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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ MARCH 23, 2013

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Seismicity at a Snail's Pace

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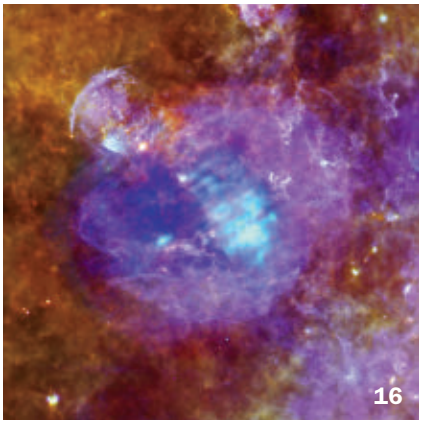

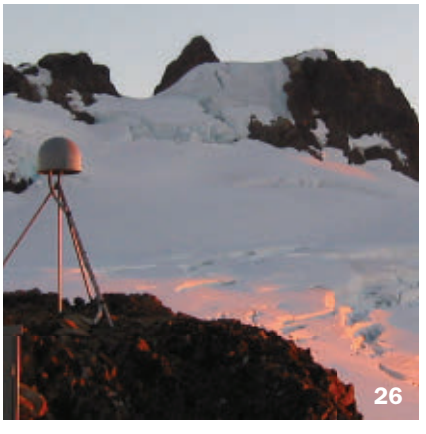

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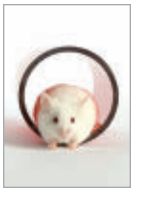

COVER Mice with human immune systems, like this one at the Jackson Laboratory in Bar Harbor, Maine, increase the animals' usefulness in disease research. *Jackson Lab*

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FROM THE EDITOR

Exploring the limits and power of the lab mouse

It's no secret that mice aren't people, and that biomedical studies that are wildly successful in mice frequently don't pan out for patients. Cancer has been cured many times in mice, the refrain goes. Sepsis, a whole-body reaction to infection, is an especially dramatic example of the mismatch between mouse models of disease and actual human illness: Out of nearly 150 clinical trials of candidate drugs to treat sepsis and similar conditions, all of which showed promise in mice, none worked in people.

That led a consortium of researchers to compare mouse and man on a molecular level. As Tina Hesman Saey reports on Page 10, people with sepsis, burns and injuries suffering from inflammatory conditions experienced a fairly consistent set of changes in gene activity. Gene activity in the mice standing in as models for the various conditions, however, was very different from what researchers saw in people. It also varied among the three mouse models. The scientists conclude that, for inflammatory diseases at least, we'd be better off doing more research in people than wasting so much time with lab mice.

But, as Susan Gaidos describes on Page 22, not all mice are created equal. Scientists have been hard at work making mice that will serve as better stand-ins for probing disease and its possible cures. Researchers have gone about it by trying to make the mice a tad more like people: transplanting bits and pieces of human immune systems into mice engineered to lack much of their own native defenses. In the last decade, researchers have created mice with a nearly full array of human immune cells. These animals respond to drugs, viruses and vaccines in ways that much more closely mimic how people respond.

Scientists have even developed ways to make "avatar" mice for individual cancer patients, as Gaidos reports. Though the approach still has its limits (expense and time, for example), such mice enable doctors to test which therapies a patient's actual tumor will respond to. While this has translated into longer lives for some patients, others have had their hopes dashed, learning that no current chemotherapy can successfully shrink their tumors.

Still, the future looks optimistic. As researchers make better, more humanlike mice, the day may come when curing cancer in mice really does mean something for people.

— Eva Emerson, Editor in Chief

PHILIP CHANNING

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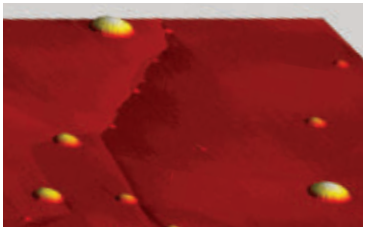

Nanobubble \ *NAH-noh-buh-buhl* \ *n.*

A minuscule gas pocket on the surface of a liquid that can last days before popping. For nearly 20 years scientists have pondered how these bubbles—no larger than 100 billionths of a meter—are able to exist, because their enormous internal pressures should cause them to burst within a second of forming. Now two physicists from the University of Twente in the Netherlands argue that the bubbles survive thanks to a flattened shape that promotes stability and a large amount of dissolved gas in the liquid, which prevents the bubbles' trapped air from seeping out. This explanation, which appears in the Feb. 1 *Physical Review Letters*, could help scientists harness nanobubbles, steering them through the body to deliver drugs to specific organs. —*Andrew Grant*

Science Past | FROM THE ISSUE OF MARCH 23, 1963

VEHICLE ON MOON MAY SINK INTO FLUFFY STUFF — The surface of the moon may be covered with deep layers of fluffy material into which landing vehicles could sink out of sight.... Research so far has shown that loose particles hit by meteoroids settle down into the moon's rock or mineral surface. This surface becomes exposed to radiation and breaks down into fine particles of dust. The dust coagulates into larger and larger clumps.... Observed characteristics of a cement powder have been found to match the reflection characteristics of the lunar surface. The conclusion is that the surface of the moon is likely to be composed of cement powder that has a cobweb-like structure.

Science Future

April 5–6

Texas A&M University hosts a physics and engineering festival. See a bubble show or meet an astronaut and two Nobel laureates. More at bit.ly/SFtamu2013

April 22

Deadline for entries to the Society of Vertebrate Paleontology illustration awards, honoring the best in art depicting dinosaurs and other ancient life. See bit.ly/SFpaleo2013

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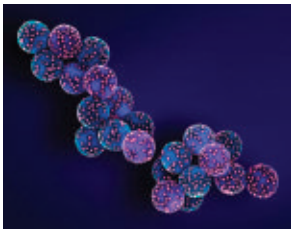
EARTH IN ACTION

Alexandra Witze ponders Earth's odds in "When an asteroid heads for Earth, it's time to reconsider those doomsday plans."

MOLECULES

Nanoparticles (below, blue) detect viruses (pink) in "Synthetic nanomaterial can recognize viruses."



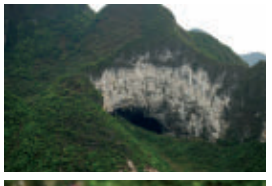
HEALTH & ILLNESS

Allergic people became able to tolerate up to a full glass of milk. See "Therapy for milk allergy offers hope, and caution."

Pregnant women taking an omega-3 fatty acid have bigger babies. Read "Fish oil component boosts newborn health."

Introducing | NEW CAVE-DWELLING NETTLE

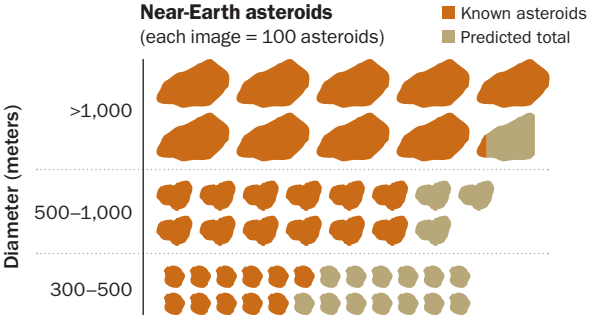
Researchers combing remote forests in southern China have turned up three new plant species, one of which seems to flourish in the low light of limestone caves. The new species are members of the genus *Pilea*, which is part of the nettle family. Alex Monro of London's Natural History Museum and his colleagues, who describe the finds December 28 in *PhytoKeys*, collected two new species from gorges in China's Yunnan and Guizhou provinces (*P. shizongensis* shown, bottom left). The third, appropriately named *P. cavernicola*, has been found only in caves (top left), where some of the plants receive as little as 0.04 percent of full daylight. —*Allison Bohac*



Science Stats | ASTEROID ROUNDUP

The good news: Based on observations by the WISE telescope, NASA estimates that most of the largest asteroids near Earth—the most potentially catastrophic in a collision—have already been spotted. But many more small asteroids remain to be found, and near-Earth asteroids less than 100 meters wide (such as the Russian meteor in February) are poorly observed and could number in the millions.

SOURCE: JPL-CALTECH/NASA



CLOCKWISE FROM TOP LEFT: MICHIEL VAN LIMBEEK AND JOOST H. WEIJS/UNIV. OF TWENTE; ALESSANDRO CUMBO AND MARTIN OEGGERLI; S. EGTS; A.K. MONRO *ET AL/PHYTOKEYS* 2012

“The uncertainty principle has been demonstrated in many different ways, but to see it on a visible mechanical object is totally awesome.” —KEITH SCHWAB, PAGE 16

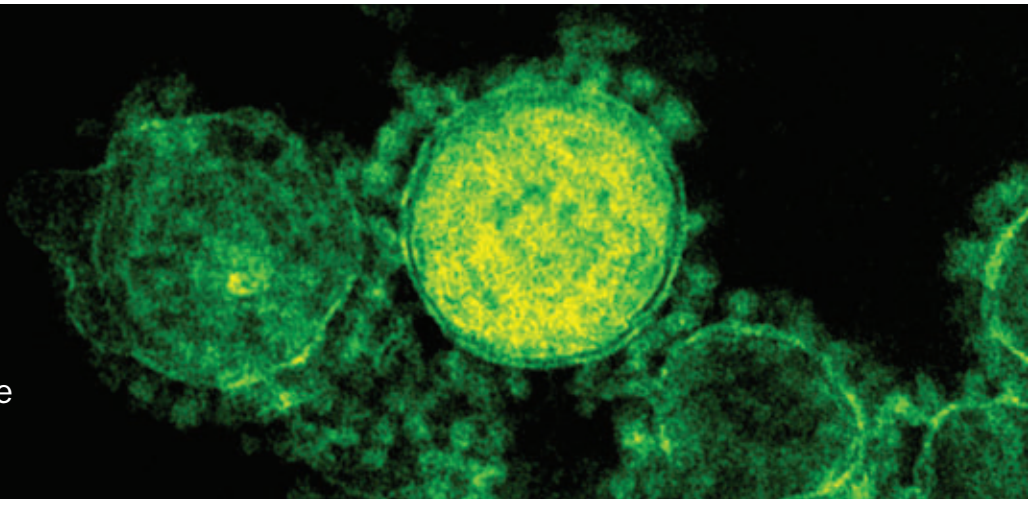
In the News

STORY ONE

Scientists race to understand deadly new virus

SARS-like infection causes severe illness, but may not spread quickly among people

By Tina Hesman Saey



NIAID/NIH

A new type of coronavirus, pictured in a false-color micrograph, has sickened 13 people, seven of whom have died.

WASHINGTON — A deadly new virus has scientists scrambling to learn more about it and figure out whether it will become a pandemic or remain a limited threat.

The virus has sickened 13 people and killed seven of them in the Middle East and England since last April. All but one of those infected were hospitalized with severe pneumonia, and several also developed kidney failure.

“We have a new and virulent virus,” Gwen Stephens, of the Saudi Arabia Ministry of Health in Riyadh, told members of the American Society of Microbiology on February 27 during the annual Biodefense and Emerging Diseases Research Meeting. “We can only guess at its risks.”

The mysterious culprit is a coronavirus, a class that includes the virus that causes SARS, or severe acute respiratory syndrome. SARS spread quickly in 2002 and 2003, infecting some 8,100 people and killing nearly 800.

The new virus is most closely related to coronaviruses that bats carry, but it probably didn’t jump directly from bats to people, Vincent Munster of the National Institutes of Health’s Virus Ecology Unit in Hamilton, Mont., said at the meeting. None of the people who got the disease had direct contact with bats, he said, and the virus is not identical to any known to infect bats.

But W. Ian Lipkin, an epidemiologist at Columbia University, won’t let bats or other animals off the hook yet as possible virus reservoirs. People may have come into contact with animals without realizing it. Such unrecognized contacts have spread the Nipah virus and hantavirus, he said.

Much like SARS, the new virus causes severe pneumonia. But that, Stephens said, is where the similarities between the viruses end.

While SARS passed easily from person to person through the air, the new virus doesn’t seem to spread that way. With the exception of one family, relatives and health care workers who have cared for people sick with the novel coronavirus have not fallen ill, Stephens said. That suggests that people must come into direct contact with the virus, such as by touching something an infected person has coughed or sneezed on.

In February, the first known case of human-to-human transmission of the virus emerged. A 60-year-old England resident who had traveled to Pakistan and Saudi Arabia got sick with the coronavirus and passed it to his son, who died. A young female relative probably also caught the virus from the older man. She had a mild case that didn’t require hospitalization, raising the possibility that other people may have contracted the new virus but mistaken it for the flu or another common respiratory illness, said Alison Bermingham of the U.K. Health Protection Agency.

Still, scientists do not know how people catch the virus or how infectious it is. Also uncertain is whether the new virus could evolve into a SARS-like pandemic, or whether it will slip away as mysteriously as it appeared. “This is a cause for concern, but not alarm,” said Susan

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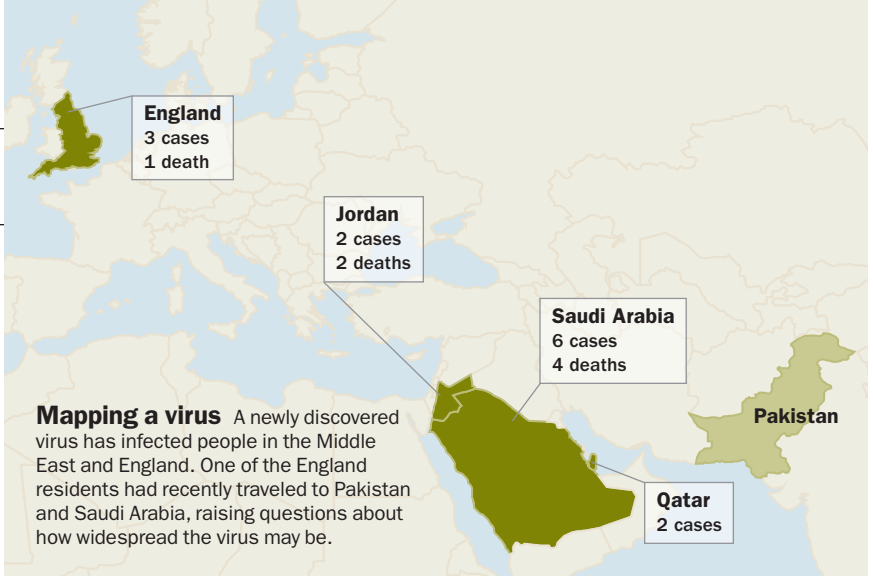
Gerber, a medical epidemiologist at the U.S. Centers for Disease Control and Prevention. "What we do know is that there is no evidence of sustained chains of person-to-person transmission."

To find out how widely the new coronavirus has spread, researchers hope to screen blood from many people for antibodies that would indicate previous exposure to the virus.

Meanwhile, Munster and his colleagues are conducting animal studies to learn how the virus produces illness and perhaps how to counter it. His group tried — and failed — to infect mice and ferrets, both common stand-ins for people in infectious disease studies. Rhesus macaques did get mildly to moderately ill when infected with the novel coronavirus.



Mapping a virus A newly discovered virus has infected people in the Middle East and England. One of the England residents had recently traveled to Pakistan and Saudi Arabia, raising questions about how widespread the virus may be.

These monkeys lost their appetites and developed fevers, goose bumps, rapid breathing and hunched postures, Munster reported. The virus damaged the monkeys' lungs but didn't show up in any other body tissues. The monkey study confirms that the coronavirus isolated by scientists really can cause disease.

To stop the virus, researchers first have to know how it damages the body. The monkey study begins to address that issue. After the monkeys were infected with the virus, the activity of 173 of their genes changed, Munster's group found. Many of those genes are known to fight viruses or produce inflammation. By day six of the infection, the monkeys were already starting to clear the virus out of their bodies, and the activity of all but 37 genes had returned to normal, Munster reported.

No therapies have proven effective against coronaviruses yet, said Eric Snijder, a molecular virologist at Leiden University Medical Center in the Netherlands. SARS was stopped by using old-fashioned quarantines. "We had to resort to 15th or 16th century techniques," he said. "People who had the plague weren't treated much differently."

Some antiviral drugs and vaccines were developed against SARS, but the virus disappeared before researchers got a chance to put the therapies through clinical trials. No one knows if the new coronavirus could be stopped by therapies directed against SARS. The new virus grabs onto a different protein than SARS did to gain entry into human cells, Marcel Müller of the University of Bonn Medical Center in Germany and colleagues reported in the January/February *mBio*.

Snijder and his colleagues are studying how the new coronavirus replicates. If they can figure it out, they may be able to design drugs to interfere with the process. ■

Back Story | VIRUS HUNTING

The discoveries made thus far about the new coronavirus have occurred thanks to serendipity and some public health sleuthing.

In June, Egyptian physician and microbiologist Ali Mohamed Zaki was working at a hospital in Jeddah, Saudi Arabia, when a 60-year-old man was admitted with a severe respiratory illness. After striking out on tests for the usual viruses, Zaki decided to see if he could find clues under the microscope. "If the patient had lived, I would not have worried" about identifying the virus, he said. But the patient died, so he persisted.

Zaki sent a sample to a lab in the Netherlands led by virologist Ron Fouchier (in the news recently for his work on H5N1 influenza, *SN: 6/2/12, p. 20*). Scientists there determined that it was a coronavirus in the same family as SARS. Meanwhile, Zaki had come to the same conclusion. "When I saw it was a coronavirus, I said, 'this is something dangerous,'" Zaki said.

In September, Zaki posted a short description of the virus and the patient's symptoms on an online public health forum called ProMed-mail, and later described the patient and the details of the virus in the Nov. 8 *New England Journal of Medicine*.

Zaki's forum post proved interesting reading for Alison Bermingham of the United Kingdom's Health Protection Agency. Bermingham and her colleagues had been trying to identify an unknown virus that caused pneumonia and kidney failure in a 49-year-old Qatari man who was being treated in a London hospital. Tests confirmed that the man was infected with a coronavirus nearly identical to the one Zaki had described.

Scientists discovered later that the coronavirus had killed two people in Jordan in April. Researchers should test anyone with symptoms for signs of the virus, regardless of whether they have been to places where the virus is known, Bermingham said. —*Tina Hesman Saey*

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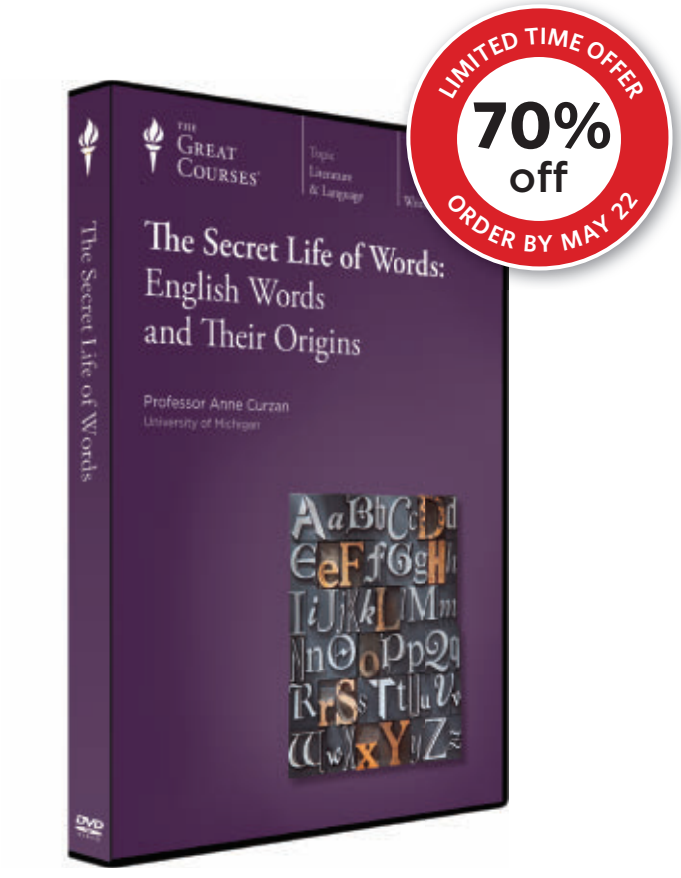

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Life

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Chilly temps turn monarchs north

Butterflies' migratory path flipped by exposure to cold

By Meghan Rosen

A string of cold weather may be all that's needed to flip monarchs' migratory compasses. Just 24 days in a chilly lab incubator is enough to switch a butterfly's flight orientation from south to north, researchers report online February 21 in *Current Biology*.

"It's pretty doggone cool," says insect ecologist Orley "Chip" Taylor of the University of Kansas, who was not involved in the new research. But the finding is also disturbing, he says. "It suggests that as temperatures warm, monarchs may be in trouble."

Each fall, millions of butterflies set off on a journey from their northern range to central Mexico to escape freezing winters. Migrating to and overwintering in Mexico is tough work: The journey strips the black-and-orange monarchs of their vibrant colors and tatters their wings. In the spring, the butterflies are strong enough to start the journey north, find food and reproduce. Then their descendants finish the long trip home.

Monarchs stop for nectar during their migration south. Cold temperatures at their Mexico destination signal the butterflies to head back north.

Until now, researchers didn't know what triggered the return trek north. They suspected environmental factors such as temperature or changing day length could cue the monarchs to the change in season. To find out, Steven Reppert of the University of Massachusetts Medical School in Worcester and his colleagues studied southward-migrating monarchs captured in the eastern United States. The scientists housed one group of migrants in an incubator for 24 days and turned down the temperature to 4° Celsius during dark "night" periods and 11° C during light "day" periods — the average temperatures in wintertime monarch butterfly roosts. The team exposed a second group of butterflies to the same temperatures while also simulating the subtle increase in daylight that monarchs see over the winter while in Mexico.

Then the researchers took the two groups outside and tethered them one by one inside a white plastic barrel that gauges flight bearings. In both experimental groups, the lab-wintered butterflies flew north. In fact, Reppert says, "the data were identical." Southern-migrating monarchs that had been captured in Texas and kept in the lab under fall conditions — with no pulse of nightly cold temperature — continued to head south when hooked up inside the barrel.

"It's astounding," says ecologist Karen Oberhauser of the University of Minnesota in St. Paul. She's convinced that just 24 days of cold temperature is enough to switch butterflies' flight direction, but she's also curious about the effects of day length alone.

Reppert says the direction-change trigger might be modified by shifts in day length, but "clearly coldness is the main factor."

Diversity breeds resistance

In the frog pond, more species can mean better health for all. More diverse amphibian communities are less likely to transmit a virulent parasite that causes limb deformities (such as the ones shown) in frogs. The report appears in the Feb. 14 *Nature*.

Pieter Johnson of the University of Colorado Boulder and colleagues studied field data from hundreds of California ponds and found that communities with high species diversity had only about 20 percent as much transmission of the trematode parasite *Ribeiroia ondatrae*, and suffered roughly half as much disease as communities with low diversity. And in lab and experimental pond studies, transmission and disease rates decreased after researchers added more species.

Many wildlife communities with greater biodiversity are better at controlling infectious disease. In these cases, preserving biodiversity, the researchers conclude, could help manage the spread of disease. — *Puneet Kollipara*

FROM TOP: COURTESY OF MONARCH WATCH; D. HERASIMTSCHUK, FRESHWATERS ILLUSTRATED

"New tissue emerges like lead in a mechanical pencil." —AYAMI SEKIZAWA

Sea slug carries disposable penis

Hermaphrodites shed organ after use, then uncoil another

By Susan Milius

A bristly hermaphroditic sea slug mates employing a use-it-then-lose-it penis, and carries one or two extras for future use, researchers have discovered.

Some 20 minutes after copulating, the still-stretched-out penis of a *Chromodoris reticulata* sea slug "just falls off," says evolutionary biologist Ayami Sekizawa of Osaka City University in Japan. The sea slug then cannot mate for a matter of hours. But when researchers waited 24 hours to offer the slug a second partner, a backup organ segment appeared in place of the discard, she and her colleagues report February 13 in *Biology Letters*.

"New tissue emerges like lead in a mechanical pencil," Sekizawa says.

The source turned out to be lengths of penis tissue coiled in reserve below the operative segment, Sekizawa found in studying tissue samples under a microscope. Given a little time between matings, the sea slug can essentially advance the tissue to extend three usable sections before having to replenish the reserves by regrowing the whole structure.

***Chromodoris reticulata* disposes of its penis after mating and then uses backup tissue for its next encounter.**

Sekizawa and her colleagues got the first hint of the *Chromodoris* detachable penis when they noticed a sea slug with a truncated organ during an earlier experiment. Watching 31 matings, the researchers determined that each slug extends its penis into the reproductive tract of the other, with mutual sperm delivery lasting nine minutes on average. As the slugs disengage, they each crawl around, penis extended. Finally the penis, with no preliminary shrinking or pinching inward, simply detaches.

Several evolutionary paths could lead to detachable penises, says evolutionary ecologist Nico Michiels of the University of Tübingen in Germany. In many species, a male plugs the female reproductive tract and stymies a rival, Michiels says. An abandoned penis could certainly serve as a plug.

Also, a left-behind penis could autonomously inject sperm after the donor flees the dangers or constraints of more intimate mating. "Not as wild as it sounds," Michiels says. "Male bees have their whole male copulatory system ripped out during copulation, and it continues to pump sperm into the queen even after the male has gone to die."

Bees tune in to flowers' buzz

Floral electric fields could offer cues to pollinators

By Susan Milius

Slight electric fields that form around flowers may lure pollinators much as colors and fragrances do.

In lab setups, bumblebees learned to distinguish fake flowers by their electrical fields, says sensory biologist Daniel Robert at the University of Bristol in England. Combining an electrical charge with a color helped the bees learn faster, Robert and his colleagues report online February 21 in *Science*.

A bit like lightning rods, plants tend to conduct electrical charges to the ground, Robert says. And bees pick up a positive charge from the atmosphere's invisible rain of charged particles.

Robert and his colleagues checked whether bees could choose flowers based solely on the electric fields the plants produce. Metal disks (topped by purple plastic so as not to shock bees) stood in for flowers. Half of them, wired for 30 volts, held sips of sugar water. The unwired ones offered a bitter quinine solution that bees don't like.

Bombus terrestris bumblebees learned to choose sweet, wired disks more than 80 percent of the time. When researchers unplugged the wired disks, the bees bumbled, scoring sugar only by chance.

"The big question is how bees do this," says Lars Chittka of Queen Mary University of London. Bees bristle with hairs, and he speculates that a charged insect nearing an oppositely charged flower feels the hairs bend.

Electric charges of bees and flowers do interact, Robert confirmed after studying bees visiting real petunias. When a bee landed, and sometimes even before, flower stems registered an electrical surge that didn't fade until after the bee had buzzed onward.

This surge in electric potential might tip off another passing bee that the flower had just lost nectar to a different visitor, a change that scent or color could not reveal, Robert says.

Variations within a flower's electric field may even hold clues to where bees should probe for nectar, Robert speculates. Researchers found striking electrical patterns when they wafted positively charged colored aerosol particles over blooms.

"I am blown away," says Anne Leonard of the University of Nevada, Reno. "I imagine that we'll all be desperate to spray our flowers down with the aerosol they describe."

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Genes & Cells

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It matters whether you're a man or a mouse

Relevance of rodent models for inflammation questioned

By Tina Hesman Saey

Mice are poor stand-ins for people in experiments on some types of inflammation, a new study concludes. But some scientists say that critique unfairly discounts the value of mouse studies, and that many biomedical experiments couldn't be done without the animals.

More attention and money should go toward studying disease in people than to mouse research, a consortium of scientists contends online February 11 in the *Proceedings of the National Academy of Sciences*. Too often, researchers make a discovery in mice and assume that humans will react in the same way, says study coauthor Ronald Tompkins, chief of the Massachusetts General Hospital burns service.

"The presumption is not justifiable," he says. As a result, drug trials — often based heavily on data gleaned from mouse studies — can fail.

But other scientists say that critique isn't new and is overstated. Clinical trials are unsuccessful for many reasons, says Derry Roopenian, an immunologist and mouse geneticist at the Jackson Laboratory in Bar Harbor, Maine. "There's frailty all along the process. That's not a failure of the mouse."

Roopenian and others worry that the study, conducted with a generic strain of laboratory mouse called Black6, unfairly tarnishes the reputation of all mice, even ones engineered to be as much like humans as possible (see Page 22). The group's conclusions, were they accepted by policy makers, could set back biomedical research by jeopardizing funding for mouse studies, critics warn.

"Without the mouse, progress is going to be slowed to a standstill," he says.

Most researchers agree that creating mice that have biological responses that more closely mirror humans is important to understand diseases and develop new drugs. The sticking point appears to be how to balance mouse-based research with research involving humans.

In the new study, a group of Canadian and American researchers compared how human and mouse genes respond to certain types of trauma. They looked at gene activity in the blood of hundreds of people severely injured either by burns or blunt trauma, such as in a car accident. They compared those results with gene activity in the blood of mice with similar injuries. The team also examined the reactions of four healthy people and 16 mice to an injection of endotoxin, a bacterial toxin that mimics blood infections.

People's immune systems reacted in predictable ways, cranking up activity in genes that cause inflammation and quieting other immune system genes, Tompkins says. The activity of most genes examined — 97 percent — reacted the same way to burns and blunt trauma. Reactions to infection and injuries were also similar, with 88 percent of genes involved in one process also working in the other.

But mouse genetic responses varied more and also differed from people's, says study coauthor Wenzhong Xiao, a genome scientist at Harvard University and Stanford University. Only 47 to 63 percent of mouse genes changed activity in the same way as human genes. The result was not far from what researchers would expect from chance.

The results indicate that humans and mice react differently to traumas that often land people in intensive care units. "We need to take those differences into serious consideration, which people currently do not," Xiao says.

No one expects mouse and human physiology to match exactly, Roopenian counters. And the differences between the species might even inform research. For instance, the study authors cite as a shortcoming that mice can withstand doses of endotoxin 1 million times the dose that sends people into shock. But Roopenian sees that difference as an opportunity to learn how to boost human tolerance to the substance.

Researchers should do more of the kinds of genetic analyses performed in this study when choosing an appropriate mouse strain, says Klaus Schughart, a geneticist at the Helmholtz Center for Infection Research in Braunschweig, Germany. Schughart studies infectious diseases, including influenza, and uses mice as human substitutes. He sees benefits in the fact that mice don't perfectly mimic complex human diseases.

"We purposely oversimplify," he says, to remove confounding factors and get at the mechanisms of disease. "We should value the mouse for what it's worth and not condemn it for its shortcomings."

Tompkins agrees that basic research should continue, but adds that "we need to recognize that its relevance to human disease is a leap of faith."

Mice from a strain called Black6 (one shown) have different molecular responses to trauma than humans do, calling into question the usefulness of mice in studies of inflammation.

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Mind & Brain

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Nothing to fear but suffocation

Loophole found in fearlessness of people with rare disorder

By Laura Sanders

Not all fear is the same. A woman who laughs at horror movies and grabs dangerous snakes nonetheless surrenders to terror at a single puff of suffocating carbon dioxide.

This woman, known as SM, has a disease that damaged her amygdala, a brain structure implicated in fear. The new results involving her and two others with the same disease, reported February 3 in *Nature Neuroscience,* show that a danger signal can hit the panic button in other parts of the brain.

Clinical neuropsychologist Justin Feinstein of the University of Iowa in Iowa City believes that the instinct to breathe might tap into a brain system that's more primal than the amygdala.

Feinstein and colleagues work with SM and other patients who suffer from a rare genetic disorder called Urbach-Wiethe disease. In late childhood, this disease destroys the amygdala, a pair of almond-shaped structures deep in the brain. Now, the scientists have found something that does scare her.

A breath that is 35 percent carbon dioxide can immediately provoke a strong, panicky fear. (Normal air is less than 1 percent carbon dioxide.) In the body, specialized proteins sense that something is amiss and send an urgent "air, now" message.

A breath of gas that is 35 percent carbon dioxide can immediately provoke a strong, panicky fear.

"It's automatic," says Feinstein, who has subjected himself to the procedure multiple times. "Your body's alarms are firing like crazy."

A recent study using mice showed that the amygdala detects carbon dioxide and helps produce fearlike behavior. So the researchers thought that SM and the two other women in the study might likewise show a blunted response to the gas.

But SM's reaction to the gas was swift and unequivocal — she was afraid. She immediately started gasping and waving her hand frantically. Her eyes flew open wide, her nostrils flared and her face flushed.

"It was a complete surprise," Feinstein says.

The other two women also panicked, though only three of 12 healthy volunteers did.

Feinstein thinks that the amygdala's usual role in panic might be to shut down a response that starts somewhere else.

"This study adds to a growing body of work showing that there are different systems for responses to different kinds of threats," says neuroscientist Joseph LeDoux of New York University.

Double duty for immune cells

In rats, a developmental role for microglia in the brain

By Laura Sanders

Immune cells that help heal injuries in the adult brain may have another job early in life, a study in rats reveals. The brain crusaders unexpectedly moonlight as sculptors, shaping a region of the brain into a male-specific form.

The cells, called microglia, are mobile garbage disposals that travel around the brain and gobble up damaged cells and infectious agents. But the new study, published in the Feb. 13 *Journal of Neuroscience*, emphasizes that these cells have diverse functions, says neuroscientist Jean Harry of the National Institute of Environmental Health Sciences in Research Triangle Park, N.C., who was not involved in the work.

Earlier results hinted that parts of the immune system have a role in building sex differences into the brain, so Kathryn Lenz and colleagues at the University of Maryland, Baltimore decided to test whether microglia pull double duty.

The team focused on the preoptic area of the rat brain — "a place where you see a ton of sex differences," Lenz says. Early in life, this brain area gets shaped by chemical messengers including estradiol and prostaglandin E2, which work on the male rat brain. In males, the preoptic area is larger, and the cells there have more elaborate shapes than in females. Scientists think those brain differences drive mating behaviors.

Lenz found another difference in the preoptic area between males and females: Young males had about twice as many active microglia as females did. What's more, a dose of estradiol or prostaglandin E2 just after birth caused female animals to produce the male number of active microglia.

This difference in number of microglia is important for behavior, the team found. After they matured, the females with the male number of microglia behaved more like male rats, mounting other females, for instance. When researchers gave these rats a drug to deactivate these microglia, the male behavior lessened.

Microglia might act as amplifiers of hormones in the male brain, Lenz says. These cells can sense and produce prostaglandin E2 — processes that could enable the male brain to develop.

Harry cautions that it's too soon to conclude that microglia cause these changes in behavior. "It's the chicken and the egg with these cells," she says.

Surprise makes recall wobbly

Painful memories weakened in therapy with drug

By Laura Sanders

The unexpected may be the key to whitewashing a painful memory. People who encountered something unanticipated were better able to shake a troubling association, a laboratory study finds. The results, in the Feb. 15 *Science*, bring scientists closer to weakening traumatic memories with help from a drug.

Understanding how the brain forms and reforms traumatic memories might lead to treatments to help people who suffer from post-traumatic stress disorder and other anxiety disorders. "The idea that an original memory could have the sting taken out of it — that's been very appealing," says psychiatrist Roger Pitman of Harvard Medical School and Massachusetts General Hospital, who was not involved in the research.

Recent results in animals and humans have shown that once called to mind, painful memories' emotional edges can be blunted. Experiments have used drugs to weaken associations between a memory and a negative response.

The new result may have uncovered an underappreciated step in that drug-triggered weakening process: Something unexpected must happen while the person is recalling the memory. This mismatch between what a person expects and what happens puts a memory into a wobbly, vulnerable form that can be washed out, says study coauthor Merel Kindt of the University of Amsterdam.

In the experiment, people learned that a picture of a gun or a spider came along with an electric shock. This taught the people to fear one of the pictures — the gun or the spider — more than the other.

The next day, some people reexperienced the same connection (a spider picture always came with a shock, for instance) while other people dealt with curve balls (a spider picture sometimes didn't come with a shock). Electrodes underneath the eye picked up people's flinches, a gauge of fear responses.

After the second day's training, every participant received propranolol, a drug that interferes with memory formation. The drug didn't affect the people who relived the original experience. But those who experienced something new were less likely to flinch in anticipation of a shock that, from the first day of training, they should have expected was coming. For these people, "it is not just a reduction of the fear response," Kindt says. "The fear response is erased."

Environment

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Antianxiety drugs affect fish

Perch eat faster and swim more with low-level exposure

By Erin Wayman

Fish let their inhibitions go when exposed to a common antianxiety medication, a new study shows. Perch living in water spiked with the drug became bolder, less social and more active than unexposed fish. They also ate much faster.

These behavioral changes could make fish living in wastewater-polluted rivers more vulnerable to predators, researchers argue in the Feb. 15 *Science*. And fishes' speedy eating could disrupt the food chain.

Fish and other aquatic animals ingest a variety of pharmaceuticals that end up in the environment. People excrete drugs and flush unused pills down the toilet. Since wastewater treatment plants don't filter these compounds out of sewage, the drugs end up in rivers.

Previous studies have shown that antidepressants slow a fish's reaction time and decrease shelter-seeking behavior.

The new study looked at oxazepam, a type of benzodiazepine. Benzodiazepines are among the most frequently used antianxiety medications. A team led by Tomas Brodin of Sweden's Umeå University measured concentrations of oxazepam in a Swedish river, five meters downstream of a wastewater treatment plant. The level of oxazepam was 0.58 micrograms per liter. European perch (*Perca fluviatilis*) that the researchers collected from the river had an average of six times that concentration stored in their muscles.

In the lab, the researchers placed young perch in tanks for seven days with either no oxazepam or with the drug at one of two concentrations: 1.8 micrograms per liter or 910 micrograms per liter. Fish exposed to the lower drug level accumulated amounts of oxazepam in their muscles similar to the wild perch's supply.

Perch exposed to both levels of oxazepam swam more, spent more time alone and gobbled up a meal of zooplankton faster than unexposed fish did. At the higher concentration, fish became more adventurous, entering a new tank more quickly than unexposed fish.

These behaviors directly relate to survival and ecology, says aquatic toxicologist Heiko Schoenfuss of St. Cloud State University in Minnesota. Brazen, solitary fish are probably easy for predators to catch. And fish foraging at high rates may take food away from other animals, altering the makeup of the ecosystem. The next step, he says, is to investigate whether these behavioral changes actually affect wild populations.

Regulators assessing environmental hazards need information about how drugs influence ecologically relevant animal behavior, says environmental toxicologist Bryan Brooks of Baylor University in Waco, Texas.

"If your regulatory toolbox is not suitably developed," he says, "you run the risk of applying the wrong tool to diagnose environmental perturbations."

Exposures to antianxiety drugs led to behavioral changes in perch.

Melting ice spurs algae

By letting more sunlight through, thinning Arctic sea ice may be promoting the growth of algae, researchers report February 14 in *Science*. The alga *Melosira arctica* grows in long strands on the underside of sea ice. In summer 2012, scientists found clumps of fresh algae (shown) up to 50 centimeters across that had fallen to the Central Arctic seafloor. Antje Boetius of the Alfred Wegener Institute for Polar and Marine Research in Germany and colleagues infer that the algae must have grown within a single year because more than 95 percent of the region's sea ice was no more than a year old. That ice, averaging less than a meter thick, was much thinner than the once dominant, multiyear, meters-thick ice that has largely melted away. The thinning may speed growth, but it also speeds the algae's death: As the ice melts, algae break free and sink to the ocean bottom, where hungry invertebrates await. — *Erin Wayman*

FROM TOP: COURTESY OF BENT CHRISTENSEN; COURTESY OF MAR FERNÁNDEZ-MÉNDEZ/ALFRED WEGENER INSTITUTE

Humans

200,000 years ago | Earliest estimated existence of Neandertals

Date of Neandertal demise debated

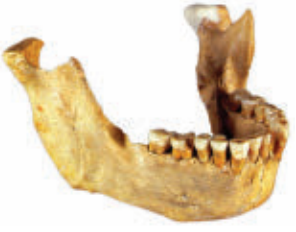
Improved radiocarbon method suggests older age for fossils

By Erin Wayman

The story of the Neandertals may need a new ending, a controversial study suggests. Using improved radiocarbon methods, scientists redated two of the youngest known Neandertal cave sites and concluded that they are at least 10,000 years older than previous studies have found.

The findings cast doubt on the reliability of radiocarbon dates from other recent Neandertal sites, the researchers suggest in the Feb. 19 *Proceedings of the National Academy of Sciences*.

This means the last Neandertals might have died out much earlier than previously thought, which could cause anthropologists to rethink how and why these hominids vanished. Researchers have long debated whether the harsh Ice Age climate, the appearance of modern humans migrating out of Africa or some other factor drove Neandertals to extinction.



A new dating of mammal remains found alongside this Neandertal jaw in a cave in southern Spain suggests the whole pile is older than expected.

"The paper is simply excellent," says archaeologist Olaf Jöris of the Romano-Germanic Central Museum in Mainz, Germany. The new research supports Jöris' own review of Neandertal dates, in which he concluded that the last of the Neandertals probably lived around 42,000 years ago. The standard view suggests that these hominids occupied Europe much longer, until about 28,000 years ago.

But other archaeologists are not convinced by the new work. "We shouldn't get too carried away over results that amount to a few radiocarbon dates from two sites," says Paul Pettitt, an archaeologist at Durham University in England.

Over the last couple of decades, archaeologists have determined that the Iberian Peninsula was one of the last Neandertal refuges. Neandertals throughout much of Europe appear to have gone extinct around the same time that modern humans reached the continent, at least 42,000 years ago. But the favorable climate of southern Spain and Gibraltar may have helped Neandertals hang on for another 10,000 years or so.

Getting a precise chronology is crucial to understanding what factors played a role in the Neandertals' demise and the degree to which Neandertals and humans interacted and possibly interbred, researchers say.

Most of the youngest Neandertal ages come from radiocarbon dating. This method dates organic material by using the steady rate at which one form of carbon transforms into another after an organism dies. But bones, charcoal and other samples can appear younger than they really are if, over time, they become contaminated with younger organic material. In the last several years, researchers have developed new ultrafiltration methods to better remove such impurities.

In the new study, radiocarbon scientist Rachel Wood of the Australian National University in Canberra and colleagues used ultrafiltration with radiocarbon dating to reassess the ages of some young Neandertal sites. The team collected 215 animal bones from 11 sites on the Iberian Peninsula. But after ultrafiltration — which strips away most of the proteins in a sample — there wasn't enough organic matter left in bones from nine of the sites to do radiocarbon testing.

Analyses of fossil finds from two caves in southern Spain (Jarama VI shown) suggest that the last Neandertals may have died out longer ago than thought.

The team did find sufficient material from two sites: At the Jarama VI site in central Spain, a rock-shelter where Neandertal tools have turned up, the researchers dated three mammal bones that contain signs of butchering. At Cueva del Boquete de Zafarraya in southern Spain, they dated three mammal bones found near Neandertal fossils. Both of the sites had been dated to between about 30,000 and 40,000 years ago. But the new analyses suggest these sites are at least 10,000 years older, the researchers say.

Given that older radiocarbon techniques underestimated the ages of these sites, other Neandertal radiocarbon dates may be inaccurate, too, Wood says. But reanalyzing other sites will be difficult because ultrafiltration requires samples to be well preserved. The Iberian Peninsula is warm and organic material in fossils quickly degrades, so many bones in the new study couldn't be dated.

Scientists have used other dating techniques in this region, but those methods are not as precise as radiocarbon dating, Wood says.

Tossing out other radiocarbon dates based on this study doesn't sit well with many archaeologists. João Zilhão of the University of Barcelona notes that Wood's team didn't actually redate the part of the cave at Jarama VI where Neandertals most recently lived. As a result, they can't say when the very last members of the group lived there.

FROM TOP: COURTESY OF J.F. JORDÁ PARDO; COURTESY OF C. BARROSO-RUÍZ

Atom & Cosmos

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Uncertainty at a grand scale

Heisenberg's principle observed in macroscopic objects

By Andrew Grant

The Heisenberg uncertainty principle, a tenet of quantum mechanics, has been demonstrated at scales visible to the naked eye. The research, described in the Feb. 15 *Science*, could help detect minuscule perturbations in the fabric of space caused by merging black holes.

"The uncertainty principle has been demonstrated in many different ways, but to see it on a visible mechanical object is totally awesome," says Keith Schwab, a physicist at Caltech who was not involved in the research.

Physicist Werner Heisenberg's famous 1927 uncertainty principle states that there is a fundamental limit to how precisely one can measure an object's position and momentum at the same time. To demonstrate his theory, Heisenberg gave the example of locating a single electron. To do so would require bouncing light off the electron. The problem, he suggested, was that even a single photon of light would give the electron a kick, changing its momentum and thus its position.

This link between position and momentum typically plays a negligible role in objects large enough to be visible to the naked eye. Nonetheless, physicist Thomas Purdy and his team at JILA in Boulder, Colo., wanted to demonstrate the uncertainty principle at a macro scale. So they set out to measure the position of a visible object made up of a million billion atoms with a laser shot consisting of 100 million photons.

Purdy's team started by creating a tiny drum about 0.5 millimeters across. To eliminate the effects of heat, the researchers cooled the drum to about 5 kelvins. The team added tiny mirrors next to each face of the drum. Then the researchers fired a laser.

As the light bounced between the mirrors, most of the photons hit the drum and transferred momentum before eventually entering a detector that calculated the drum's position. The drum vibrated on the order of picometers, or trillionths of a meter, due to little kicks from the photons.

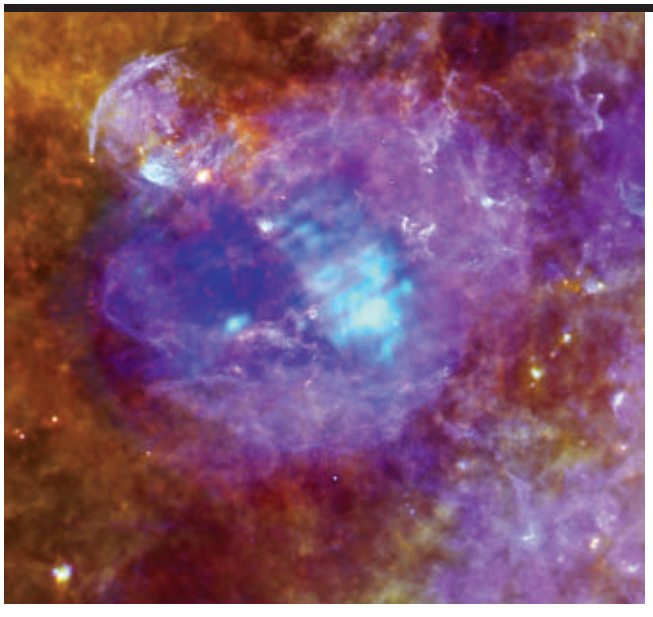
This uncertainty in measurement is only a couple of picometers' worth but is extremely important for scientists who need very precise measurements.

In a project in Louisiana and Washington called the Laser Interferometer Gravitational-Wave Observatory, or LIGO, physicists use experimental setups that are similar to but much larger than Purdy's to hunt for gravitational waves — ripples in the fabric of space caused by merging black holes and other massive astronomical phenomena.

Each LIGO apparatus consists of laser beams that bounce between two mirrors separated by four kilometers. LIGO physicists use the beams to measure the position of each mirror and thus the distance between them.

According to Einstein's theory of general relativity, a passing gravitational wave should cause the measured distance between mirrors to change slightly — around a billionth of a billionth of a meter — for the briefest of moments.

Engineers have developed such precise instrumentation that they will soon be faced with separating the distance fluctuations that stem from real gravitational waves from those caused by subtle kicks from the laser.



HOBYS/HERSCHEL, XMM-NEWTON, ESA

Cosmic ray factories

Remnants of two supernovas have provided the best proof yet that supernova shock waves churn out the high-energy protons that whizz through the galaxy.

Many of these speedy protons, known as cosmic rays, have more energy than those in any earthbound particle accelerator. Stanford University astrophysicist Stefan Funk and colleagues spent four years probing W44 (shown), the remains of a supernova located about 9,500 light-years away in the constellation Aquila, and another remnant called IC 443, roughly 5,000 light-years away in the constellation Gemini, with NASA's Fermi Gamma-ray Space Telescope. The scope picked up a steady stream of gamma radiation at energies that could have come only from a nuclear reaction involving cosmic rays slamming into slower-moving interstellar material, the researchers report in the Feb. 15 *Science*.

—*Andrew Grant*

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Virus used as cancer killer

Treatment triggers immune system to kill malignancies

By Nathan Seppa

A tweaked virus that invades malignant cells has allowed patients with advanced liver cancer to survive for months longer than usual. Scientists report online February 10 in *Nature Medicine* that the virus can sabotage tumors and expose them to attack by the immune system.

The virus-based treatment will add a new weapon to medicine's armory of tumor killers, says Ulrich Lauer, a hepatologist at the University of Tübingen in Germany who wasn't involved in the research. Patients whose cancer has grown resistant to chemotherapy or other treatments might benefit, he says, because the virus's mechanism of killing cancer cells is unusual.

Viruses trigger a prompt immune response. Researchers have been trying since the 1990s to convert viruses into treatments aimed at tumor cells. The new study employs a partially disabled cowpox virus dubbed JX-594 that does not produce full-blown symptoms of the skin disease.

Study coauthor David Kirn, a physician and researcher at Jennerex Biotherapeutics in San Francisco, says earlier tests showed that the altered virus has a potent effect on tumor cells. "It multiplies and blows the cell apart from the inside out," he says. The immune system sees the cell's debris as a Mayday signal and sends sentinels that recognize suspicious tumor proteins and present the proteins to enforcer cells. This triggers an immune attack on other tumor cells.

Kirn and his colleagues identified 30 people with advanced liver cancer that was not treatable with surgery. Survival for such patients is typically three to six months. Seven of the patients had failed to improve on other treatments and 19 had multiple tumors. Kirn says cancer had probably spread beyond the liver in all of them, even though doctors hadn't spotted some patients' tumor offshoots.

Each patient received three infusions of JX-594 into their liver tumors over a month. Sixteen got a large dose while 13 others received a smaller one. One patient left the trial for reasons unrelated to the cancer treatment.

Median survival was 14 months in patients getting the larger dose and seven months in the others. Two-thirds of high-dose patients were still alive after one year and at least four patients survived for more than two years. Those survivors included two whose tumors had failed previously to respond to medication. The predicted survival for such patients is two to four months, the authors note.

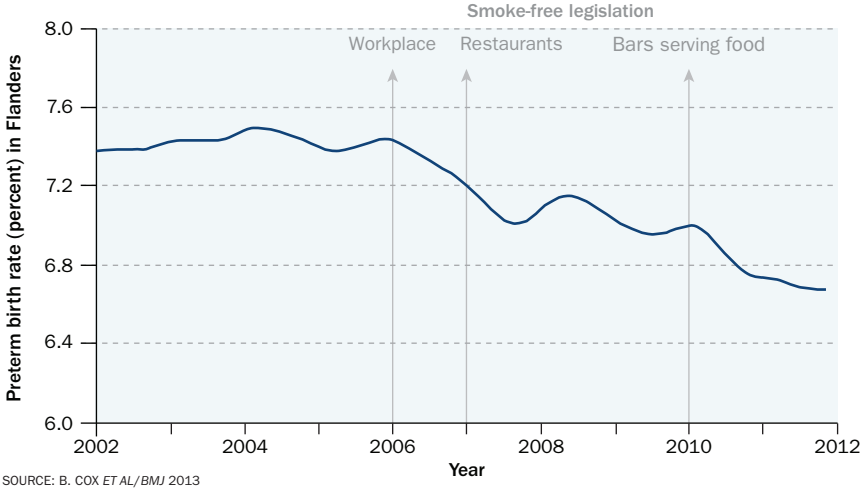
JX-594 caused only mild, flulike symptoms in most patients. It preferentially hit tumor cells because the researchers had engineered the virus to lack an enzyme called thymidine kinase, which it needs. Cancerous cells produce an abundance of the enzyme, and the virus thrives in them, says study coauthor Tony Reid, an oncologist at the University of California, San Diego.

"We're just hijacking the machinery and turning it into an Achilles' heel of the tumor," he says. ■

Smoking ban cuts preterm births

Since Belgium instituted a phased-in ban on smoking in public places, the Flanders region has seen a steady decline in preterm births, researchers report February 14 in *BMJ*. Smoking during pregnancy is known to impair fetal growth and shorten gestation, and some research suggests secondhand smoke can contribute to the same problems. But the effect of smoking bans on gestation is less clear. Belgium halted smoking in most public buildings in 2006, then extended the ban to restaurants in 2007 and to bars serving food in 2010. A research team led by Tim Nawrot at Belgium's Hasselt University pored over health records and found a 3.1 percent decline in preterm births in 2007 and a further drop of 2.7 percent in 2010. The researchers accounted for other factors that might influence preterm births, such as the age of the mother, her national origin and even local air pollution. Earlier work has linked smoking bans to decreased heart attacks in Minnesota and less asthma in Scottish children. —*Nathan Seppa*

Smoking bans linked to decrease in preterm births



SOURCE: B. COX *ET AL*/*BMJ* 2013

"We're just hijacking the machinery and turning it into an Achilles' heel of the tumor." —TONY REID

Pollution's links to asthma, allergy

Airborne PAH tied to disabled immune-regulating cells

By Nathan Seppa

Bad actors in air pollution may contribute to asthma and allergy by subverting protective cells in the body that tone down immune reactions, researchers report. The pollution components also seem to rev up overactive immune warriors — already linked to allergies — that need no such prompting.

The airborne culprits are polycyclic aromatic hydrocarbons, or PAHs, the products of incomplete burning of fuel in diesel engines, furnaces, wood fires, wildfires and even barbecue grills. Past research has tied air pollution to asthma and allergy, but the link between PAHs and these immune problems is still unclear.

In the new study, researchers found that children exposed to high levels of PAHs had poorly functioning T-regulatory cells, or T-regs, which normally ratchet down immune-caused inflammation as needed.

"T-regs are peacekeeper cells," said Kari Nadeau, a physician and biochemist at Stanford University who presented the findings February 23. "But in asthma, T-regs are impaired."

The team also found that kids exposed to a lot of PAHs made excess amounts of an antibody called immunoglobulin E, or IgE. The IgE antibody normally helps the body fight parasites. But in developed countries, where parasitic infections are largely a thing of the past, IgE has become better known for its role in allergy. The body often cranks out IgE as part of a misguided immune reaction against noninfectious substances in the environment. IgE also shows up in asthma, which can be triggered by allergy.

To study the effect of air pollution on these immune players, Nadeau and her colleagues obtained blood tests, lung function readings and health information from 153 children with a median age of 14 in Fresno, Calif. The researchers sampled airborne PAHs to estimate exposure, and chose Fresno because of its relatively high air pollution levels.

Children with high exposure to PAHs, based on air sampling in and around their homes, made high amounts of IgE and had lower T-reg function than children exposed to low levels. High PAH exposure during the most recent three months was linked to 51 percent higher risk of being diagnosed with asthma.

"This is a very interesting and thought-provoking study," said Todd Rambasek, an allergist at ENT and Allergy Health Services in Lorain, Ohio. He said that other studies that had linked air pollution with asthma and allergy had failed to distinguish between PAHs and other pollutants that could contribute to the conditions, such as ozone or particulate matter.

Nadeau also reported that consistent PAH exposure coincided with changes in a gene called *Foxp3*.

As reported by Alexander Rudensky of Memorial Sloan-Kettering Cancer Center and colleagues in *Nature* in 2010, *Foxp3* seems to be a master regulator of T-reg populations in the body. Unfortunately, Nadeau said, the changes observed in *Foxp3* seem irreversible and widespread.

The asthma rate is 22 percent among children in Fresno, Nadeau said; the CDC estimates the rate for the United States as a whole is 9.5 percent. Up to 70 percent of people in Fresno have an allergy, she notes, more than double California's average lifetime risk of having an allergy.

The new study adds PAHs to a known pollution problem in the area: In 2012, the American Lung Association ranked Fresno fifth-worst among California cities for overall particulate pollution. ■

MEETING NOTES

Promising treatment for hives

People with hives that recur for years might get relief from an allergy drug called omalizumab, marketed as Xolair. Scientists assigned 323 patients with chronic hives, or urticaria, to get three shots of the medicine spaced four weeks apart. Patients getting the study's two highest doses had substantial declines in itching that lasted until four weeks after the last shot, and 40 percent of the highest dose group saw their hives disappear completely during treatment. "Many of us in the field view [omalizumab] as a game changer," said coauthor Allen Kaplan of the Medical University of South Carolina in Charleston. The researchers reported the finding February 24 and in the *New England Journal of Medicine*. — *Nathan Seppa*

Vitamin D may boost efficacy of hepatitis B shot

Compared with men who have low levels of vitamin D, men with high levels might get more out of a hepatitis B vaccination, South Korean researchers reported. Scientists obtained blood samples from 5,025 men who had been immunized against hepatitis B. Men who had low levels of vitamin D had fewer protective antibodies against hepatitis than did those in the high vitamin D group, Ju-Suk Lee of Sungkyunkwan University in Changwon reported on February 23. The findings are in line with other evidence that vitamin D helps regulate immune function. Men who smoked, were older, had diabetes or were heavier also had a muted response to the vaccine. — *Nathan Seppa*

News in Brief

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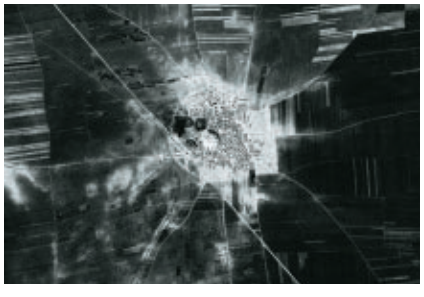
MIND & BRAIN

Smoking damages mouse brains

Cigarette smoke damages the lungs, but it also wreaks havoc in the brain, a study in mice suggests. Signs of Alzheimer's disease increased in the brains of animals that breathed cigarette smoke for four months, scientists report February 19 in *Nature Communications*. Scientists led by Claudio Soto of the University of Texas Medical School at Houston exposed mice bred to develop Alzheimer's symptoms to cigarette smoke (one cigarette's worth in air the mouse breathed for an hour, five days a week). The animals had more amyloid-beta plaques, a higher load of abnormal tau protein and more severe inflammation in their brains. The scientists don't know yet how cigarette smoke causes these changes, or whether a similar process happens in people. —*Laura Sanders*

HUMANS



Radial routes outside Mesopotamia

Spoke-like dirt paths extend up to five kilometers from several ancient Mesopotamian cities that have been excavated in northeastern Syria and northern Iraq. Although often considered unique to these more than 5,000-year-old sites, new evidence reveals similar radial paths in western Syria (shown) and southwestern Iran that date to as recently as 1,200 years ago. Archaeologist Jesse Casana of the University of Arkansas in Fayetteville made these discoveries by analyzing declassified, Cold War–era satellite images that show Middle Eastern landscapes before intensive farming erased all traces of ancient dirt roads. Some researchers think that Mesopotamia's radial routes were created more than 4,000 years ago by herders leading sheep and goats to and from grazing lands through narrow strips that separated cultivated fields next to city centers. That same pathbreaking process occurred over the next four millennia at cities across the Middle East, Casana proposes February 13 in the *Journal of Anthropological Archaeology*. —*Bruce Bower*

Sleep loss affects gene activity

People who don't get enough sleep tend to experience a wide variety of health problems, but scientists haven't known why at a molecular level. Now, Derk-Jan Dijk and colleagues at the University of Surrey in England report changes in the gene activity of 26 people who had built up a sleep deficit. For one week the volunteers slept at least eight hours per night. Then, participants were allowed just under six hours of sleep each night for another week. People were sleepy and sluggish after that week, and blood tests showed that the activity of 711 of their genes had changed, the researchers report February 25 in the *Proceedings of the National Academy of Sciences*. Among genes affected by the shift were those that govern the immune system. Some of those genes stopped cycling in a daily, or circadian, pattern. The activity of other genes that don't usually follow the clock fell into a such a pattern. The researchers conclude that skimping on sleep can drastically change the body's daily rhythms, which may lead to the observed health problems. —*Tina Hesman Saey*

MOLECULES

Lipstick identity revealed

WASHINGTON—Discerning one lipstick from the next can be tough for consumers but it's been even harder for scientists. Now forensics researchers have found a quick method to tell apart individual lipsticks, no matter the color or brand. The approach could help investigators analyze evidence in cases that hinge on a smear of lipstick on glass, paper or clothing. Experts typically examine a lipstick's dyes or its chemical or elemental composition. To distinguish closely related cosmetics, those tests usually need to be combined, said Paige Gardner of Virginia Commonwealth University in Richmond. She decided to tackle 80 lipsticks with Raman spectroscopy, which zaps a sample with laser light, making some of the molecules vibrate. These tickled molecules emit light of a frequency different from what came in, and researchers can read that shift in frequency like a chemical bar code. The approach allowed Gardner to distinguish 95 percent of the lipsticks from one another, she reported February 22 at the American Academy of Forensic Sciences annual meeting. Whether crimson, paprika, plum or even two closely related reds, the Raman bar codes were unique. Tests of lipstick smears on some fabrics weren't as conclusive: On silk, for example, the lipstick signature was discernible, but on red car upholstery the signal was lost. —*Rachel Ehrenberg*

GENES & CELLS

Bitter and sour taste detectors also say 'too salty'

Salt has a split personality, with its ability to enhance deliciousness or ruin a dish. Now scientists have revealed the underpinnings of salt's dark side: Heavy doses trigger taste cells that detect bitter and sour flavors. Mice without working versions of these taste cells find high levels of saltiness appetizing rather than repugnant, scientists report in the Feb. 28 *Nature*. The researchers don't know how high salt levels kickstart the bitter and sour detectors, but the research suggests that a triumvirate of taste cells must oversee salt detection. Dedicated salt detectors enable attraction to salt, while bitter and sour detectors take over when salt levels skyrocket. —*Rachel Ehrenberg*

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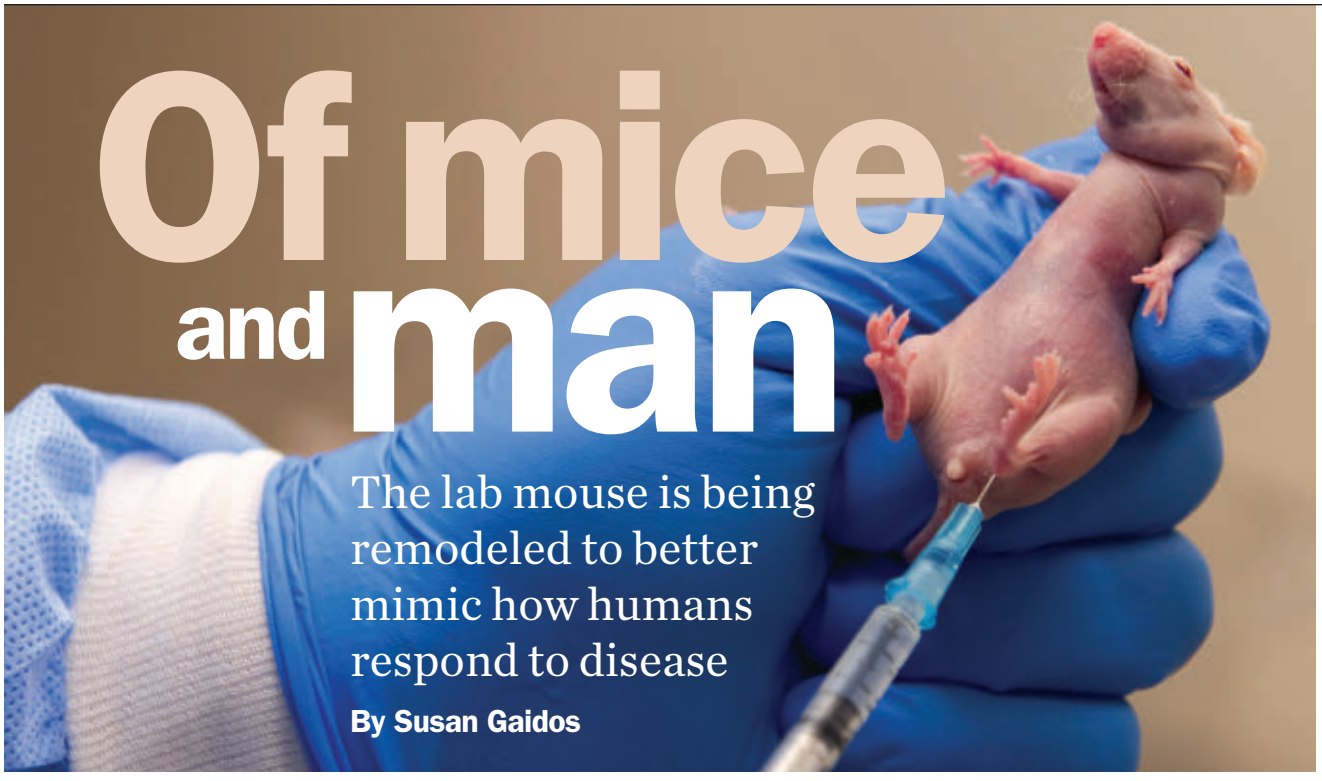
Of mice and man

The lab mouse is being remodeled to better mimic how humans respond to disease

By Susan Gaidos



A humanized mouse implanted with a human tumor is one of the many being used to help doctors identify the best treatments for a patient's cancer.

MARY CALVERT/THE NEW YORK TIMES/REDUX PICTURES

It looked like a lost cause: a 61-year-old patient with advanced pancreatic cancer — one of the deadliest diagnoses. Ordinarily, doctors wouldn't have much choice. They could try chemotherapy drugs one at a time to see if any worked, or they could slam the tumor with a cocktail of chemicals that had shown some success with similar cases.

But this time the doctors had another option: trying out potential treatments on mice. These weren't run-of-the-mill lab rodents, though. Scientists had created 17 mice with an immune system matched to the patient's. After growing "copies" of the patient's cancer in the mice, doctors tried a slew of different treatments on the tumors and discovered that the cancer was extremely sensitive to one particular drug.

Scientists had implanted human tumors in mice before. But there was no guarantee that mice would respond to drugs the way a person would. In the new approach, young mice engineered to lack immune systems receive transplants of portions of a patient's immune system. As the mice mature, they develop a humanlike immune response to drugs or infectious agents.

"The idea is that you can take human immune systems and put them in a mouse and make them functional. Then you can manipulate them as if you were manipulating little humans, so to speak, without ever putting patients at risk," says Dale Greiner of the University of Massachusetts Medical School.

Such "humanized" mice may not be able to dance, drive cars or play chess like a human. But they might soon help doctors predict how people will respond to a particular treatment or warn of deadly side effects in new medicines before they go on the market.

The day your local doctor can grow a colony of mice with your immune system and particular ailment is still years away, but the scenario gets more plausible every week. Already, for dozens of cancer patients, the mice have helped identify potential treatments before therapy began. And at the University of California, Davis, scientists are using such mice to test a new kind of bladder cancer treatment. That treatment is now moving toward human clinical trials.

Cancer is only one area where humanized mice may be useful. Studies now underway are using the mice as stand-ins to see how the immune system responds to infectious agents such as dengue virus or HIV. Other research uses humanized mice to learn how complex diseases such as rheumatoid arthritis and diabetes operate. In his own lab, Greiner studies the immune system in hyperglycemic mice to shed light on the development of type 1 diabetes, an autoimmune disease.

Man or mouse?

Medical researchers have always relied on animal tests to see how cells and organs react to infections or drugs. Such tests would be hazardous and unethical to perform on people.

But animal anatomies are not the same as human bodies. There is no animal that gets acquired immune deficiency syndrome in the same way humans do, for example. For years, the rhesus monkey has served as the animal alternative for HIV research, but in most cases the monkey can be infected only

with a related virus. Differences in the viruses, along with differences between the human and monkey immune systems, limit studies designed to understand HIV or to test drugs and vaccines.

Humanized mice, however, respond to HIV more like a man than a mouse, researchers reported last July in *Science Translational Medicine*. A group led by Todd Allen of Harvard Medical School showed how the virus mutates and attempts to evade these responses just as it does in humans.

"For the first time we have an animal model that accurately reproduces these critical host-pathogen interactions," Allen says. His lab at the Ragon Institute (a joint venture of Harvard, MIT and the Massachusetts General Hospital) is now using the mice to see how human immune responses might succeed or fail to control HIV. His ultimate goal is to find an effective HIV vaccine.

To get a mouse to mount a human-like immune response is no easy feat. Normally, mice are immune to many of the infectious agents that make humans sick. That's because many aspects of a mammal's immune system are designed to fight off specific types of invaders that vary from species to species. One form of a flu virus, for example, plagues dogs, while other types affect birds, horses and humans.

Despite these differences, all mammals have immune systems that are divided into two categories: the innate system and the adaptive system. Innate immunity is present in all animals, providing immediate but short-lived relief. As the first line of defense against bacteria and viruses, innate immune cells take a broad-based, slug-it-out approach to immunity, mounting a defense against any agent that appears "foreign."

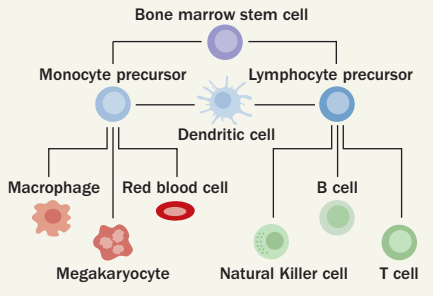
"It's like putting up a flypaper stick in the window and anything that flies by gets stuck," says Greiner.

Adaptive immunity, by contrast, is exquisitely specific and much more complex. It produces long-lasting protection against specific pathogens after an initial exposure. Vaccines, for example, work because the body generates antibody responses and cellular responses that are capable of targeting a specific molecule.

One way to get around these defenses is to wipe out the mouse's own adaptive immune system. Another more common practice is to work with mice that have no such system. Years ago, scientists discovered a strain of mice with a genetic defect that prevents their immune system from developing, leaving them defenseless against all environmental microbes and infectious agents. Called severe combined immunodeficient mice, or SCID mice, they have the equivalent of what is known as "bubble boy" disease. Because their bodies' defenses are almost totally absent, the mice do not reject foreign tissue.

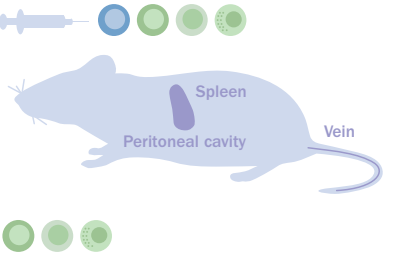
In 1988, two research groups successfully used SCID mice to create a small, working model of a human immune system. One group transplanted tissue

Making mice more human Using mouse strains engineered to lack normal immune systems, researchers have succeeded in transplanting part or all of the human immune system into mice, including many of the disease-fighting cells derived from stem cells (right). A number of different approaches (below) are used to transform the mice, each with its own benefits. Key to the efforts' success has been the creation of mice with human genes that foster the growth and development of the human immune components added to the mice.



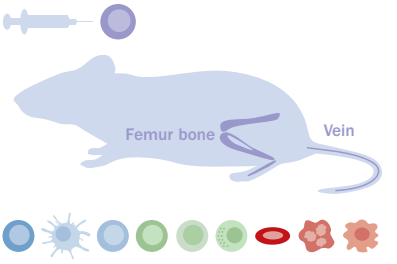
Transforming mice with human immune players

Mature human lymphoid cells are injected into spleen, vein and peritoneal cavity.



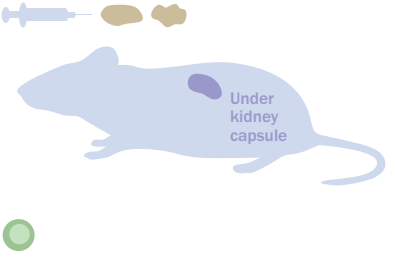
Mature T cells and low levels of B cells and other immune cells result, providing a good model for studying infectious agents and graft-versus-host disease, common in transplant medicine.

Human bone marrow stem cells are injected into femur bone and vein.



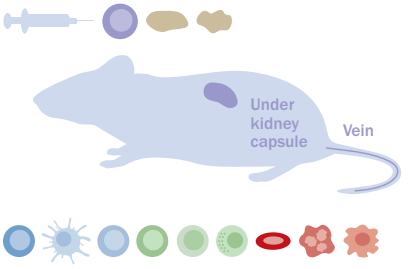
A naive human immune system results; T cells recognize mouse cells as self, not human cells. This model is useful for studying the naive immune system's responses to various viruses.

Fragments of human fetal liver and thymus are injected under kidney capsule.



A weak overall immune system results. T cells recognize human cells, but few other human immune cells are produced. The model was used to study HIV infection in T cells.

Human fetal liver and thymus fragments are injected under kidney capsule; stem cells in vein.



A near-complete human immune system, with human-educated T cells and a stable supply of other cells, provides a model for studies of HIV, dengue and other diseases.

SOURCE: L. SHULTZ *ET AL*/*NATURE REVIEWS IMMUNOLOGY* 2012

E. FELICIANO

Model mice Humanized mice are helping to galvanize studies of cancer, infectious disease and autoimmune disorders.

Spread of melanoma

Sean Morrison of the University of Texas implanted tumors from 25 patients with melanoma into humanized mice. The cancer's metastatic spread in the mice correlated with the spread of disease in the patients, showing the mice's utility in studying cancer metastasis.

Breast milk and HIV

Moms with HIV are told not to breast feed their babies. But J. Victor Garcia-Martinez of the University of North Carolina has found that humanized mice fed human breast milk contaminated with HIV were unlikely to become infected.

Viral studies

Carried by mosquitoes, the dengue virus sickens millions of people worldwide each year. It can be fatal and has no treatment. Recently, a Texas Biomedical Research Institute team transmitted the virus to humanized mice, providing the first animal model of the disease.

Arthritic mice

Researchers at Northwestern University recently used stem cells to create a humanized mouse that duplicates the human response in rheumatoid arthritis, an autoimmune disease.

taken from the liver, thymus and lymph glands of fetuses. The other team injected human white blood cells into the mice. Both types of transplants produced mice with human immune system cells and antibodies.

In the early 1990s, scientists developed a way to get human blood stem cells into the mice. Such cells, isolated from blood or bone marrow, can renew themselves and differentiate into various cell types. In 1995, Leonard Shultz and his group at the Jackson Laboratory in Bar Harbor, Maine, created an improved strain called the NOD/SCID mouse, which allowed more types of human cells to grow when engrafted in mice.

NOD/SCID mice offered another advantage: They could generate most of the disease-fighting components of the human immune system, with the important exception of T cells. T cells attack invaders directly, looking for them by recognizing distinctively shaped structures on their coats. In 2004, scientists at the Institute for Research in Biomedicine in Switzerland found a way to get T cells to develop from stem cells in mice. The following year, Shultz and his team found that disabling a gene called interleukin-2 receptor gamma also enabled T cell development. With robust T cells, the mice could help scientists study how the human body fights disease.

Since then, humanized mice have been tweaked to become an even friendlier host to human cells. Today, three main "brands" of mouse strains are used in laboratories around the world. The NOG mouse, developed by Mamoru Ito of the Central Institute for Experimental Animals in Japan; the BRG mouse, developed in Switzerland; and the NSG mouse, developed and distributed by the Jackson Laboratory. Some support high levels of T cells, while others allow engraftment of bone marrow or other particular tissue types. Scientists select a strain based on their needs.

Over the last few years the mice have been adapted as tools to study human biological processes in a wide range of fields. In regenerative medicine, for example, scientists looking for ways to replace damaged tissues such as muscle, liver or nerve cells are placing human stem cells in the mice to see how the cells function. Researchers figure if the stem cells can repair some kind of damage in the humanized mice, chances are they will work in a person as well.

Other studies focus on infectious disease, using humanized mice as guinea pigs to forecast how humans will respond when infected with agents such as dengue virus, typhoid fever or HIV.

Personalized treatments

Physicians, too, are latching onto the humanized mice, using them as "avatars" for real people. These avatars can help doctors find the most effective cocktail of cancer drugs to combat a particular tumor before treating a patient.

Traditionally, doctors follow a course of treatment based on tumor type. That's likely to change as more studies show how humanized mice can help tailor treatment to the individual, says Manuel Hidalgo of the Spanish National Cancer Research Center in Madrid.

Hidalgo and his collaborators identified life-extending treatments for 11 out of 14 cancer patients after developing colonies of mice customized for each person. (One patient died before treatment began, and in two cases, the tumors simply did not grow in the mice). In each case, the drug activity in the mice matched the outcome for the patients, the team reported in *Molecular Cancer Therapeutics* in 2011.

"Every time we saw a response in the mouse, we later saw it in the patient. If there was no response, we didn't see it in the patient," says study coauthor David Sidransky, an oncologist at the Johns Hopkins School of Medicine.

Since then, Hidalgo, Sidransky and their colleagues have used avatar mice in a larger study to analyze response rates in more than 60 patients. The mice successfully identified treatment options for 94 percent of the patients. In some of those cases, the mice predicted that there was no treatment available, and indeed, attempts to treat those patients failed.

"The beauty of the grafts is that they allow you to test any kind of drug in any kind of combination," Sidransky says. "You can line up 100 mice with that tumor and try every concoction you want to throw at it to see if one will work.... Standard tests just allow you to look at one drug at a time, and give you a very limited scenario for what you can do."

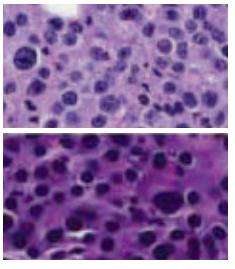
In their initial study, Sidransky and Hidalgo found treatments that worked in the mouse tumor that would have otherwise been overlooked because they were not considered "standard care" for those tumor types. One such serendipitous finding helped extend the life of the 61-year-old pancreatic cancer patient. After engrafting pieces of the patient's

COURTESY OF THE JACKSON LAB

tumor onto 17 mice and trying a score of different treatments, the scientists discovered that the tumor was extremely sensitive to a drug called mitomycin C. When treated with this drug, the patient's tumor went into remission and he went on to live for nearly seven years.

Later, Hidalgo and his group performed a genetic analysis on that patient's tumor and found that it contained a mutation that made the cancer susceptible to that particular drug. Those findings were published in a separate 2011 paper in *Molecular Cancer Therapeutics*. The group is now using an approach that combines genomic analysis with the avatar models. That means that while the tumors are growing in the mice, the scientists can look at genetic features of the patient's cancer — helping to zero in on potential therapies faster.

Besides their use in tailoring known treatments to patients, avatar mice are also test-driving entirely new approaches to chemotherapy. At UC Davis, Chong-Xian Pan has transplanted tumors from bladder cancer patients directly into NSG mice to test a new type of drug delivery system. Generally, tumor cells taken from patients are cultured in a laboratory dish before implantation in a mouse. Or, as is often done when testing new drugs, researchers draw on cell lines that have been grown in the lab for years or even decades.



A human bladder tumor (top) grown in a humanized mouse (bottom) is used to test of a new type of treatment.

"Cancer cells are not very stable and easily mutate, so the cell lines most likely are different from those in the patient," Pan says.

After placing tumor cells directly into mice, Pan and his team were able to deliver higher doses of anticancer drugs directly to the cancer cells, without upping toxicity. The new therapy, outlined recently in *Nanomedicine*, will soon move into clinical trials, testing the first new approach to treating bladder cancer in nearly three decades, Pan says.

They're only human

Even though human trials are moving forward for some types of cancer, the humanized mouse has its limits. Not all tumor types grow in the humanized mice. Breast cancer, for example, generally won't take hold. But the main drawback to the system is time, Hidalgo says. It takes four to six months for a colony of mice to grow the tumors and to test how the cancers respond to treatment. So the mice may not be useful for people with primary, easily treatable tumors. But for those who have relapsed and run out of options, the mouse avatar may provide useful, and life-saving, insights.

"It's a good model, if you have everything in place," Hidalgo says. "The ideal situation is to get the study going very early so that by the time the patient's cancer has become resistant to standard treatment, you have new targets on hand."

Meanwhile, researchers continue to tweak the mice to make them even more humanlike. So far, no single mouse can fully replicate a human immune system. And some human immune cells are killed off by proteins made by the mice. As those killer proteins are identified, scientists are working to find ways to disable them.

Scientists also hope to develop strains that include human features beyond the immune system, such as liver cells. Because mice metabolize some drugs differently from the way people do, adding a human liver would allow researchers to test the toxicity of candidate drugs before going to human trials.

"Humans are not mice, so you need to see how the immune cells respond in a system with living human cells," Greiner says. "These mice allow that opportunity." ■

Explore more

- Leonard Shultz, *et al*. "Humanized mice for immune system investigation." *Nature Reviews Immunology*, November 2012.

Mouse milestones

The effort to remake mice to more closely resemble humans in their response to disease began with mice that were missing their own functional immune system. Genetic engineering has allowed further refinements of these mice to enhance the establishment of human immune cells.

1966: Scientists discover a strain of mice, called "nude" mice, with a genetic defect that prevents their immune system from developing. This defect results from a mutation in a single gene. Scientists later discover that the nude mouse lacks a functioning thymus, a gland that plays a key role in immune development.

1983: Scientists discover a mouse strain that lacks both T cells and B cells, and other components of the immune system. This defect results from two flawed copies of a gene and is known as severe combined immunodeficiency, or SCID. Mice with this mutation are ideal for scientific research as they will not reject transplanted foreign tissue.

1988: Two different research groups implant major parts of the human immune system into SCID mice. One group engrafts tissue taken from the liver, thymus and lymph glands into the mice. The other team injects mature immune cells that circulate in the blood (white blood cells) into the animals. Both sets of mice produce some human immune cells and antibodies.

1992–1995: Researchers find a way to get human blood stem cells into the mice. Because stem cells can give rise to the full array of immune cells, this allows more types of human cells to grow when engrafted into mice. In addition, it helps the mice to generate more components of the human immune system, though they still can't produce T cells.

2004–2005: Scientists spur human T cell development in mice by disabling a human gene called IL-2 receptor gamma along with blood stem cells. With working T cells, the mice can be used to study how the human body fights disease.

Today: Mice with a near-complete array of human immune cells are used to study a variety of diseases.

T. LIN *ET AL*/*NANOMEDICINE* 2012

QUAKES IN SLO-MO

Barely detectable tremors may portend major destruction

By Alexandra Witze

Herb Dragert didn't know what to make of his wayward station.

In the early days of GPS satellites, Dragert had set up four benchmarks in the bedrock of Vancouver Island, British Columbia, to watch how their positions changed over time. Maybe, he thought, he could capture the ground moving during the earthquakes that occasionally shake the Pacific Northwest.

But instead Dragert saw one of his stations, at Albert Head on the southern part of the island, throwing a slow-motion tantrum. Every year or two it would inch westward for a few weeks, then stop, then do it again. The movement was far too slow to be an earthquake, but too fast for the ordinary creep of tectonic plates.

Twenty years later, Dragert and his colleagues know that they were seeing something new and important at Albert Head. The phenomenon, known as "slow slip," happens when two sides of a geologic fault shift the same amount as in a large quake, but over weeks to months rather than seconds. "It's like an earthquake, only slower," says codiscoverer Kelin Wang, who, like Dragert, is a geophysicist at the Geological Survey of Canada in Sidney, British Columbia.

Geologists are now learning that slow slip happens in all sorts of places, from Japan to New Zealand to Costa Rica.

Geologists use GPS stations (one shown below on Mount Olympus in Washington state) to detect the gentle ground movements known as "slow slip."

New discoveries reveal how slow slip serves as a transition between ordinary quakes at the surface and those in deeper parts of the Earth where rocks flow like softened butter. And because periods of slow slip have heralded several large recent quakes, including Japan's 2011 Tohoku quake, studying slow-motion events could hint at new and better ways to anticipate the next big one.

Balancing the books

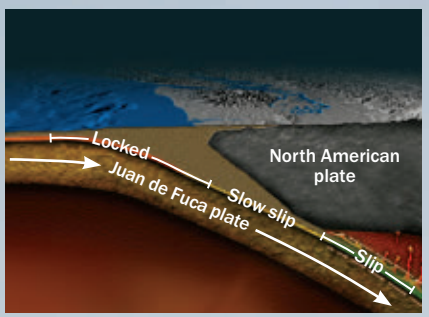
Albert Head sits in a sprawling region of islands, peninsulas and waterways spanning the northwestern corner of the United States and southwestern British Columbia. This cross-border area is in a geological subduction zone known as Cascadia, where one huge plate of Earth's crust dives beneath another. Stress builds up where the Juan de Fuca plate slides beneath the North American plate, and that stress relieves itself occasionally in earthquakes. The last great "megathrust" quake shook the Cascadia coastline in the year 1700, as shown by drowned trees and other silent evidence along the water's edge.

Once the diving, or subducting, plate gets about 50 kilometers down, temperatures are warm enough that the brittle crust starts to flow more easily. Here, rocks move past each other in a sort of flowing motion, relieving accumulated stress so that it doesn't have a chance to build up. Between the upper, rigid part and the lower, flowing part is what scientists had long and unimaginatively called the transition zone. Now it is more accurately called the slow slip zone, because this is where slow slip events happen.

Slow slip helps scientists balance the books at subduction zones by explaining where all the motion of the subducting plate goes. A single slow slip event can account for as much ground movement as a magnitude 7.0 quake or greater. "For many, many years we were missing a lot," says Susan Schwartz, a seismologist at the University of California, Santa Cruz. "It's a very important component that we never knew was there before."

In some locations slow slip happens like clockwork — in Cascadia it appears about every 15 months — while in other places it comes more sporadically. A region can have more than one kind of slow slip; Japan, for instance, has both long-term events that happen every three to five years for several months, and briefer ones that crop up far more frequently.

Along with Cascadia, Japan is the best-studied slow slip spot in the world. In large part that's thanks to sophisticated instruments placed across the country after the devastating 1995 Kobe earthquake. By the early 2000s, Kazushige Obara of the University of Tokyo found that the ground under southwestern Japan shook intermittently with a low

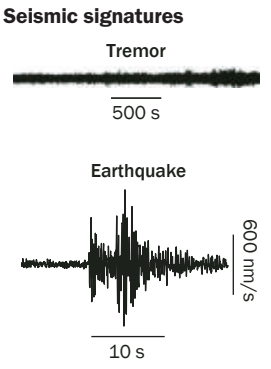


Slip sliding away Off the Pacific Northwest coast, where one slab of crust plunges beneath another, plates are locked in place near the surface but slip slowly and regularly deep underground. SOURCE: UNIV. OF WASHINGTON

PACIFIC NORTHWEST GEODETIC ARRAY/CENTRAL WASHINGTON UNIV.

seismological rattle. This "tremor" appeared on a seismic readout as squiggly lines, like background noise from a windstorm. But it, too, turned out to be a totally new phenomenon.

Slow slip and tremor may represent two faces of the same thing happening deep underground. Usually, slow slip happens too slowly to generate seismic waves. But occasionally small patches along either side of the fault may slip rapidly enough to create seismic waves, just barely distinguishable above background noise. That's tremor.



SOURCE: J. GOMBERG *ET AL*/ *GSA BULL.* 2010

Slow slip is often accompanied by tremor (top), which appears on a seismogram as a signal quite different from that of an ordinary quake (bottom).

Tremor often happens along with slow slip, although sometimes it crops up before or after slow slip, or not at all. Occasionally tremor reverses direction and speeds up dramatically for a little while. Why that happens remains a mystery. "There's a lot we don't know," says John Vidale, a seismologist at the University of Washington in Seattle.

Seeds of destruction

Answering such questions is crucial because of the risk involved. Cascadia is either due or overdue for its next great megathrust quake, and building codes and earthquake preparedness in the region lag behind seismically active parts of California. A 2009 paper in *Geophysical Research Letters* reported that slow slip is happening so deep within Cascadia that when a really big earthquake does happen, its shaking could extend farther inland than once thought. That means it could hit people in much of western Washington and some of Canada, not just along the coast (*SN Online: 11/24/09*).

The biggest slow slip event ever, by some measures, took place last year in Cascadia. It began in late August, when seismometers picked up tremors in a patch beneath southern Vancouver Island. Over time the patch spread; defying national borders, it moved south into Washington and then beyond Seattle. In mid-October it faded, but by then it had lasted 42 days and included a total of 618 hours of tremor — the longest such episode yet detected. Slow slip events have been detected in this area at least six times before, but never for this long, Vidale says.

The big question is what this means for seismic hazards in Cascadia. Some scientists think that slow slip may increase stress on particular patches of the subducting plate. Studies of an August 2009 slow slip event in Cascadia found that the tremor was concentrated in areas where the fault was slipping most rapidly. So slow slip may be transferring stress and loading up patches that become more prone to rupture in a great quake.

Theoretical work backs this up. Paul Segall, a geophysicist at Stanford, uses computers to simulate what happens when factors like fluid pressure, heat transport and friction change at subduction zones. Some of his results suggest that slow slip events could lead to runaway slipping that culminates in a megathrust quake. But so far there's no way to tell in advance when the slip is going to run away and when it's not, Segall and Andrew Bradley reported in September in *Geophysical Research Letters*.

One scientific challenge is understanding whether big quakes are actually triggered by slow slip. The 1999 Izmit quake in Turkey may have been; so too the March 2011 Tohoku quake in Japan.

A team led by Aitaro Kato of the University of Tokyo has found that two sets of slow slip happened very near the epicenter of the Tohoku quake just before it happened. The first slow slip event ran from middle to late February 2011. After the slow slip came a magnitude 7.3 earthquake; this would turn out to be the largest of the foreshocks before the great quake.

Next, a second episode of slow slip began and shifted westward toward the place where the seafloor eventually ruptured in the massive 9.0 quake. Scientists still don't understand what all this means: whether the slow slip directly triggered the huge quake, or whether it was all simply associated seismic activity but not a direct cause.

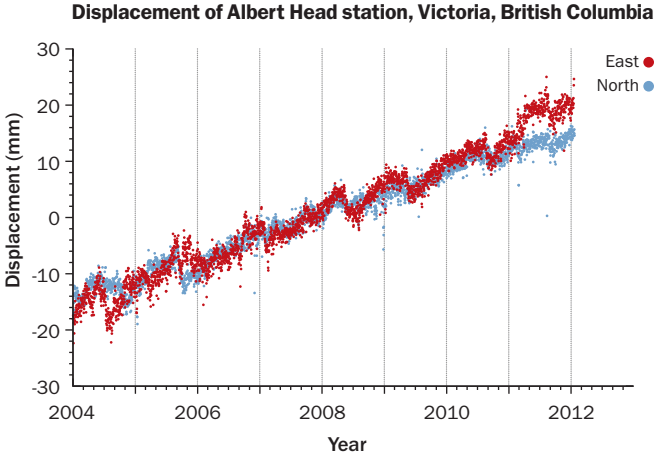
"We would love to understand how preparation for large earthquakes happens," says Schwartz. "If slow slip is part of that preparation process, it's really important — but I don't think we can say that yet."

Winning the lottery

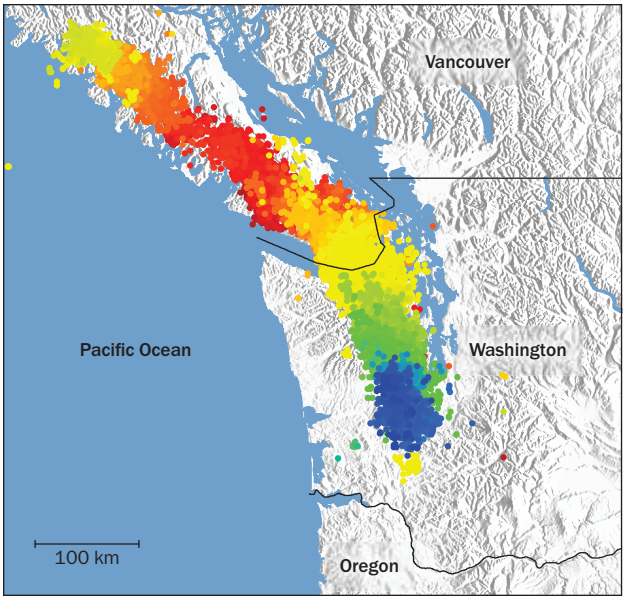
One approach is to look at places both before and after a big quake to see what role slow slip played, if any. That kind of

Repeat slip

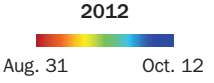
Every 15 months or so the GPS station at Albert Head, British Columbia, makes a sharp westward movement, temporarily reversing its gradual eastward drift (red dots). These sudden reversals over the course of weeks to months signal slow slip deep underground. (Blue dots represent north-south motion.)



SOURCE: BEN WEBBER/ WIKIMEDIA COMMONS



Migrating quakes
Between August and October 2012, the Pacific Northwest experienced the longest slow slip event ever recorded. It started on Vancouver Island in Canada (red dots), and over several weeks migrated south into the Olympic Mountains and deeper into Washington state (blue dots).
SOURCE: A. WECH



measurement is tough in Japan, where much of the subducting plate is buried far offshore. It's a little easier in places like Costa Rica, where the Cocos crustal plate dives down quite close to the Nicoya peninsula. That means researchers can set up monitoring stations on land, much closer to the place where slow slip is happening — about 15 kilometers below the shoreline — than they can at other subduction zones. "In Costa Rica, we can really sense what's going on in the shallower part," says Schwartz.

Schwartz and her collaborators have studded the Nicoya peninsula with GPS stations and seismometers, and find that big slow slip events accompanied by tremor happen there about once every two years. The last was in summer 2011, so the next big slow slip event may begin this summer.

Catching that event would be interesting because Costa Rica had a magnitude 7.6 earthquake last September. There may have been some tremor in the weeks before that quake, Schwartz says. Her team originally set up their instruments because they knew the subduction zone there was getting ripe for a large quake, and they caught it right in the act. Her goal now is to keep monitoring the plate boundary to see how it heals after a great rupture. One major question, for instance, is whether future slow slip events will happen in the same place on the subducting plate as they did before the September earthquake.

Other scientists will be watching a little farther north along the same subduction zone, off the Mexican state of Oaxaca. There, in March 2012, a magnitude 7.4 quake hit after slow slip — but not tremor — increased in the months beforehand. The slow slip seemed to migrate over time, moving toward where the eventual main shock struck. "Large earthquakes don't occur during each slow slip event, but it does appear that chances of a large earthquake may increase during a slow slip event," Stefany Sit of Miami University in Ohio reported at an American Geophysical Union meeting in December in San Francisco. In 2001–2002, seven months of slow slip in Mexico released the equivalent of a magnitude 7.5 quake — the biggest ever by that measure.

As seismologists gather more field data on how slow slip happens, other scientists are working to simulate the phenomenon using computers. One recent simulation seems to reproduce pretty well what's happening in places like Cascadia, Costa Rica and Oaxaca.

Harmony Colella of Miami University in Ohio crunches numbers to recreate what might happen for hundreds of thousands of slow slip events in real subduction zones. The model incorporates four important factors, including how much slip happens in each event and how long it lasts. Colella's results show that far more slip tends to happen toward the deepest part of the subducting plate, and far less at shallower depths.

Conceptually that finding sounds obvious, but "with the model we can say this is how it works, based on the physics," says Colella. "We see a lot of really interesting things as well" — like the fact that the largest slow slip events are followed by a relatively quiet period. That may mean that it takes a while for stress to build up again to the level where it can trigger another slow slip event. "These things are so much more complex than traditional earthquakes," Colella says.

For now, scientists are thinking about how to find out as much about slow slip as they can, as quickly as possible. One idea is to drill directly into a fault that experiences slow slip, to study properties like how fluid flows through slipping rock and how stress varies from place to place.

A group led by Laura Wallace of the University of Texas at Austin has its eye on the Japanese drill ship *Chikyu*, which can drill deeper into the seafloor than any other research vessel. The scientists are trying to drum up support to drill into the Hikurangi subduction zone off the coast of New Zealand, where slow slip happens every 18 to 24 months at depths of just five to 15 kilometers. "If we could do this it would be like winning the lottery," Wallace said at the AGU meeting.

To some, scientists have already won the lottery merely by discovering slow slip. "Each passing year we're finding this phenomenon is more and more pervasive," says Vidale. "It's not just a few places where we have our best networks — it's everywhere we know how to look." ■

Explore more

- Pacific Northwest Seismic Network: www.pnsn.org
- Cascadia Hazards Institute: www.cascadiahazards.org

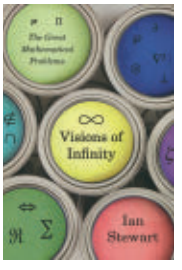
Visions of Infinity

Ian Stewart

The four-color map problem can be understood by a bright fourth-grader (the question: whether four colors are enough to ensure that no two countries with a common border share a color). By junior high, most kids can grasp prime numbers and learn something about their properties and patterns. High school algebra students can comprehend what Fermat's last theorem means. Yet these topics have for decades, or even centuries, occupied the world's most sophisticated mathematicians.

Stewart tells the story of the quest to prove propositions related to these and other topics, not so easily understood, posing problems regarded as the toughest in the mathematical world. For some, proofs have been found; for others, large cash prizes await whoever ultimately succeeds. Stewart describes the steps and missteps along the way that have sometimes opened up entirely new fields of mathematical research, or have revealed deep connections among fields previously thought unrelated.

Stewart's take is no superficial popularization; although much is accessible to a determined reader with deep interest, following the threads he weaves in detail requires a fair amount of willingness to reread. And while his grasp on mathematical history is thorough, physics fans may notice that in a chapter on a mathematical physics problem he stumbles on historical detail: He misstates the chronology of particle physics discoveries and attributes the explanation of atomic isotopes in 1911 to Amedeo Avogadro, who died in 1856.



Still, as a guide to the inner workings of the mathematical jungle, Stewart provides an engaging and informative experience. If you wish to intelligently discuss the Riemann hypothesis, P/NP problems or the Hodge conjecture, you ought to read this book first.

— Tom Siegfried

Basic Books, 2013, 340 p., $26.99

The Lady and Her Monsters

Roseanne Montillo

If the makers of *Downton Abbey* want to capitalize on the popularity of costume dramas, they might look for their next Lady Mary in Mary Shelley, the author of *Frankenstein*. Shelley's life needs no embellishment, complete with preposterous plots and love triangles set in an era of intense scientific curiosity about the human body. In this biography, Montillo explores how the science of that time inspired Shelley's work.

In her 1831 update of *Frankenstein*, Shelley wrote that the story she had penned 15 years earlier emerged from a summer telling ghost stories on Switzerland's Lake Geneva. Montillo paints a more detailed picture of the book's origins.

A key character in the backstory is Italian physician Luigi Galvani, who spent years studying "animal electricity," the idea that an electric shock or current could restore activity to a dead body. The technique brought Shelley's fictional monster to life. After Galvani had sacrificed countless frogs, his nephew Giovanni Aldini set his sights on reanimating a person. He famously tried in London in 1803 with a recently hanged criminal but, of course, failed.

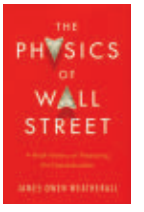
Montillo lays out how the well-read Shelley may have learned of Aldini's experiments, come across the ideas of Renaissance alchemists Paracelsus and Cornelius Agrippa that turn up on Victor Frankenstein's bookshelf, and even perhaps stumbled upon the name Frankenstein. By putting Shelley's exceptional life story in historical context, Montillo tells a tale more compelling than any soap opera. *— Kate Travis*

William Morrow, 2013, 322 p., $26.99

Mankind Beyond Earth

Claude A. Piantadosi

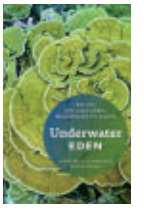
This history of the American space program illuminates the obstacles facing future space expeditions. *Columbia Univ., 2013, 279 p., $35*



The Physics of Wall Street

James Owen Weatherall

A physicist explores the growing role of scientists in Wall Street decision making and recent financial abuses. *Houghton Mifflin Harcourt, 2013, 286 p., $27*



Underwater Eden

Gregory S. Stone and David Obura

Amazing photos make visible the reasons behind efforts to save a biological wonder: the coral reefs of the South Pacific's Phoenix Islands. *Univ. of Chicago, 2013, 170 p., $40*

The White Planet

Jean Jouzel, Claude Lorius and Dominique Raynaud

A team of scientists tells the story of ice on Earth, from ice ages to the latest discoveries from ice cores. *Princeton Univ., 2013, 306 p., $29.95*

Science on American Television

Marcel Chotkowski LaFollette

This history of science programs illustrates the shifting line between education and entertainment. *Univ. of Chicago, 2013, 306 p., $45*

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Grand Canyon rising

If the geology of the Grand Canyon dates back to 70 million years ago ("Grand Canyon's age pushed back," *SN: 1/12/13, p. 15*), that would be around the same time the Rocky Mountains were being pushed up by the subduction process originating off the western continental coast. Could the lifting of the Colorado Plateau be related to the lifting of the Colorado Rockies?
Joe Flynn, Spanaway, Wash.

The timing of the Colorado Plateau's uplift remains fairly controversial, including whether it occurred all at once or in several discrete stages. But many geologists think it may have been at least partially related to the same event (known as the Laramide orogeny) that raised the Rocky Mountains between about 70 million and 40 million years ago. —Alexandra Witze

Seeing clearly

Regarding your article on the surge of nearsightedness ("Urban eyes," *SN: 2/9/13, p. 22*): When my mother taught third grade in the early 1930s, teachers were instructed to have their students stop their work on a regular basis and look out the large bank of windows on the north to the distant mountains. She often said that there is nothing new in education; the experts just rediscover the old.
Carolyn Conner, Chula Vista, Calif.

As a teenager in the '60s, I saw an article on submarines mentioning that men on sub duty become nearsighted (irreversibly, it was thought). The thinking was that living weeks at a time with nothing more than six feet away was to blame.
Simon G. LePorte, Hanson, Mass.

A 1983 report from the U.S. Naval Submarine Medical Research Laboratory in Connecticut cited French research showing some visual discomfort among submariners. Shortly after returning from sea, some reported they "could not see as well as usual" and had problems driving at night, the study found. But no abnormalities were found in their eyesight two weeks later. The report also cited a study of 18 men kept indoors for 45 days who showed changes in eyesight toward myopia, mainly in men who were already nearsighted or farsighted. But these also normalized after two weeks of outdoor light exposure. —Nathan Seppa

Going between sun and shade exercises iris muscles; looking from flower to bird in tree to clouds exercises lens muscles. Exercise improves muscle fitness, helping to maintain normal vision. As in all muscles, use it or lose it.
Patricia Thomas, Streator, Ill.

New Silicon Valley

I enjoyed Alexandra Witze's article on synthetic biology ("Factory of life," *SN: 1/12/13, p. 22*), but it left something out. Clearly, that area of the country now needs its own nickname, a la Silicon Valley. I propose that it be called Carbon Harbor, which sounds especially good with a Boston accent.
Drew Massey, Sherman Oaks, Calif.

As someone whose father hails from Providence and talks about pahking the cah in Hahvahd Yahd, I think this reader is onto something. —Alexandra Witze

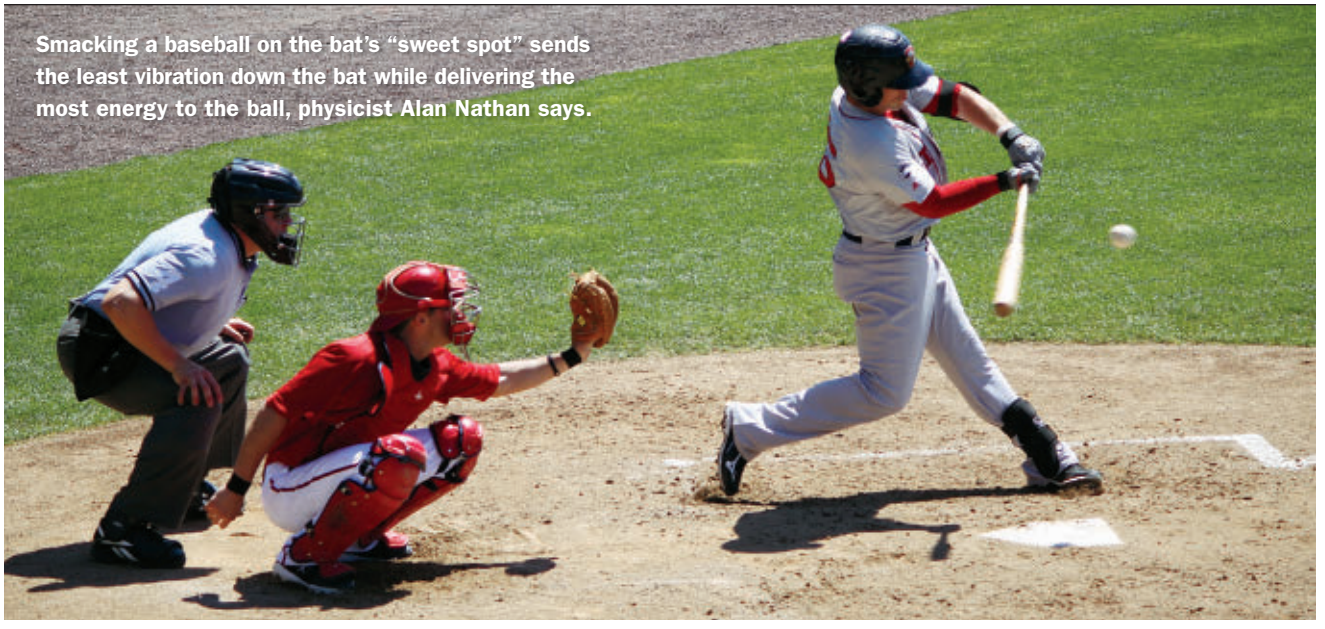
Women's sports sociology

I believe the middle female football player in the "girls on the gridiron" photo ("Tackling women's pro football," *SN: 2/9/13, p. 32*) is my mother, the former Alice Holmberg, who graduated from Gustavus Adolphus College in 1927. My mother said the whole thing was mostly a lark, as none of the girls knew anything about football. In her defense, I should mention that my mother was quite an accomplished athlete in other sports. She is memorialized in the Gustavus Adolphus athletic hall of fame for her basketball skills. Thanks for taking me down memory lane.
Jack Meyer, St. Peter, Minn.

Send communications to: Editor, Science News, 1719 N Street, NW, Washington, D.C. 20036 or **editors@sciencenews.org**. Letters subject to editing.

The Science Life

Learn more about baseball physics at **baseball.physics.illinois.edu**



Smacking a baseball on the bat's "sweet spot" sends the least vibration down the bat while delivering the most energy to the ball, physicist Alan Nathan says.

Baseball's resident physicist

Picture sitting at a baseball park, leisurely watching a game. Your mind wanders, torn between a box of Cracker Jack and the conversation drifting down from the row behind you. Suddenly the crack of a bat snaps you to attention, and you scan the field for the ball. Physicist Alan Nathan would say your attention is piqued because well-hit balls make a different noise than weak pop-ups do.

"You hear a high-frequency sound," says Nathan (left). There's a duller thud when the ball misses the bat's "sweet spot," he says, the point where a hit maximizes the outgoing speed of the ball.

Physics and baseball are a natural marriage, as Nathan realized 16 years ago when he took a break from experimental particle physics to talk to high school kids about science and casually chose baseball as the topic. He got hooked and spent a whole sabbatical analyzing the bat-ball collision, the dynamic core of the sport.

His early work centered on the idea that the energy of the collision must go somewhere, and he spelled out how much gets transferred to the ball and how much is lost as vibrations run down the bat. Nathan recently tallied the average speed needed for a hit ball to leave each major league park. It turns out that players really need to whack a ball to launch it in Atlanta and Phoenix — not so much in Denver (thin air) and Boston (short distance to the left field stands). He has also debunked some long-held ideas. For instance, he has shown that "corking" a wood bat by hollowing out its center doesn't make a ball fly farther. A corked bat moves faster but is lighter, so it doesn't deliver more force, and the wood fails to deliver a "trampoline effect" like aluminum would (see sidebar, right).

These investigations have given Nathan a high regard for players. "The way the game is played evolved over 150 years, through trial and error," he says. "Ballplayers know how to play the game. They may not know the reason for the right way to do it," he says, but they manage anyway. "My goal is to merely understand what's going on."

Now retired from the University of Illinois at Urbana-Champaign, Nathan continues to do research and consults with college and professional baseball organizations, including serving on the committee that established minimum bat weights for college baseball.

— Nathan Seppa

Greatest hits of physics

A baseball heats up when hit, and so does the science. Here's what physicists have observed about the bat-ball collision.

- **Look ma, no hands** In 2012 Cincinnati Reds player Todd Frazier (above) hit a home run with one hand completely off the bat and the other barely touching it, showing that from a physics standpoint it doesn't matter how (or if) a batter holds a bat.
- **Bat speed matters** A fast swing transfers measurably more energy to a ball than a slow swing does. Each mile per hour added to the swing speed is probably worth an extra six feet on a long fly ball, after accounting for wind speed and loft.
- **The secret of metal** Hit balls travel farther off a metal bat than a wooden one of the same weight because aluminum bats are hollow and provide a slight "trampoline effect" that returns energy to the ball.

CLOCKWISE FROM TOP: RICHARD PAUL KANE/SHUTTERSTOCK; MLB; THOMPSON/MCCLELLAN PHOTOGRAPHY; PHYSICS DEPT./UNIV. OF ILLINOIS AT URBANA-CHAMPAIGN

Chicago Doctor Invents Affordable Hearing Aid

Outperforms Many Higher Priced Hearing Aids

Reported by J. Page

CHICAGO: A local board-certified Ear, Nose, Throat (ENT) physician, Dr. S. Cherukuri, has just shaken up the hearing aid industry with the invention of a medical-grade, affordable hearing aid. **This revolutionary hearing aid is designed to help millions of people with hearing loss who cannot afford—or do not wish to pay—the much higher cost of traditional hearing aids.**

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Dr. Cherukuri knew that untreated hearing loss could lead to depression, social isolation, anxiety, and symptoms consistent with Alzheimer's dementia. **He could not understand why the cost for hearing aids was so high when the prices on so many consumer electronics like TVs, DVD players, cell phones and digital cameras had fallen.**

Since Medicare and most private insurance do not cover the costs of hearing aids, which traditionally run between $2000-$6000 for a pair, many of the doctor's patients could not afford the expense. Dr. Cherukuri's goal was to find a reasonable solution that would help with the most common types of hearing loss at an affordable price, not unlike the **"one-size-fits-most" reading glasses** available at drug stores.

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He evaluated numerous hearing devices and sound amplifiers, including those seen on television. Without fail, almost all of these were found to amplify bass/low frequencies (below 1000 Hz) and not useful in amplifying the frequencies related to the human voice.

Inspiration from a surprising source

The doctor's inspiration to defeat the powers-that-be that kept inexpensive hearing aids out of the hands of the public actually came from a new cell phone he had just purchased. **"I felt that if someone could devise an affordable device like an iPhone® for about $200 that could do all sorts of things, I could create a hearing aid at a similar price."**

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"We ordered two hearing aids for my mother on Sunday, and the following Wednesday they were in our mailbox! Unbelievable! Now for the best part—they work so great, my mother says she hasn't heard so good for many years, even with her $2,000 digital! ***It was so great to see the joy on her face.*** *She is 90 years young again."* —Al Peterson

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BBB RATING A

Time travel at the speed of a 1935 Speedster?

The 1930s brought unprecedented innovation in machine-age technology and materials. Industrial designers from the auto industry translated the principles of aerodynamics and streamlining into everyday objects like radios and toasters. It was also a decade when an unequaled variety of watch cases and movements came into being. In lieu of hands to tell time, one such complication, called a jumping mechanism, utilized numerals on a disc viewed through a window. With its striking resemblance to the dashboard gauges and radio dials of the decade, the jump hour watch was indeed "in tune" with the times!

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