the same area. Further animal experiments showed that BP5 can keep nerve cells alive in strokelike conditions. Tan says that he hopes to find a drug that can activate or mimic BP5.

COMBO LESSONS The NXY-059 failure illustrates another reason why it has been so difficult to find neuroprotective drugs: Many candidate compounds affect only one or a few of the mechanisms that kill brain cells after a stroke, Shuaib says. "A single drug is not really the answer," he says.

So Shuaib and others have turned to treatment combinations. The Canadian researcher recently gave each of nine stroke patients six treatments normally given in isolation or in limited combinations. Within 3 hours of their strokes, the volunteers received tPA, and for 12 hours they wore helmets that cooled their brains by about 3°C. The cooling slows many processes that cause nerve cells to die.

The other treatments were an infusion of albumin to inhibit free radicals and improve blood flow; a statin drug, also for improving blood flow; an anti-inflammatory agent; and a day of magnesium infusions. Shuaib says the test established the safety of the overall therapy but that it wasn't designed to determine its effectiveness at protecting brain cells. The combination treatment has protected brain cells in animals, however.

James Grotta of the University of Texas in Houston combines cooling with an infusion of the equivalent of Irish coffee: caffeine and ethanol in a combination researchers call caffeinol. Caffeine, Grotta says, blocks dying cells' glutamate release, and ethanol blocks glutamate's action on surviving cells. Both compounds are cheap, their side effects are well-known, and they can easily enter the brain, he says.

In rats, a caffeinol infusion 2 to 3 hours after an induced stroke can reduce the brain area in which cells die by about 60 percent, compared with the area in animals receiving no treatment. If the regimen includes cooling the animals by 2°C, the area of dead cells

is reduced by 80 percent. A person would have to get the equivalent of two cups of strong coffee and one cocktail to get a dose equivalent to what the animals received, Grotta says. He's currently testing the treatment's safety in people.

DON'T DELAY Stroke researchers seem to be a long way from any breakthrough in stroke treatment, with current progress measured in small increments. For instance, doctors physically removed

"A single drug is not really the answer."

– ASHFAO SHUAIB. UNIVERSITY OF ALBERTA IN EDMONTON

blood clots from the brains of about 2,000 stroke patients last year in the United States using a device called the Merci (Mechanical Embolus Removal in Cerebral Ischemia) Retrieval System made by Concentric Medical in Mountain View, Calif. During this procedure, a catheter threaded from the groin into a brain artery is used to deploy a retriever that snags and pulls out the clot. Catheters inserted in this way can

also deliver tPA directly to where it's needed.

The FDA has approved such devices for safety, but there's no solid evidence that any of them benefit stroke victims, says S. Claiborne Johnston, the director of the stroke service at the University of California, San Francisco. What's more, a Merci device can cause bleeding, he says.

With tPA still the only sure ischemic-stroke treatment available, doctors and researchers emphasize the importance of getting victims to the hospital quickly. That gives a person a chance to receive tPA and prevent the death of more and more brain cells over several hours. "Time is brain," Wyeth's Feuerstein says.

Anderson Holness, for her part, is glad to be out of a wheelchairthe stroke had paralyzed her left leg for a few weeks, but she's now back to running every day. She says the specialist told her,"It's a good thing they didn't take you home. You would not have survived."

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OF NOTE

BIOMEDICINE Mouse method turns skin cells to stem cells

Scientists have reprogrammed mouse skin cells to mimic embryonic stem cells that can morph into any type of cell in the body. Last year, Shinya Yamanaka and his

team at Kyoto University in Japan found that they could insert into skin fibroblast cells active copies of four genes earlier identified as essential to a stem cell's pluripotency the ability to turn into any cell. But the researchers had trouble isolating the less than 0.1 percent of skin cells that became fully reprogrammed by the added genes.

Yamanaka and his col-

leagues now describe online and in an upcoming *Nature* how they also inserted a novel marker gene that singled out the completely reprogrammed stem cells. The team implanted some of those selected cells into mouse embryos, where they acted as stem cells and grew into adult mice carrying DNA from the inserted genes. Two other research groups corroborated the results.

"It's one of the most exciting findings in recent years," says Marius Wernig of the Whitehead Institute of Biomedical Research in Cambridge, Mass., coauthor of one of the corroborating studies, which also appears in *Nature*. "It's really hard to believe that [cells] can be reprogrammed so easily."

However, scientists haven't yet shown that human cells can be reprogrammed in a similar way, Wernig cautions. Furthermore, 20 percent of Yamanaka's mice died of cancer brought on by two of the genes. —C.B.

PLANETARY SCIENCE Dust delays Martian rover

For 9 months, the Mars rover Opportunity has inched along the perimeter of an 800meter-wide crater called Victoria, while NASA scientists debated where—and whether—the robot should attempt a descent into the 70-m-deep hole. Although the downhill journey is risky, the payoff is great: The farther the rover travels, the older the rock it can study in the crater's walls and the more insights it can gain into the Red Planet's apparently wet ancient past. Last month, NASA announced that Opportunity would begin crawling down the crater at a place called Duck Bay, which features several gentle slopes.

But now the rover is waiting for the dust to clear. In late June, a giant dust storm began brewing on Mars, reducing the sunlight that powers both Opportunity and its

> sister rover, Spirit. The amount of dust in the atmosphere at Spirit's location, Gusev crater on the opposite side of the planet, remains lower than at Victoria crater. Opportunity won't embark on its descent until July 13 at the earliest, says rover scientist Steve Squyres of Cornell University.

> Researchers are using images from the Mars Reconnaissance Orbiter

to monitor the storm and plan rover operations. Both rovers have been operating on Mars since January 2004, far exceeding their expected 3-month lifetimes. -R.C.

Gooey solution to a sticky problem

Researchers in Japan have extracted a new, gooey, and potentially useful protein from the bodies of jellyfish. The sugarladen molecule, a member of the mucin family, is similar to proteins found in human mucus and other natural lubricants and protective coatings.

Booming jellyfish populations have caused serious problems in waters around Japan and elsewhere, as the animals' bodies have tangled with ships' propellers and clogged coolant lines in coastal power plants. Hoping to offset the expense of removal, Kiminori Ushida and his colleagues at the Riken Institute of Physical and Chemical Research in Wako and Shinwa Chemical Industries in Kyoto looked for a commercially valuable product from the sea creatures.

The researchers identified the new protein in a goo that they extracted from the carcasses of five jellyfish species, they report online in the *Journal of Natural Products*. They named the protein quiumucin, from a Japanese word meaning local rebirth.

Mucin molecules, laden with complex carbohydrates, are difficult to synthesize in the laboratory. Mucins derived from pigs and cows are widely used in cosmetics, food additives, and drugs. Some have antibiotic properties. Considering the massive number of jellyfish bodies available, the animals could be an important new source for such proteins, Ushida says. He and his colleagues extracted up to 2 grams of qniumucin from every 10 kilograms of wet jellyfish and hope that small-scale production will begin within the next year.

With a simpler carbohydrate structure than that of other animal mucins, qniumucin could serve as a foundation for more-complex synthetic mucins tailored to specific applications, Ushida says. —S.W.

Hepatitis B drug creates HIV resistance

In people infected with both the hepatitis B virus and the AIDS virus HIV, a widely used treatment for hepatitis also causes HIV to develop drug resistance, scientists report.

Chloe Thio of the Johns Hopkins University School of Medicine in Baltimore and her colleagues studied HIV infection in three patients who were taking the drug entecavir to treat hepatitis B, a virus that attacks the liver. The scientists found that entecavir reduced the amount of HIV in all three patients' bloodstreams.

Entecavir combats hepatitis B by inhibiting an enzyme called DNA polymerase, which plays a central role in viral replication. Thio and her team found that the drug also stymies a similar enzyme, RNA reverse transcriptase, that HIV uses to copy itself.

The team then discovered that one of the patients developed a mutated HIV strain known to resist a variety of anti-HIV drugs. To confirm the change, the scientists engineered the mutation into a lab strain of HIV and showed that it resisted two common anti-HIV drugs.

"We need to really be sure that [hepatitis] drugs don't have HIV activity so they don't harm a patient's chances of being able to have an optimal response to HIV therapy in the future," Thio says. Roughly 10 percent of people worldwide with HIV also have hepatitis B.



MIGHTY RARE MOUSE This mouse was produced from reprogrammed skin stem cells that acted like embryonic stem cells.

DGDEN/WHITEHEAD INST.

OF Note

The case study, reported in the June 21 *New England Journal of Medicine*, prompted the Food and Drug Administration's Treatment Guideline Panel to revise its label for entecavir, cautioning doctors not to prescribe the drug in a patient with HIV until that person has already started receiving drugs specifically targeting HIV. —C.B.

EARTH SCIENCE As the last ice age waned, a great lake

was born

During the final millennia of the last ice age, what was briefly the world's largest lake sat along the southwestern edge of the ice sheet that smothered eastern Canada. Researchers have now determined when that lake first formed.

At one point, Canada's Lake Agassiz contained about 30 percent more water than all freshwater bodies of the world hold today (*SN: 11/2/02, p. 283*). Numerous studies have suggested that the lake disappeared about 8,400 years ago, when the center of the Laurentide Ice Sheet collapsed into Hudson Bay, allowing the lake to drain into the ocean. Despite widespread agreement on the timing of Lake Agassiz' demise, researchers hadn't pinned down when the lake originated, says Kenneth Lepper, a geoscientist at North Dakota State University in Fargo.

He and his colleagues carbon-dated samples of wood retrieved from sediments in lakes that had formed atop the rocky debris, or moraine, that had been pushed south by the advancing ice sheet. Those dates are clustered around 13,950 years ago, the team reports in the July *Geology*. That's the time when the Laurentide Ice Sheet began to melt and retreat from northern portions of the Great Plains, the team suggests. The nascent Lake Agassiz accumulated between the retreating ice sheet and the moraine, says Lepper. —S.P.

BIOTECHNOLOGY More bang for the biofuel buck

Microbes that ferment ethanol from glycerol, a by-product of biodiesel production, could add an economically valuable

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new ingredient to the biofuel industry, researchers report.

Industrial plants that make biodiesel by processing vegetable oils and animal fats churn out about 10 kilograms of glycerol for every 100 kg of fuel. Increasing biodiesel production has generated a glycerol glut, with much of this previously useful product now going to waste. Its market value has plummeted by 90 percent since 2004.

But certain microbes can anaerobically ferment glycerol into biofuels and other marketable chemicals, say Ramon Gonzalez and his colleagues at Rice University in Houston in the June *Current Opinion in Biotechnology*.

The researchers tweaked growth conditions for *Escherichia coli* so that the bacteria efficiently produced ethanol from glycerol in a benchtop fermenter. Although the process is currently about 40 percent slower than yeast fermentation of ethanol from corn, Gonzalez adds that genetic engineering of the bacteria could make fermentation faster. Other microbes can ferment glycerol to make useful industrial chemicals such as succinic acid and 1,3-propanediol, he says.

If the new process can be made to work on an industrial scale, it could enhance the economics of the biodiesel industry. "In one single plant they will get two biofuels—the biodiesel and the bioethanol," Gonzalez says. —S.W.

BIOTECHNOLOGY Adding to nature's repertoire

Out of thousands of possible kinds of amino acid, virtually all organisms use just 20 to build the proteins they need. Now, scientists have expanded this palette by coaxing some cells from mice to make proteins that include syn-

thetic amino acids. The researchers have

already used the customized proteins to study basic cell biology, but the technique could someday make novel proteins for medicine or biotechnology, says study coleader Lei Wang of the Salk Institute for Biological Studies in La Jolla, Calif. "With a much larger set of amino acids, you can introduce novel biological properties into cells," Wang says.

To use an amino acid, a cell must first attach it to a molecule of a type called transfer RNA (tRNA). One end of a tRNA grips the amino acid while the other end matches up with a specific sequence in the cell's genetic code. The researchers needed a tRNA that could hold the artificial amino acid as well as enzymes that would make the two molecules link up.

To prevent interference with the cell's own tRNAs, Wang's team used *Escherichia coli*'s bacterial tRNA, which operates somewhat differently than the mammalian variety does. They then tested billions of slightly different enzymes to find ones that would attach the tRNA to the three kinds of synthetic amino acids used in the study. After the researchers inserted the genes for the tRNAs and the enzymes into cultured mouse-nerve cells, the cells made proteins that incorporated the artificial amino acids. The results are in an upcoming *Nature Neuroscience*. —P.B.

GENETICS Anemone reveals complex past

Animals evolved complex genomes surprisingly early, new research shows. The recently completed genome of the starlet sea anemone, a primitive animal that last shared an ancestor with humans and other vertebrates about 700 million years ago, has a greater number of genes in a more complex arrangement than scientists had expected.

"The assumption was that the more complex an animal you are, the more complex your genome must be," says study coauthor Mansi Srivastava of the University of California, Berkeley. People have about 20,000 genes, while fruit flies, a relatively primitive animal widely used for genetics research, have only about 14,000 genes. Srivastava's team expected the starlet sea anemone, which belongs to an even more primitive group of animals that includes jellyfish, to have still fewer

genes. But when they sequenced its genome, the scientists found that it contains roughly 18,000 genes, they report in the July 6 *Science*.

The anemone genome also contains many segments of noncoding DNA that must be delicately spliced out of genes when they are transcribed to RNA. The study suggests that this sophisticated genetic feature—one that scientists thought occurred

primarily in higher animals—was present even in the ancient common ancestor of people and anemones.

"It challenges our ideas about what it means to be a complex organism," Srivastava says. —P.B.



NOT SO PRIMITIVE The

starlet sea anemone has an

unexpectedly complex genome.

Books

A selection of new and notable books of scientific interest

GLUT: Mastering Information through the Ages ALEX WRIGHT

As people become increasingly connected through digital means and a flood of information is readily



available through the Internet, the idea of information systems has gained greater visibility. In this historical account, Wright provides evidence to support his assertion that the concept of information systems is not a modern construct. The author, a journalist and an information architect, looks at the concept of

networked information systems from the vantage point of evolutionary biology and cultural anthropology and concludes that the roots of these systems lie in monasticism, mythology, and modern print technologies, as well as in the rise of computers. What's more, people are not the only organisms capable of creating networks and hierarchies to store information. Wright examines, for instance, the formation of networks and hierarchies in fish and insect colonies. He also recounts preliterate peoples' efforts to develop language and taxonomies. These early attempts at classifying the world, he asserts, are evidenced in the formation of mythology and of symbolic communication through beads, which evolved into written alphabets, books, and, finally, libraries. Religious texts, as well as secular literature, flourished during the Middle Ages, and the emergence of the scientific method led to a plethora of scholarly writing and encyclopedias in 17th-century Europe. The 18th century ushered in discoveries about the natural world that resulted in Carolus Linnaeus' taxonomy. Wright concludes by describing modern achievements in classification, such as the Dewey decimal system and the precursors to the World Wide Web. He suggests that the Internet's fluid, networked structure may be the ultimate solution to communication and to the efficient organization of information. Joseph Henry Press, 2007, 286 p., hardcover, \$27.95.

JACQUARD'S WEB: How a Hand Loom Led to the **Birth of the Information Age** JAMES ESSINGER

It seems unlikely that a loom would spark the computer age. But, as Essinger explains, French inven-

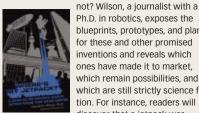


tor Joseph-Marie Jacquard did just that with his amazing machine. After presenting a brief history of weaving, Essinger introduces the man who would revolutionize the entire process. Invented in 1804, the Jacquard loom transformed the silk industry and catapulted its inventor to fame. What made his machine

unique was the use of punched cards that, when strung together, produced the same pattern with each use. Shortly thereafter, mathematician Charles Babbage recognized the potential of Jacquard's idea and put it to use in a computer that he called the Analytical Engine. Essinger describes the intellectual curiosity and acumen that led Babbage to invent this machine, which could be programmed using punch cards. Babbage's device was put to widespread use during the 1900 census of the United States. That device ultimately led to the birth of the modern computer and, in 1924, to the founding of IBM. Oxford, 2007, 302 p., b&w images, paperback, \$16.95.

WHERE'S MY JETPACK? A Guide to the Amazing Science **Fiction Future that Never Arrived** DANIEL H. WILSON

Robotic maids, wholly enclosed cities, and jetpacks. These are among the life-altering advances imagined by science fiction visionaries in the early 20th century. But here we are in the 21st century, and none of these things has been fully realized. Why



Ph.D. in robotics, exposes the blueprints, prototypes, and plans for these and other promised inventions and reveals which ones have made it to market, which remain possibilities, and which are still strictly science fiction. For instance, readers will discover that a jetpack was

indeed invented in 1961 by Bell Aerosystems Company and used in the James Bond film Thunderball. Other inventions under development include selfsteering cars, underwater hotels, X-ray vision, and an elevator to space. Wilson chronicles these devices, spanning fields as diverse as transportation, entertainment, housing, and space travel. This entertaining and humorous guide will appeal to fans of science fiction and cutting-edge fact alike. Bloomsbury, 2007, 192 p., b&w illus., paperback. \$14.95.

TROPICAL PLANTS OF COSTA RICA: A Guide to Native and Exotic Flora WILLOW ZUCHOWSKI

Did you know that the coffee plant has a relative with showy, bright-red flowers that's known as hot lips? Or that guava has been used to treat ailments such as diarrhea and dysentery? These two plants are among those described in this detailed field guide to tropical flora of Costa Rica-a small coun-



try that is home to more than 9,000 native plants. Zuchowski, a botanist and writer, profiles some of the Costa Rica's most common native and introduced species, paying particular attention to those in the Monteverde region. He organizes the plants into general categories, including painted treetops, roadside

and garden ornaments, fruits and crops, and conspicuous grasses. Each entry includes a description of the plant, its English and Spanish common names when available, and information on flowering and fruiting, distribution, and related species. The introduction provides a how-to guide for observing plants as well as background information on Costa Rica and on conservation efforts under way to save the country's tropical forests. The book concludes with a glossary of botanical terms and a list of species by family. Zona Tropical, 2007, 529 p., color images, paperback, \$35.00.

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LETTERS

At least a few years to prepare

"Northern Exposure: The inhospitable side of the galaxy?" (SN: 4/21/07, p. 244) posits that every 64 million years a mass die-off occurs due to increased cosmic rays. When will the cosmic rays again be at their maximum?

ROBERT RICHARDS, METAIRIE, LA.

The article failed to mention when the next cosmic-ray bath is due. Now. I'm worried that it might be so imminent that Science News didn't have the heart to provide its readers the bad news.

STEVE BANKHEAD, WATSONVILLE, CALIF.

Study coauthor Adrian Melott of the University of Kansas in Lawrence says that as we head to the north side of the galaxy, cosmic ray exposure will get gradually more intense and peak in about 14 mil*lion years.* —D. CASTELVECCHI

Fuel me once?

Ethanol is not an alternative to petroleum-based fuel to reduce air pollution ("Not-So-Clear Alternative: In its airquality effects, ethanol fuel is similar to gasoline," SN: 5/5/07, p. 278). It is a growit-at-home alternative to foreign-source petroleum-based fuel. It takes only 2 years to build an ethanol-extraction plant but 10 years to build a petroleum-extraction plant. Right now, as long as ethanol doesn't increase air pollution, I'll take it. LIN DANIEL, CHATSWORTH, CALIF.

I wonder if Mark Jacobson included in his model comparing the air-quality effects of burning ethanol versus gasoline the decrease in automobile gas mileage with ethanol. I regularly track my mileage and routinely suffer a loss of 2 to 3 miles per gallon during the winter months when Albuquerque-area gas stations switch to an ethanol mix. Even if the polluting effects of both fuels were the same, increased efficiency would favor gasoline.

JACKIE ERICKSEN, PLACITAS, N.M.

The article seems to have overlooked that ethanol, from plants, contains carbon directly removed from the atmosphere. JOHN BODENSCHATZ, SAN FRANCISCO, CALIF.

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