Tracking Antarctic Invaders | Wordy Baboons | Super Statins

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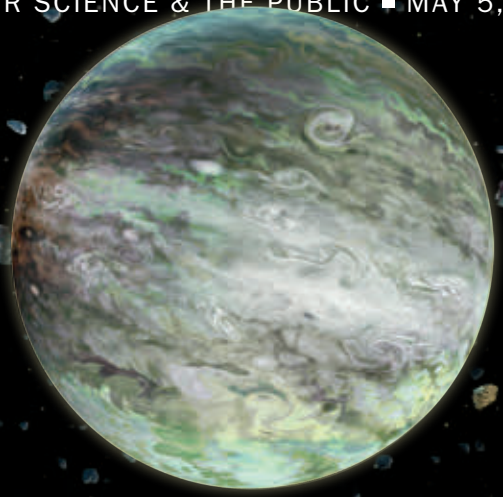
ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE & THE PUBLIC ■ MAY 5, 2012

T. Rex's Feathered Cousin

Playing with Stone Age Fire

Einstein Vindicated Again



Born Giants

How the solar system's outer planets came to be

Technology Simplified

WOW…A Computer Designed For YOU, Not Your Grandchildren!

NEW

…It's easy to read. It's easy to see. It's even easier to understand and use!
Just plug it in!!!

Have you ever said to yourself "I'd love to get a computer, if only I could figure out how to use it." Well, you're not alone. Computers were supposed to make our lives simpler, but they've gotten so complicated that they are not worth the trouble. With all of the "pointing and clicking" and "dragging and dropping" you're lucky if you can figure out where you are. Plus, you are constantly worrying about viruses, spam and freeze-ups. If this sounds familiar, we have great news for you. There is finally a computer that's designed for simplicity and ease of use. It's the WOW Computer, and it was designed with you in mind.

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> I just wanted to tell *first*STREET that I am having a great time on my WOW computer. I am learning something new everyday. I am 79 years old and cannot believe that I am typing and sending e-mails to all my friends now. My daughter and granddaughter are so excited now that I have a computer. They use computers on their jobs everyday, but they cannot believe what you can do on this computer. It is wonderful… Thanks.
>
> – Johnnie E., Ellijay, Ga

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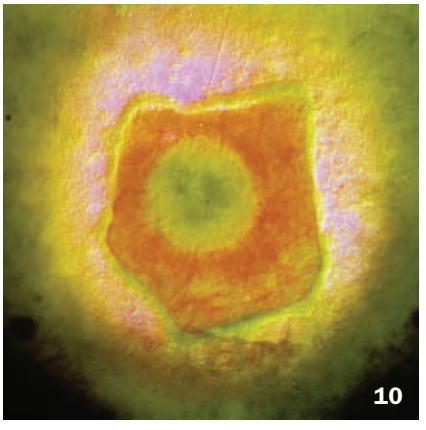

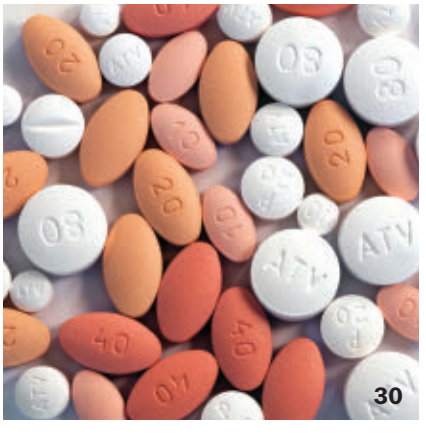

PILLS: © KUMAR SRISKANDAN/ALAMY

ScienceNews

In The News

Features

Departments

COVER An illustration of the early solar system depicts one giant planet (center) that might have been ejected, one theory proposes, leaving the four known giants behind.
Nicolle Rager Fuller

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Preferred periodicals postage paid at Washington, D.C., and an additional mailing office.
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FROM THE EDITOR

DNA disease tests should come with warning label

Possessing the complete catalog of all human genes — the genome — has a lot of scientific value. It enables studies that illuminate aspects of evolution through comparison with genomes of other species. It facilitates scientific studies of the roles various genes play in normal biological activities. And it even aids the effort to understand the relationships between certain gene variants and human diseases.

It's not so good, though, at forecasting what specific diseases any individual human is likely to suffer from.

A new mathematical analysis, based on health reports from more than 50,000 pairs of identical twins, finds that analyzing your genome would help predict your risk for only a very few diseases, as Tina Hesman Saey reports (Page 11). She cites one scientist's view that you're better off weighing a patient and asking about smoking habits.

All this should come as no great surprise. Even though there has been a lot of hype over the years about the medical benefits of cataloging the genome — and companies sell DNA testing services that supposedly identify medical risks — sober analyses have repeatedly noted that the science doesn't support some of the more extravagant claims. Nearly three years ago, for instance, readers of *Science News* encountered a feature article on tests people could buy to have their DNA analyzed (*SN: 7/4/09, p. 16*). As the feature noted, test-buyers received reports linking specific features in their DNA to elevated or reduced risks of various diseases. "But the genetic report cards these amateurs are reading may not be as definitive as they assume," the article pointed out. "Despite progress in linking genetic differences with disease risk and other traits, the predictive power of these links has fallen short of expectations."

It's not that such tests have no value at all. Genetic testing can identify elevated risks for a few diseases. And as genetic knowledge improves, and better medical interventions are devised, and the interactions of genes with environmental effects are better understood, DNA testing will no doubt play a role in providing better, and more personalized, medical treatment. But that will come only as exaggerated expectations are tempered by solid evidence. And sorting the hype from the scientific substance is what both good medical practice and good science journalism are all about.

—Tom Siegfried, Editor in Chief

JIM WEBB

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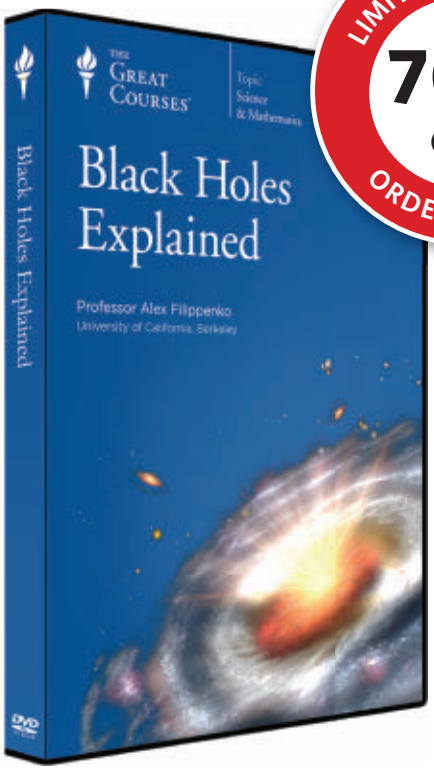

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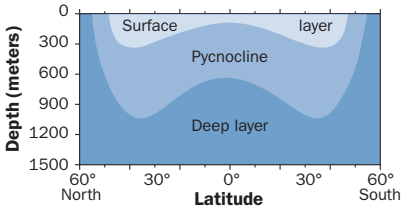
Say What?

Pycnocline ***PIHC-noh-kline***\ n. A boundary or layer with varying water density caused by changes in temperature or salinity. In oceans and lakes, these stratifications help control which organisms live in which water layers, for instance by concentrating plankton that attract predators. Now, researchers have found that the density changes have a fundamental effect on marine creatures. Using calculations of how a pycnocline physically affects a single cell, scientists at the University of Notre Dame and MIT report March 6 in the *Proceedings of the National Academy of Sciences* that differences in buoyancy and viscosity force small creatures to use more energy as they swim. —*Alexandra Witze*



SOURCE: AMERICAN METEOROLOGICAL SOCIETY

Science Past | FROM THE ISSUE OF MAY 5, 1962

CANCER CAUSE IN TOBACCO — "You might as well ask a person if he believes the earth is round as to ask him if he is one of those who believes cigarettes cause cancer," Dr. Charles B. Huggins, director of the Ben May Laboratory for Cancer Research, University of Chicago, told SCIENCE SERVICE.... Sixty known cancer-causing compounds have been tested.... Two components of deoxyribonucleic acid — guanine and cytosine — were made into a molecular model and a plastic frame was constructed to surround it. In this frame, Dr. Huggins showed, in slides, how all known cancer-causing aromatic hydrocarbons fit neatly. Also similar atomic models of the steroids testosterone (male hormone) or progesterone or estradiol (female hormones) fit.

Science Future

May 16
Test your mettle at science trivia night at Washington, D.C.'s Koshland Science Museum. Prizes go to the winning team. See bit.ly/SFtrivia

May 19
The Orlando Science Center holds a Science of Wine event, with educational events and wines from around the world. More information at bit.ly/SFoscwine

SN Online

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EARTH
Horizontal motion makes a magnitude 8.6 quake less dangerous. Learn more in "Indonesian quake passes without major tsunami."

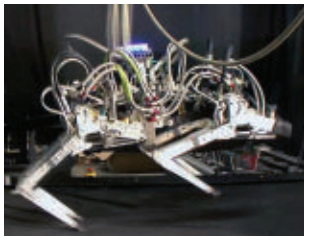
DELETED SCENES BLOG
A video game puts birds into orbit. Read "The Newtonian physics (or not) of *Angry Birds Space*."

GENES & CELLS
Altering gene activity may make chemotherapy more effective. See "Old cancer drugs offer new tricks."

SCIENCE & THE PUBLIC BLOG
Recent U.S. record warmth was not shared globally. Read "March: American heat vs. global temps."

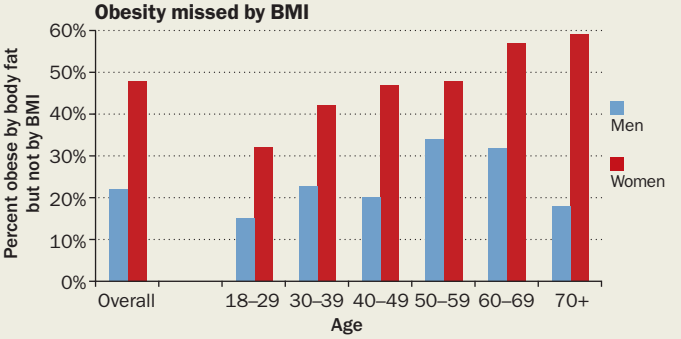
The (-est)

Reaching speeds up to 29 kilometers per hour (18 miles per hour), a new robot is the fastest of its kind. Called Cheetah and designed by DARPA, a U.S. Department of Defense research agency, the device breaks the previous record of about 21 km/h for land-based, legged robots. Cheetah's leg movements mimic the strides of some of nature's speediest runners. Though there is no immediate application planned for the robot runner, a DARPA spokesperson says that scientists are interested in building speedy machines for a variety of purposes, such as assisting in dangerous military missions.
—*Rebecca Cheung*



Science Stats | FATTER THAN YOU THOUGHT

About a third of Americans are estimated to be obese, based mostly on measurements of body mass index, or BMI. But new research shows that BMI underestimates obesity compared with directly measuring the percentage of body fat. Underestimates are most common in older women, who lose more muscle with age than men do.



SOURCE: N.R. SHAH AND E.R. BRAVERMAN/*PLOS ONE* 2012

CLOCKWISE FROM TOP LEFT: STEPHEN EGTS; © 2012 ROVIO ENTERTAINMENT LTD. ANGRY BIRDS; DARPA

“You can just dress your satellite in a thermal cloak.”
—SEBASTIEN GUENNEAU, PAGE 19

In the News

STORY ONE

Baboons learn to tell words from nonwords

Reading may rely on visual skills shared by all primates

By Bruce Bower

Baboons such as this one learned to distinguish real words from nonsense words in a new study. The work suggests that the initial stages of learning to read involve purely visual recognition of letters and letter combinations.

COURTESY OF J. FAGOT

Baboons hang out in the bush, not in bookstores. Yet these avid nonreaders can learn to tell written, real words from nonsense words, a new study finds.

That surprising achievement is not the same as reading, say psychologist Jonathan Grainger of the University of Aix-Marseille in France and his colleagues: Baboons tested in the new study didn’t attach meanings to words. Crucially, though, these animals demonstrated that the roots of deciphering alphabetic script lie in brain functions that have nothing to do with language, Grainger’s team reports in the April 13 *Science*.

“We think our baboons learned to distinguish between specific combinations of letters that mostly appear in words versus combinations of letters that mostly appear in nonwords,” Grainger says.

If so, his investigation challenges the long-standing assumption that knowledge about spoken language informs the earliest stages of reading acquisition. According to that perspective, children get the literary ball rolling by matching written letters to corresponding speech sounds.

Instead, Grainger proposes, reading initially taps into brain regions that recognize different objects by sight and that evolved in monkeys and humans — and perhaps all primates. The baboons in the study drew on this capacity to track pairs of letters that distinguish real from bogus words. That knowledge enabled the monkeys to learn dozens of four-letter English words and to tell whether new four-letter sequences qualified as words or not.

“For the first time, we have an animal model of a key component of literacy — the recognition of the visual word form,” comments cognitive neuroscientist Stanislas Dehaene of the INSERM-CEA Cognitive Neuroimaging Unit in Gif-sur-Yvette, France.

For the new investigation, Grainger’s team studied six baboons housed in a research facility with indoor and outdoor areas. Monkeys had free access to touch-screen computers, which they could reach through openings in a partition.

In a series of computer sessions, the baboons learned to recognize English words, such as *done* and *vast*, and to distinguish actual words from four-letter nonsense strings, such as *dran* and *virt*. Animals received food if they touched a cross on a computer screen after seeing a word, or if they touched an oval shape after seeing a nonword. New words were presented to baboons as the animals’ pool of learned words expanded.

Over a month and a half, individual

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baboons learned to recognize between 81 and 308 words, which they distinguished from more than 7,000 nonwords with about 75 percent accuracy.

Rather than simply memorizing what specific words looked like after many presentations, monkeys came to realize that certain letter combinations distinguished novel real words from fake ones, Grainger says. Baboons correctly identified novel nonwords as bogus more often than they incorrectly tagged actual words seen for the first time as nonwords. Absent knowledge of letter patterns that characterize genuine words, baboons would have mislabeled many more unfamiliar words as nonwords, Grainger holds.

The results set the stage for future studies examining whether brain areas activated while baboons identify words and nonwords correspond to the area of the human brain that's stimulated during reading, Dehaene says.

Dehaene and his colleagues have reported that, in people, reading selectively activates a left-brain region that they call the visual word form area. Responses of this neural tissue to written material become stronger as children get older and are related to reading ability.

Reading and writing originated roughly 5,000 years ago, long after the modern brain had taken shape. So

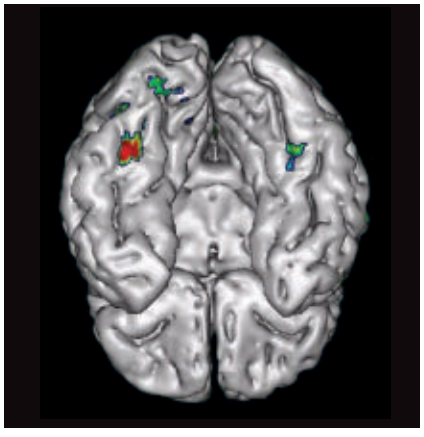
In a new study, baboons pressed symbols on touch-screen computers to indicate whether four-letter strings, such as *vast* and *virt*, were words or not. Correct answers yielded food rewards.

neural terrain built for object and face recognition was probably recruited for the visual word form area as people learned to discern letters and letter arrangements in words, Dehaene says.

The new findings also fit with a proposal by evolutionary neurobiologist Mark Changizi of 2AI Labs in Boise, Idaho. He suggests that the shapes of written scripts derive from the contours of objects in natural scenes that human brains' visual systems home in on.

By grounding written letters in shapes inherently preferred by the brain, "writing systems could be more easily learned and were thus more likely to survive and spread through a culture," write neurobiologists Michael Platt and Geoffrey Adams, both of Duke University in Durham, N.C., in a commentary also published in the April 13 *Science*.

Just as some people labor to read even deftly assembled scripts, two baboons in the new study struggled to learn to tell words from nonwords. "Understanding what determines how well a baboon performs on our discrimination task might offer some insights into a possibly visual component of reading deficits in certain dyslexic children," Grainger says. ■



Back Story | READING ON THE BRAIN

Written language, and thus the ability to read, evolved relatively recently. So scientists assume that the act of reading must co-opt brain areas that originally evolved for other functions, such as vision and speech. Still, imaging studies suggest that learning to read may tune a chunk of the brain called the visual word form area to recognize the written word. Scans (left, brain viewed from below) have shown that looking at words turns on a spot (red) in the left hemisphere. This patch of cells, in the fusiform gyrus, doesn't respond as strongly to letters arranged nonsensically and ignores words spoken aloud. "It's picking up on statistical patterns and playing a huge role in the information processing of visual words," says neuroscientist Bruce McCandliss of Vanderbilt University. But critics tend to emphasize other activities that turn on this part of the cortex. Naming colors or deciphering Braille can also activate it—suggesting that reading is just one use of this multifunctional area. —*Devin Powell*

FROM TOP: COURTESY OF J. FAGOT; S. DEHAENE

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Demand– The red-hot demand for precious silver, coupled with the low worldwide mintage of only 20,000 caused the quick sell-out of the 2010 coin. It now sells for as much as $242 in high grade—only one year later! With the same low mintage, the 2011 coin could be the next to sell-out.

Silver Trend Chart: Prices based on monthly averages.

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Environment

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Pesticide-dosed bees can lose royals, way home

In tests, nonfatal exposures lead to shrinking colonies

By Susan Milius

What does not kill them does not in fact make them stronger when it comes to bees and pesticides. Two unusual studies with free-flying bumblebees and honeybees find that survivable exposure to certain pesticides can lead to delayed downturns in bee royalty and a subtle erosion of workforces.

Pesticides appear as a suspect in widespread declines, some subtle and some striking, of bees and other animals that pollinate crops and wild plants. And in one of the most dramatic still-unsolved mysteries in those declines — why honeybee colonies suddenly collapse — one leading hypothesis combines chronic pesticide exposure with other stressors such as disease.

Both new studies, appearing online March 29 in *Science*, test the risks of foraging on flowers treated with common insect killers from the nicotine-related class called neonicotinoids. These pesticides course through the whole plant, killing aphids and a range of other nibbling and sipping pests, but also work their way into the nectar and pollen that bees collect.

To simulate pesticide exposures that bumblebees might encounter when a field of canola blooms, entomologist Dave Goulson of the University of Stirling in Scotland and his colleagues fed 50 *Bombus terrestris* lab colonies nonfatal doses of the pesticide imidacloprid. After two weeks of eating spiked pollen and sugar water, bees were set outside and allowed to forage around the Stirling campus at will. By season's end, the pesticide-dosed colonies were an average of 8 percent to 12 percent smaller than 25 unexposed neighbor colonies.

A great yellow bumblebee makes its rounds. Low doses of pesticides caused bumblebee colony sizes to drop and the colonies to produce fewer queens.

More noticeably, the contaminated colonies managed to produce only about two young queens each. The other colonies averaged about 14. Pitiful production of new young queens bodes ill for bumblebees because all other colony members die at the end of the growing season. Young queens represent each group's sole hope for making new colonies the next year.

A drop in pollinator reproduction is the kind of finding that can get the attention of agencies regulating pesticide use, says Jeffery Pettis, a U.S. Department of Agriculture bee researcher in Beltsville, Md. With these and previous studies, concerns are growing that usage rules for neonicotinoids may need to be tightened.

Goulson's study ranks as the first in bumblebees of pesticide side effects under natural field conditions, says Guy Smagghe of Ghent University in Belgium. Smagghe also works with bumblebees, which many wildflowers and crops such as tomatoes and peppers depend on.

For honeybees, earlier tests have raised the possibility that chronic, nonfatal exposure to neonicotinoids impairs learning, memory and other capacities that bees need for good flower hunting. To set up a test with bees flying freely outdoors, a research team in France used dental cement to fasten electronic identifiers onto more than 600 bees. Feeding bees low doses of the pesticide thiamethoxam in sugar water provided a realistic exposure, says coauthor Mickaël Henry of the French National Institute for Agricultural Research in Avignon.

After sipping the pesticide-tainted solution, the honeybees were moved up to a kilometer from their hives and released to find their way home. Researchers challenged bees with both familiar territory and landscapes the bees had never seen. Automated counters at hives logged the returnees.

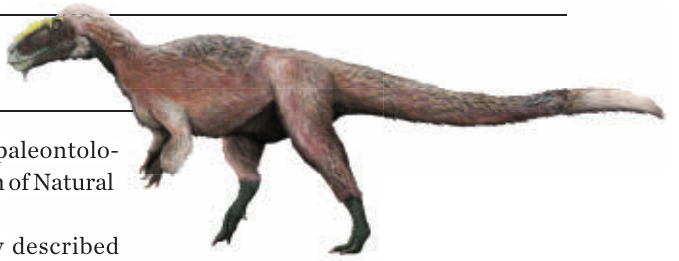
By comparing the homing success of dosed versus untreated hives, researchers concluded that pesticides roughly doubled the risk on any given day that a forager would not make it home. Such a population drop substantially weakens a colony, Henry says.

Ecotoxicologist David Fischer of Bayer CropScience, which markets imidacloprid products, questions the realism of the pesticide dosage. Researchers essentially fed bees all at one time the amount of pesticide they might encounter over a whole day, says Fischer, who is based in Research Triangle Park, N.C.

For common pesticides, Goulson says, "there are obviously big question marks as to whether the safety testing that was done on these was really adequate." Chronic effects may not show up without tests of free-ranging bees confronting real-world problems.

There's even less known about the multitude of pollinating bees that don't live in colonies, says entomologist Mace Vaughan in Portland, Ore., with the Xerces Society for Invertebrate Conservation. Most wild bees living around farms are solitary. "If an individual bee is lost, she cannot be replaced and her reproduction stops," he says.

Life



Yutyrannus **(artist's impression shown) is the largest feather-covered dinosaur species discovered to date.**

T. rex had a fine feathered cousin

Fossils of generously plumed dino found in northeast China

By Rebecca Cheung

From 125-million-year-old rocks, scientists have unearthed the remains of a new species of extensively feathered dinosaur that weighed up to about 1,400 kilograms and stretched 9 meters from nose to tail.

The fossils, from one adult and two juveniles, were found in northeast China in a region known for keeping soft tissues of ancient animals well-preserved, scientists report April 5 in *Nature*.

"It changes the way we really look at things — from these big, scaly, Jurassic Park animals to ones that were big and fluffy," says Mark Norell, a paleontologist at the American Museum of Natural History in New York City.

The biggest of the newly described creatures — the largest extensively feathered dino known to date — was about one-quarter the weight of *Tyrannosaurus rex*, its relative and fellow meat-eater. The new species is named *Yutyrannus huali*, which translates to "beautiful feathered tyrant."

The species' feathers were at least 15 centimeters long and appear to have covered the dinosaur's skin, which would have given it a shaggy appearance, says study coauthor Corwin Sullivan of the Institute of Vertebrate Paleontology and Paleoanthropology of the Chinese Academy of Sciences in Beijing. Still, the full extent of the plumage isn't known because the specimens aren't complete.

What the feathers were for remains unclear. They might have helped the dinosaurs show off and attract mates. Until now, all known full-feathered dinosaurs have been much smaller, and more likely to lose body heat because of their size. So these petite creatures may have used a fluffy layer to stay warm.

The newfound dinos also may have needed insulation, Sullivan says. But Norell isn't convinced. Many large animals that live in warm climates, such as modern giraffes, have external covering but don't need it for insulation, he says.

Yutyrannus had a high, bumpy nose plate, and Sullivan speculates that full-grown it stood about 2.5 meters tall.

BRIAN CHOO

Genes & Cells

For longer versions of these and other Genes & Cells stories, visit **www.sciencenews.org**

Inducing cells to make cartilage

Kartogenin triggers activity that could fight osteoarthritis

By Nathan Seppa

A small molecule called kartogenin encourages stem cells to take on the characteristics of cells that make cartilage, a new study shows. Treatment with kartogenin allowed many mice with arthritis-like cartilage damage in a knee to regain the ability to use the joint without pain.

The findings provide new clues in the long-running effort to find ways to regenerate cartilage, a central puzzle in the battle against osteoarthritis, researchers report online April 5 in *Science*.

The new approach taps into mesenchymal stem cells, which naturally reside in cartilage and give rise to cells that make connective tissue. These include chondrocytes, the only cells in the body that make cartilage. Kartogenin steers the stem cells to wake up and take on cartilage-making duties, an essential step in cartilage repair that falls behind in people with osteoarthritis.

"In the blue-sky scenario, this would be a locally delivered therapy that would target stem cells already there," says study coauthor Kristen Johnson, a molecular biologist at the Genomics Institute of the Novartis Research Foundation in San Diego.

Johnson and colleagues screened 22,000 compounds in cartilage and found that one, kartogenin, induced stem cells to take on the characteristics of chondrocytes. The molecule turned on genes that make cartilage components called aggrecan and type II collagen. Tests of mice with cartilage damage similar to osteoarthritis showed that kartogenin injections lowered levels of a protein called cartilage oligomeric matrix protein. People with osteoarthritis have an excess of that protein. Kartogenin enabled mice with knee injuries to regain weight-bearing capacity on the joint within 42 days.

Lab work revealed that kartogenin inhibits a protein called filamin A in the mesenchymal stem cells. This action unleashes other compounds that orchestrate the activity of genes that help turn stem cells into functional chondrocytes. In so doing, Johnson says, kartogenin seems to protect and repair cartilage.

Millions of people develop osteoarthritis as they reach old age. Surgery to clean out torn cartilage has limited success, as does surgery to induce growth of a fibrous kind of coating at the ends of bones that have lost their natural cartilage caps. This losing battle leaves bone-on-bone friction, inflammation and pain.

"Our cartilage wasn't meant to live this long," says molecular biologist Mary Goldring of Weill Cornell Medical College in New York. A cartilage imbalance results from wear and tear, literally, as people age, she says.

Suspect virus may counter lupus

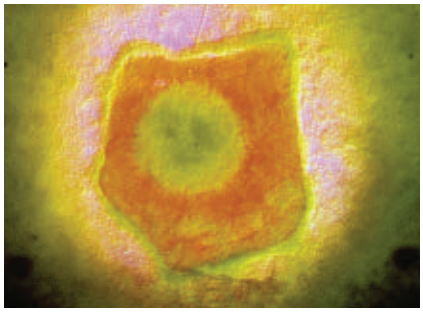
Mouse version of Epstein-Barr fights autoimmune disorder

By Rebecca Cheung

A version of the Epstein-Barr virus, a prime suspect in lupus, actually prevents certain features of that autoimmune disease, a study in mice shows.

"It might be that this virus has positive effects," says study author Roberta Pelanda of the National Jewish Health hospital and the University of Colorado Denver School of Medicine. "We really don't know what these chronic viruses do to the immune system." Pelanda and her colleagues describe the findings online April 2 in the *Proceedings of the National Academy of Sciences*.

In people with lupus, the immune system makes autoantibodies that attack the body, including the kidneys, heart, skin and blood. While the cause of lupus is poorly understood, some work suggests that the Epstein-Barr virus may trigger the disease in susceptible individuals.



The Epstein-Barr virus (shown in false-color) has been linked to lupus. In mice, a related virus appears protective.

UNITÉ DES VIRUS ONCOGENES/INSTITUT PASTEUR/PHOTO RESEARCHERS, INC.

A member of the herpesvirus family, Epstein-Barr is best known as the cause of mononucleosis. Infection with Epstein-Barr is extremely common, and in most people the virus remains dormant for much of a person's lifetime.

In the new work, scientists examined the link between Epstein-Barr and autoantibody production in mice. Because Epstein-Barr doesn't actually infect mice, Pelanda's team used a similar rodent virus, the murine gammaherpesvirus 68. The team found something unexpected: lupus-susceptible mice infected with the mouse virus tended to have healthier, higher-functioning kidneys than mice that had not been infected.

Though levels of autoantibodies rose initially after infection, after a year, lupus-prone female mice tended to make lower levels of autoantibodies compared with noninfected females.

These results are intriguing, says lupus researcher Laurence Morel of the University of Florida in Gainesville. But she cautions that what's seen in mice might not represent what goes on in humans.

45 percent | Average U.S. man's lifetime risk of any cancer

32–42 percent | Estimated cancer risk for a man following a negative genomic screen for cancer

DNA flunks disease predicting test

Genetic blueprint can foretell risk for only a few disorders

By Tina Hesman Saey

The human genetic instruction book is as lousy at predicting disease as an almanac is at predicting the weather, a prominent cancer researcher concludes based on data from identical twins.

Deciphering the genetic books, called genomes, is quicker and cheaper than ever. Scientists have touted the genome as a crystal ball for peering into people's medical futures. But Bert Vogelstein of Johns Hopkins University School of Medicine wondered just how informative a person's genetic makeup could be.

So Vogelstein and colleagues gathered medical data from 53,666 twin pairs from around the world. Identical twins share their genetic makeup, so looking at one twin's health history may reveal what medical complications the other twin's genome can foretell. The researchers did not decipher the twins' genomes but used the medical data to develop a mathematical formula to predict the minimum and maximum risk of getting 24 different diseases, including several cancers, heart disease, diabetes and Alzheimer's.

For all but four diseases, the genetic data would fail to determine who is likely to contract the condition in most cases, Vogelstein reported April 2. The results were also published online April 2 in *Science Translational Medicine*.

"Basically, you can still do better just by putting somebody on the scales and asking about their smoking history," says epidemiologist Walter Willett of the Harvard School of Public Health.

> **"Basically, you can still do better just by putting somebody on the scales and asking about their smoking history."**
> WALTER WILLETT

A test result was considered positive if it showed that a person has a 10 percent or greater chance of developing a particular disease. For most of the diseases, only a small fraction of people would get a positive result from a genome analysis, the researchers found.

For ovarian cancer, for example, only 1 percent to 23 percent of women who will eventually develop the cancer would get a positive result. So, most women who will get ovarian cancer would have received a negative test result.

That's because genetics are only part of the story when it comes to determining health. Lifestyle, environment and chance play a bigger role than genes, or work with genes, to influence disease.

For the small number of people who do get a positive test result, such information could be very important. "Even if the majority of individuals will receive negative test results, you don't know until you check," says George Church, a Harvard geneticist who founded the Personal Genome Project to relate genetic variation to individual traits. "It is analogous to fire insurance. You don't know in advance if you are in the majority who will not lose their house."

Genetic testing for four diseases — thyroid autoimmunity, type 1 diabetes, Alzheimer's and death from coronary heart disease in men — could predict 75 percent or more of patients who would get the disease. These diseases may have fewer genes as the underlying cause, or the genes may have a stronger effect than for other diseases, Vogelstein said.

Vogelstein said that he and his colleagues aren't making a value judgment about the usefulness of genome sequencing. "What we're trying to do is simply introduce a reality check," he said.

MEETING NOTES

Why brain cancer resists chemo

Some people with glioblastoma, a deadly brain cancer, have mutations in a gene called *EGFR*. Doctors hoped that drugs targeting *EGFR* could shrink tumors as the drugs do in lung cancer, but they didn't. The reason, said Ingo Mellinghoff of Memorial Sloan-Kettering Cancer Center in New York City, is that brain cancer patients have mutations in a different part of *EGFR* than lung cancer patients do. Glioblastoma mutations tend to affect a part of the EGFR protein that sticks out of the cell, whereas the lung cancer defects and drugs work on a part of the protein inside the cell, Mellinghoff said March 31. He and colleagues also describe the finding online March 31 in *Cancer Discovery*. —*Tina Hesman Saey*

Cancer fate tied to protein location

An old real estate axiom may also be a key to pancreatic cancer patients' fate. The location of a protein called survivin may influence whether pancreatic cancer cells are susceptible to chemotherapy after surgery, researchers led by Barbara Burtness of Fox Chase Cancer Center in Philadelphia reported April 1. Patients whose tumor cells harbored survivin in the nucleus lived longer without a recurrence of cancer than patients whose tumor cells contained the protein outside the nucleus. No difference was detected between the two groups after surgery alone, but surgery followed by radiation or chemotherapy kept tumors with survivin in the nucleus in check longer. The result supports previous research indicating that survivin's location can make cancer cells more or less vulnerable to chemotherapy. —*Tina Hesman Saey*

Body & Brain

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Scans foretell who will choke

Activity in key brain regions predicts math test success

By Laura Sanders

As any high school senior staring down the SAT knows, when the stakes are high, some test-takers choke. A new study finds that activity in distinct parts of the brain can predict whether a person will remain cool or crumble under pressure.

The results, presented April 1, may help scientists better understand how the brain copes with stressful situations, said psychologist Thomas Carr of Michigan State University in East Lansing. "Sometimes you come across a study you wish you'd done yourself," he said. "This is such a study."

Andrew Mattarella-Micke and Sian Beilock, both of the University of Chicago, had volunteers perform math problems, some easy, some hard, while undergoing a functional MRI scan. These two-step calculations were designed to tap into a person's working memory: Participants had to hold an intermediate number in mind to correctly calculate the final answer.

After volunteers had performed about 25 minutes of low-stakes math, they were told that their performance had been monitored, and if they improved, they would get $60 instead of $30. The researchers also added social pressure, telling volunteers that if they failed to improve, a teammate would lose money.

> "The more you engage these regions, the less you'll choke."
>
> ANDREW MATTARELLA-MICKE

Extra pressure didn't interfere with performance on the easy questions. On hard questions, accuracy dropped by about 10 percent on average. But individually, some people handled the pressure better than others.

Activity in brain regions that have been linked to working memory predicted choking, Mattarella-Micke said. In particular, higher activity in two regions — the intraparietal sulcus and the inferior frontal junction — meant that a person was less likely to crumble. "The more you engage these regions, the less you'll choke," Mattarella-Micke said.

A third brain area, the ventromedial prefrontal cortex, or vmPFC, often behaves in sync with these regions. The weaker the link between vmPFC activity and the other regions, the less likely a person would choke.

This result makes sense, said psychologist Florin Dolcos of the University of Illinois at Urbana-Champaign. Taking the vmPFC out of the equation might help people stay focused on the math. "I think this is actually a really interesting phenomenon, and I'm surprised we don't know more about it," Dolcos said.

Slightly different brain systems might be involved in athletic choking, where working memory might not be as important as brain processes that control motor activity, Mattarella-Micke said. ■

Jolt to brain aids language recovery

Electrical stimulation improves stroke patients' word recall

By Laura Sanders

A brain-zapping technique helps people recover language after a stroke, new research shows. The results may point to a better way for people to relearn how to talk after a brain injury.

"I think this work is very promising," said cognitive neuroscientist Roi Cohen Kadosh of the University of Oxford. The study, presented April 2, is one of the first attempts to apply brain stimulation methods to a clinical population, he said.

Speech therapist and neuroscientist Jenny Crinion of University College London and collaborators focused on people who had trouble finding the right word after a stroke. Known as anomia, the condition is frustrating, leaving people unable to call the correct word to mind.

Crinion and colleagues paired a word-training technique with brain stimulation. In the lab and at home, participants studied 150 cards with pictures of one-syllable words of everyday objects — cat, bed, car and so on — for a total of about 60 hours over six weeks.

Six volunteers received an electrical brain stimulus known as transcranial direct current stimulation three days a week while training on the words. Seven volunteers received a sham treatment.

Stimulation was targeted to Broca's area in the left side of the brain, a region linked to speech production.

Initial results look promising, Crinion said. All of the volunteers improved, because they all received the normal word-recognition training. But people who also received brain stimulation showed a startling improvement. At the end of the six-week study, the volunteers receiving stimulation nearly doubled their scores on a picture-naming task, improving by 92 percent. Volunteers without brain stimulation improved on average by 56 percent.

Three months out, volunteers who received the stimulation still performed 82 percent better than before treatment.

"These are huge effects," Crinion said. "It's really encouraging."

The team doesn't know whether the improvements will apply to other types of language use, such as carrying on conversations. But some of the patients have noticed improvements in their quality of life, Crinion said.

"What's interesting is that it seems to be the same structures that are involved in both [eating] extremes." —SUSAN CARNELL

Brain plays role in extreme eating

Same regions involved for obese and underweight people

By Laura Sanders

Certain brain areas are sluggish in people who eat too little and hyperactive in people who eat too much, a new study finds.

The results, presented April 3, are based on brain activity in people who ranged from dangerously thin to morbidly obese. The findings help clarify the complicated relationship between the brain and food, and may even offer ways to treat conditions such as anorexia and obesity, said study coauthor Laura Holsen of Harvard Medical School and Brigham and Women's Hospital.

One of five groups studied by the researchers consisted of people with anorexia. The other groups included people who formerly had anorexia but had recovered to a healthy weight, healthy people at a normal weight, and people who were obese. In the final group were people with a disorder called Prader-Willi syndrome, characterized by developmental delays, mental deficits and an insatiable appetite.

Holsen and her colleagues had participants come into the lab hungry and undergo fMRI brain scans while viewing pictures of food. After eating a meal, the volunteers underwent another scan.

While hungry, volunteers with anorexia had lower than normal activity in brain areas associated with the rewarding feelings that food usually elicits: The hypothalamus, amygdala and hippocampus all showed lackluster responses to pictures of food. At the other end of the spectrum, people with Prader-Willi had higher-than-normal activity in those brain regions. Volunteers in the middle three groups showed a similar trend, though less extreme.

After the meal, activity in another brain area — a part of the outer layer of the brain called the dorsolateral prefrontal cortex — revealed a big difference between groups. Activity there kicked on in most of the participants, except for those with Prader-Willi syndrome.

The results show that seemingly opposite eating disorders such as anorexia and extreme overeating that accompanies Prader-Willi syndrome may share some common underlying brain circuits. "What's interesting is that it seems to be the same structures that are involved in both extremes," said psychologist Susan Carnell of the New York Obesity Nutrition Research Center.

Body & Brain

6.7 per 1,000 | U.S. children with autism spectrum disorders, 2000 estimate

11.3 per 1,000 | U.S. children with autism spectrum disorders, 2008 estimate

Fatty diet triggers nerve cell birth

Extra neurons in brain linked to more pounds in mouse study

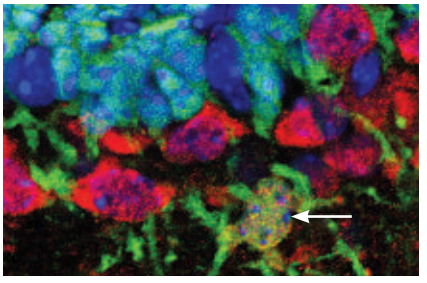
By Laura Sanders

Cheeseburgers pack on the pounds, but in mice a high-fat diet also packs on new nerve cells in the brain. More brain cells may seem like a good thing, but these newly sprouted cells appear to trigger weight gain, a new study finds.

If the same thing happens in humans, these nerve cells may be a target for anti-obesity treatments.

"This kind of work will definitely inform how we think about the underlying factors that relate to obesity," says endocrinologist Jeffrey Flier of Harvard Medical School in Boston. There's increasing interest, he says, in how long-term changes in brain circuitry — like new nerve cell production — affect eating and hunger. "That is going to be a very interesting frontier."

With some key exceptions, most regions in the adult brain don't make new nerve cells. But in a small sliver of brain tissue called the median eminence, new nerve cells are born throughout life, neuroscientist Seth Blackshaw of Johns Hopkins University School of Medicine and colleagues report online March 25 in *Nature Neuroscience*. The median eminence is part of the brain's metabolism hub known as the hypothalamus.



A special cell called a tanycyte (green) was caught giving birth to a new nerve cell (white arrow) in a brain region called the median eminence.

One signal to step up production in the median eminence, the team found, is a diet high in fat.

In the study, mice that ate the rodent version of a steady stream of Big Macs gained weight. This unhealthy diet also kicked nerve cell production into high gear. Adult mice eating a fatty diet for several weeks pumped out about four times as many new nerve cells in the median eminence as mice that ate regular chow.

To see whether these newborn nerve cells were up to no good, Blackshaw and his team shut down production with a carefully targeted laser. Even while continuing to gorge on a high-fat diet, these mice started moving around more and didn't gain as much weight as mice on a high-fat diet that could still make the new nerve cells. Take away the steady stream of new nerve cells, and the pounds didn't pile on as fast.

The newborn cells' parents turn out to be a kind of brain cell that resides in the median eminence. Both mice and people have these cells, called tanycytes. "There's been a lot of speculation about what their function may be," says Blackshaw.

The scientists don't yet know how these newborn nerve cells can influence metabolism. Other studies, including those by Flier, have found that a high-fat diet actually reduces nerve cell turnover in other parts of the hypothalamus.

Blackshaw warns that it's too soon to know if a similar thing is going on in people. "This is the very first step in trying to understand this process," he says.

U.S. autism rate continues to rise

Prevalence estimate hits new high at 1 in 88 children

By Bruce Bower

New federal data indicate that 1 in 88 U.S. children had autism or other autism spectrum disorders in 2008, up from 1 in 110 kids in 2006 and 1 in 150 in 2002.

Although that's a worrisome trend, the reasons for autism's rising prevalence — measured in nonrepresentative national samples of 8-year-olds — remain unclear. The Centers for Disease Control and Prevention in Atlanta released the latest autism figures on March 30.

CDC researchers used health and educational records to identify children with autism spectrum disorders among more than 38,000 kids in parts of 14 states.

"Such a big increase ... in such a short time seems a little odd, and there's a lot of noise in these data," says psychiatrist Fred Volkmar of the Yale Child Study Center in New Haven, Conn.

Some of the clatter stems from divergent diagnostic and record-keeping practices, Volkmar says. Some children with learning problems may get labeled with autism spectrum disorders to receive special education services, he notes.

Rates of such disorders fluctuated markedly state to state, the CDC reports. Prevalence ranged from 21.2 cases for every 1,000 children in Utah to 4.8 cases for every 1,000 kids in Alabama.

Overall, 1 in 54 boys, versus 1 in 252 girls, had autism spectrum disorders. The magnitude of that sex difference also varied substantially across states.

CDC's autism data show rate hikes among black and Hispanic children, reflecting corrections for underdiagnosis in minority populations, says anthropologist R. Richard Grinker of George Washington University in Washington, D.C.

Grinker says the new figures underestimate autism's prevalence, because they rely on school and medical records rather than in-person screening.

DANIEL LEE/BLACKSHAW LAB

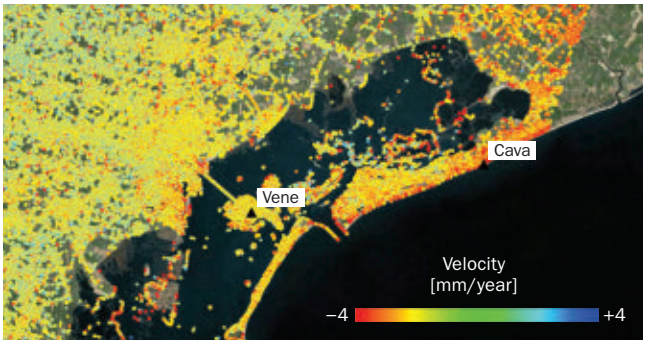
Earth

"It was thought that the sinking had ... stabilized, but now we know it will continue into the future indefinitely." —YEHUDA BOCK

Venice hasn't stopped sinking

Subsidence continues as soil compacts beneath city

By Devin Powell



Data from satellite radar systems and GPS devices reveal that Venice (section shown) sank at an average rate of 1 to 2 millimeters per year from 2000 to 2010, with some areas (red) subsiding even faster.

Venice is still sinking and will probably continue to do so for a long time, a new study suggests. That's bad news for the local government, which had already put a stop to groundwater pumping in an effort to curb the city's subsidence.

"It was thought that the sinking had pretty much been stabilized, but now we know it will continue into the future indefinitely," says Yehuda Bock, a geodesist at the Scripps Institution of Oceanography in La Jolla, Calif.

Today's sinking has little to do with human activities, Bock and colleagues report online March 28 in *Geochemistry, Geophysics, Geosystems*. Soil is compacting beneath Venice, bringing the city down. It rides atop a slab of Earth's crust that is slowly diving beneath the Apennine Mountains, giving the city a tilt noticed for the first time in the new study.

Data collected by GPS devices and satellite radar systems showed the city dropping an average of 1 to 2 millimeters per year from 2000 to 2010.

That's slower than it sank decades ago, says Bock, and slower than other cities such as New Orleans are sinking today. So some researchers don't think this natural subsidence is worth worrying about.

"We think this is a very small number," says Tazio Strozzi, a physicist with the remote-sensing company GAMMA in Gümligen, Switzerland. Previous satellite measurements have detected a similar amount of subsidence, he says.

Molecules

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Genes matter for truffle aroma

Geography not as important to a prized fungus' scent

By Rachel Ehrenberg

Mon dieu! A truffle's delectable aroma may be as much about genetics as it is about geography.

For years a truffle's flavor has been attributed mostly to environmental factors, akin to how *terroir* — the soil, climate and geology of a region — bestows qualities to wine. But a new analysis, published in the May *New Phytologist*, finds that a truffle's particular blend of chemical compounds is linked instead to its genetic background.

By casting light on what gives these elusive underground fungi their prized flavor, the study could help transform some truffles from species harvested in the wild to consistent crops.

"Truffles are a really valuable natural resource, and it's the aroma that really gives them their value," says Gregory Bonito of Duke University, an expert in truffle evolution.

But teasing out what gives a truffle its aromatic oomph has been tough, thanks in part to the complexity of the truffle lifestyle. Truffles are the fruiting body of a particular group of fungi. These fungi strike up partnerships with various tree species, so that the fungal spores germinate and grow into threadlike structures on the trees' roots. When fungal threads of different mating types find one another in the soil, sexual reproduction occurs and small truffles develop, 10 to 30 centimeters deep. The most prized species of the gastronomic delicacies may sell for thousands of dollars a pound.

Some enthusiasts attribute a truffle's aroma to its patch of soil, or whether spring was wet enough or winter cold enough. Another factor may be the tree species colonized. And truffles themselves are colonized by microbes thought to contribute to the warty lumps' flavor.

Just as yeast strains affect the flavor of wine, fungal genetics affect the aroma of Burgundy truffles, a new study finds.

Although the genetic blueprint for the famed black Périgord truffle (*Tuber melanosporum*) was published in 2010, the genetic particulars of other truffles aren't well understood. "We wanted to add the genetic dimension into the picture," says Richard Splivallo of the University of Göttingen in Germany, who led the new work.

So the researchers collected more than 200 Burgundy truffles from seven countries over four years. The scientists examined the profile of some of the 20 to 50 volatile chemicals that contribute to the truffle's delicate, nutty flavor.

When the researchers grouped the truffles by their genetic relatedness, truffles that were more closely related had more similar volatile profiles than more distantly related truffles.

"This is the first study to show a clear link between volatiles and genetics," says Claude Murat, who studies truffle genetics at the French National Institute for Agricultural Research in Nancy.

Smoking out clues from gun residue

Laser method identifies caliber of weapon without a bullet

By Rachel Ehrenberg

As forensic evidence goes, gunshot residue can be full of holes. But a new technique could provide a more definitive link between suspect and gun. A tool that employs lasers may allow investigators to match residue alone to a specific caliber of firearm, scientists report in two recent papers in *Analytical Chemistry*.

"Anything that's going to enhance or expedite the detection of gunshot residue and provide stronger evidentiary value is a way forward," says analytical chemist Jason Birkett of Liverpool John Moores University in England. "This work is very good and very novel and will do nothing but assist."

Along with the bullet, a cartridge or round also contains a propellant (such as gunpowder) and a primer. When the firing pin strikes the primer cap, the primer ignites, igniting the propellant and expelling the bullet from the gun. These reactions result in a spray of residue that can land on clothing, skin or anything nearby.

Gunshot residue analyses typically assess particles of barium and antimony (from the primer) and lead (from primer and bullet). Finding these particles indicates that a gun was fired and can help determine the distance the bullet traveled, but the approach is often not specific enough to link the residue to a gun of a particular caliber.

Now two teams have tackled gunshot residue with Raman spectroscopy, in which laser light of a specific wavelength induces vibrations in some of the residue molecules. Researchers in Spain analyzed spectra of six kinds of ammunition and compared these spectra with residue from fired guns, revealing a particular signature for each type of ammunition. These findings suggest that Raman spectroscopy could be used in situations where the bullet was not recovered or was too mangled to match to a weapon.

A team at the University of Albany in New York used Raman spectroscopy to distinguish residue from a 9 mm pistol from that of a .38-caliber revolver.

R. SPLIVALLO

Atom & Cosmos

"At this stage, we are all trying to fire bullets at the cosmological constant." —ADAM RIESS

Einstein passes acceleration test

Data support dark energy as cause of cosmic repulsion

By Elizabeth Quill

Einstein is still the boss, say researchers with the BOSS project for measuring key properties of the universe.

BOSS, for Baryon Oscillation Spectroscopic Survey, has measured the distance to faraway galaxies more precisely than ever before, mapping the universe as it existed 6 billion years ago. The findings, reported April 1, suggest that the mysterious "dark energy" causing the universe to expand at an accelerating rate was foreseen by Einstein.

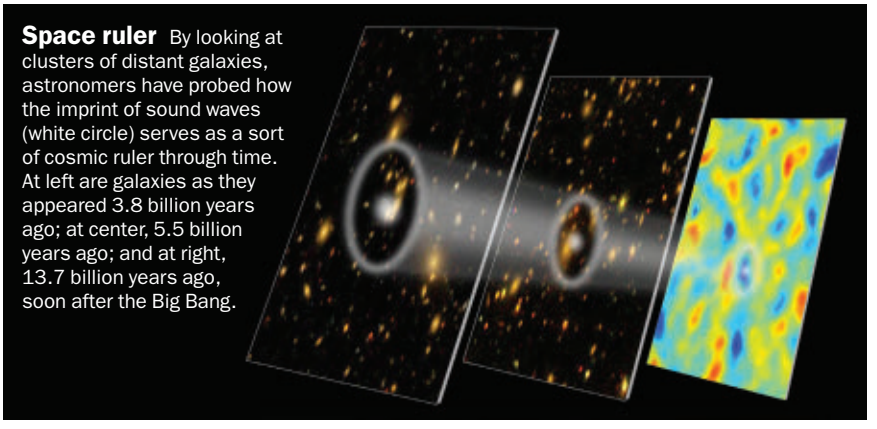
To keep the universe static, Einstein added a term called the "cosmological constant" to the equations for his theory of general relativity. In recent years, the cosmological constant, which describes a repulsive force occupying all of space, has been invoked to explain the discovery in 1998 that the universe is expanding faster and faster.

Evidence for accelerated expansion could be explained either by the negative pressure exerted by the cosmological constant (or some other form of dark energy) or by some flaw in general relativity. The BOSS results support the dark energy picture. "We find no deviations from general relativity on these very large scales," said Nikhil Padmanabhan, a physicist at Yale University who presented the results and is a coauthor on a series of papers reporting the findings online at arXiv.org.

BOSS, a part of the third Sloan Digital Sky Survey, analyzed about a quarter million galaxies. The key to the measurements is the imprint of sound waves — called baryon acoustic oscillations — frozen into the radiation from 300,000 years after the Big Bang, about 13.7 billion years ago. These sound waves caused regularly spaced bumps in the distribution of galaxies throughout the cosmos, and so can serve as a sort of space ruler.

This ruler has allowed the researchers to measure the distance back to an epoch when the universe was 63 percent of its current size, at 2,094 million parsecs plus or minus 34 million parsecs, a precision of 1.7 percent. (A parsec equals about 3.26 light-years.) The findings are consistent with dark energy as described by the cosmological constant.

"At this stage, we are all trying to fire bullets at the cosmological constant," said Adam Riess of Johns Hopkins University, who shared the Nobel Prize in physics last year for the discovery of the universe's accelerated expansion. "It is a pretty sharp bullet when you are making measurements that are more precise than ever before."



Space ruler By looking at clusters of distant galaxies, astronomers have probed how the imprint of sound waves (white circle) serves as a sort of cosmic ruler through time. At left are galaxies as they appeared 3.8 billion years ago; at center, 5.5 billion years ago; and at right, 13.7 billion years ago, soon after the Big Bang.

E.M. HUFF, SDSS-III, SOUTH POLE TELESCOPE, GRAPHIC BY ZOSIA ROSTOMIAN

MEETING NOTES

Newborn pulsars may explain iron-rich rays

Newly born pulsars might explain a weighty cosmic ray puzzle. Iron nuclei emitted from these dense cores left behind by supernova explosions could account for the composition of the highest-energy cosmic rays, Ke Fang of the University of Chicago reported April 2. Scientists had thought these cosmic particles were primarily protons. But observations at the Pierre Auger Observatory in Argentina have shown a surprising abundance of heavier iron nuclei in the rays (*SN: 7/18/09, p. 8*). Simulations by Fang and her team suggest that young pulsars could emit iron nuclei capable of escaping through the supernova's expanding envelope of matter that would block protons. Fewer than 0.01 percent of young pulsars outside the galaxy could emit enough iron to explain the Auger observations, Fang reported. The results are also described online at arXiv.org. —*Elizabeth Quill*

Mysterious neutron couplings

A beryllium nucleus packed with 12 neutrons has been spotted emitting two coupled neutrons, the first time such a neutron pair has been seen leaving a nucleus. The finding, from the MoNA collaboration, was presented March 31 by physicist Artemis Spyrou of Michigan State University. The result offers a new look into the forces that hold protons and neutrons together in atomic nuclei and may help scientists better understand astrophysical phenomena, such as the processes occurring in neutron stars. —*Elizabeth Quill*

Humans

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Ancient ancestor climbed, walked

Foot fossils unveil a species that lived near Lucy's kind

By Bruce Bower

An ancient member of the human evolutionary family has put what's left of a weird, gorilla-like foot forward to show that upright walking evolved along different paths in East Africa.

A 3.4-million-year-old partial fossil foot unearthed in Ethiopia comes from a previously unknown hominid species that deftly climbed trees but walked clumsily, anthropologist Yohannes Haile-Selassie of the Cleveland Museum of Natural History and colleagues report in the March 29 *Nature*.

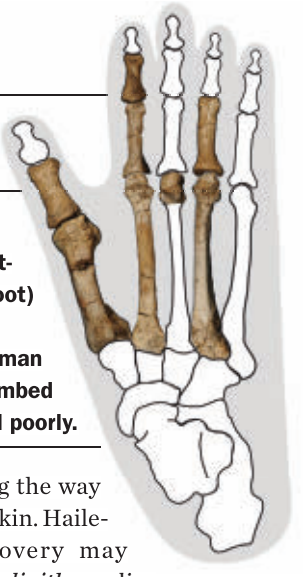
To the scientists' surprise, this creature lived at the same time and in the same region as *Australopithecus afarensis*, a hominid species best known for a partial skeleton dubbed Lucy.

"For the first time, we have evidence of another hominid lineage that lived at the same time as Lucy," says anthropologist and study coauthor Bruce Latimer of Case Western Reserve University in Cleveland. "This new find has a grasping big toe and no arch, suggesting [the species] couldn't walk great distances and spent a lot of time in the trees."

Lucy's flat-footed compatriot adds to limited evidence that some hominids retained feet designed for adept tree climbing several million years after the origin of an upright gait, writes Harvard University anthropologist Daniel Lieberman in a commentary also published in the March 29 *Nature*.

Armed with only eight foot bones, Haile-Selassie's team doesn't have enough evidence to assign a species name to the new find. But this hominid's foot shares more in common with 4.4-million-year-old *Ardipithecus ramidus*, largely known from a partial skeleton nicknamed Ardi, than with Lucy's crowd.

Ardipithecus evolved into an initial *Australopithecus* species by 4.1 million years ago, paving the way for Lucy and her kin. Haile-Selassie's discovery may come from an *Ardipithecus* lineage that survived near *A. afarensis* for hundreds of thousands of years before dying out, notes anthropologist C. Owen Lovejoy of Kent State University in Ohio.



Eight foot bones (shown in the outline of a gorilla foot) found in Ethiopia belonged to a human ancestor that climbed deftly but walked poorly.

As in gorillas, Ardi and the newly discovered hominid possessed short, curving big toes capable of grasping against the second toe. Other toe bones from Ardi and the new discovery formed joints that enabled a two-legged stride.

"Haile-Selassie's discovery highlights our lack of knowledge about hominid feet," says anthropologist Tim White of the University of California, Berkeley.

Stone Age fire rises from the ashes

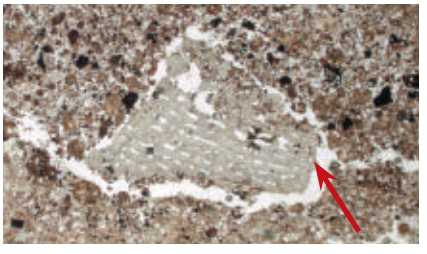
Early humans may have used controlled blazes for cooking

By Bruce Bower

A 1-million-year-old fire lit by human ancestors has flickered back to life in South Africa's Wonderwerk Cave.

Microscopic plant ashes and burned bone bits come from cave soil that previously yielded several dozen stone tools, say archaeologist Francesco Berna of Boston University and his colleagues. A member of the *Homo* genus, perhaps *Homo erectus*, made a fire that produced those remains, the researchers write online April 2 in the *Proceedings of the National Academy of Sciences*.

Berna's team says the finds provide the oldest secure evidence for controlled fire use. Although it's unclear exactly how members of an extinct *Homo* species used this fire, the findings fit with an idea that *H. erectus* began to cook food nearly 2 million years ago, the scientists propose.



A piece of charred bone a few millimeters across (indicated by arrow) from South Africa's Wonderwerk Cave offers evidence of an ancient controlled fire.

An age estimate for the Wonderwerk fire relies on measurements of radioactive elements in soil that signal how long ago dirt covered the burned material. Molecular characteristics of burned bone fragments show they were heated to about 500° Celsius, consistent with a controlled fire of some kind, the researchers say.

Several stone artifacts from the same ancient soil display fractures produced by heating during tool production.

Human ancestors were probably responsible for the Wonderwerk Cave fire, but fires were not produced regularly that far back in the Stone Age, say archaeologists Wil Roebroeks of Leiden University in the Netherlands and Paola Villa of the University of Colorado Boulder. Remains of a hearth or campfire area, where fires would repeatedly have been lit, have not turned up in Wonderwerk Cave.

FROM TOP: Y. HAILE-SELASSIE/CLEVELAND MUSEUM OF NATURAL HISTORY; PAUL GOLDBERG

Matter & Energy

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Proposed thermal cloak hides heat

Satellites, computers could withstand high temperatures

By Rebecca Cheung

A new type of "invisibility" cloak could take the heat off some devices. The theoretical cloak, which could shield an area from intense temperatures, is described online March 26 in *Optics Express*.

"You can just dress your satellite in a thermal cloak," says study author Sebastien Guenneau of the French National Center for Scientific Research and the University of Aix-Marseille. A heat cloak just 1 or 2 centimeters thick might potentially protect a satellite from overheating as it re-entered the atmosphere, he says.

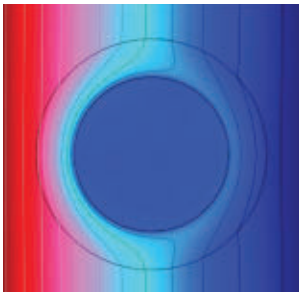
Until now, most invisibility cloaks have been made from metamaterials that guide light waves around a certain region of space, obscuring anything that lies within from sight. Other cloaks have been designed to distort the trajectory of sound waves.

In the new study, similar ideas were applied to bend heat as it disperses in space. In a two-dimensional cloak, most of the heat could be manipulated to move around a cloaked region 300 micrometers across, or the size of a typical amoeba.

Though heat isn't completely blocked, but only slowed, from dissipating into the cloaked area, Guenneau says there are potential applications for preventing materials from heating up too fast.

As computers are made on smaller scales, it's harder to incorporate fans or heat sinks to deal with heat, says Nicholas Fang, a professor in mechanical engineering at MIT. This paper "opens up new opportunities to dissipate the heat laterally, uniformly through all directions," he says.

Guenneau's team also describes another theoretical tool that uses metamaterials to direct heat within a specific region, which could be useful in building better solar panels. ■



S. GUENNEAU/INSTITUT FRESNEL, CNRS, AMU

With a constant 100° Celsius heat source coming from the left-hand side, the area contained inside a theoretical thermal cloak (center circle) remains cool relative to its surroundings.

Aliens in Antarctica

Visitors carry unwelcome species into a once pristine environment

By Devin Powell

Tourists and scientists are traveling to Antarctica more than ever. A new study quantifies the imported life they carry.

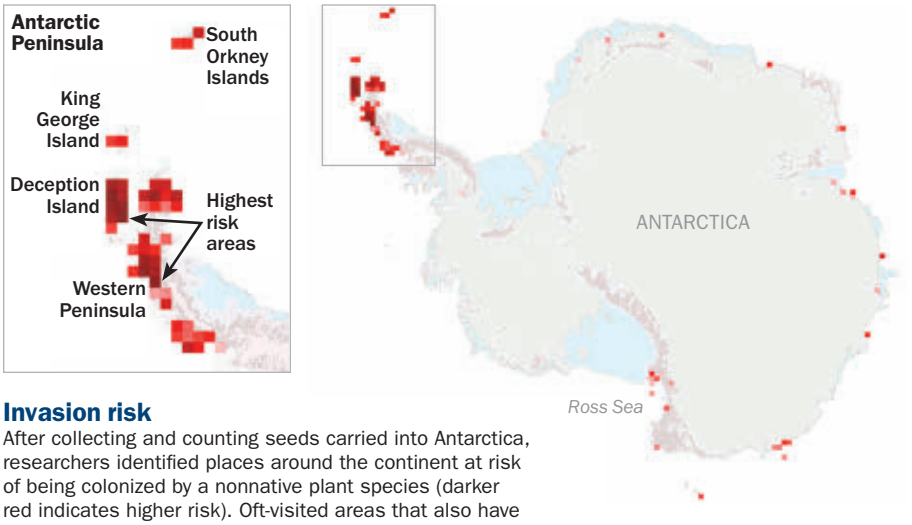
It was a summer day in January when Peter Convey pulled up a weed in Antarctica for the first time. The alien plant stuck out among the native species eking out an existence on the rocky debris beneath his feet.

Convey doesn't know for sure how the intruder, a rugged relative of the ornamental plant gerbera, traveled from its usual home 1,000 kilometers away in Tierra del Fuego. A seed may have drifted in on the wind or hitched a ride on the feather of a bird crossing the Southern Ocean. But Convey suspects that some human unwittingly delivered the species during fieldwork or while touring the remains of a whaling station nearby.

With Antarctica more trafficked by human feet than ever, scientists fear that Convey's pulled plant and others like it herald a coming invasion. In the same way stink bugs, Asian carp and kudzu have become abundant enough to alter ecosystems across North America, earning the name "invasives," species entering the Antarctic could multiply and spread. If so, nonnative plants — and even insects — may disrupt the most pristine landscape on Earth.

A handful of foreign creatures, including a particularly hardy kind of grass, have already shown that they're tough enough to put down roots at the end of the Earth.

"Several nonindigenous species have now gotten a toehold on the Antarctic continent," says Convey, a polar ecologist with the British Antarctic Survey in Cambridge, England. "Some of these species have already proven to be invasive in other places."



Invasion risk
After collecting and counting seeds carried into Antarctica, researchers identified places around the continent at risk of being colonized by a nonnative plant species (darker red indicates higher risk). Oft-visited areas that also have relatively mild climates may be ideal spots for an outside invader to take hold and flourish.

When the 50 countries signed on to the Antarctic Treaty meet in June, the issue of nonnative species will be on the agenda. Researchers will present findings on potential plant invaders from the first study that counts how many seeds enter Antarctica and identifies hot spots where the seeds could sprout.

Planting red flags

Worrying about immigration in a land mostly covered by ice may seem like a waste of time. But about a third of a percent of the continent is ice-free during the summer, an area almost the size of the Dominican Republic. Microbes, mosses, invertebrates and two species of flowering plant that can tolerate the inhospitable conditions call these bare patches of ground home. Having long lived in isolation, the creatures aren't used to competing with outsiders.

But isolation, at least from people, is no longer an option. Tourism brings about 33,000 people to the continent each year. Scientists and their support staff add another 7,000. More than 70,000 seeds cling to the shoes, clothing and bags of a single summer's worth of visitors, say researchers from eight countries who have collected, counted and identified thousands of stowaway seeds. The scientists, who reported the results in March in the *Proceedings of the National Academy of Sciences*, also checked the places where humans hang out for spots warm enough to nurture these unwelcome seeds.

About half of the tiny passengers come from cold climates to begin with, giving them an edge in Antarctica. Areas near the coast, where the climate is milder than in the continent's interior, could serve as landing sites, the new analysis reveals. The Antarctic Peninsula, jutting northward from the continent and home to many research stations, stood out as having an especially high risk of invasion. Red flags also popped up along the Ross Sea and the coast of East Antarctica.

Climate change will make Antarctica even more vulnerable in the coming years, particularly on the peninsula, the researchers say. The peninsula's western coast has warmed faster than most places on the planet — by about 2.5 degrees Celsius since the 1950s. Balmier temperatures raise the odds that would-be interlopers will survive and spread.

Safety precautions could help curb the influx of life, says polar ecologist Kim Crosbie, a coauthor on the new study who is with the International Association of Antarctica Tour Operators in Providence, R.I. Tourists tend to be clean visitors. Before setting foot on Antarctic soil, they are instructed to step in

Researcher Peter Convey pulled up this plant, a relative of an ornamental, in Antarctica. The species normally resides 1,000 kilometers away.

FACING PAGE: THOMAS PICKARD/GETTY IMAGES; FROM TOP: S. CHOWN *ET AL./PNAS* 2012; P. CONVEY

disinfectant, and their clothes are vacuumed. But scientists and the staff manning tourist expeditions are laxer, as revealed in the new seed counts.

"Everyone has a favorite jacket, and there's a tendency for staff to wear the same gear a lot and not clean it as much," Crosbie says.

Some areas flagged in the new study have already been infiltrated. An upcoming paper in *Conservation Biology* reports that an aggressive species of alien grass has appeared outside three scientific research stations on the Antarctic Peninsula.

Poa annua made its Antarctic debut in the 1980s, after having been brought — unintentionally — to a Polish outpost on King George Island. The weedy plant escaped and grew in patches on the island, which is close enough to the mainland to be considered part of Antarctica. Wind then carried seeds 1.5 kilometers away from the station, where the grass sprouted on debris left behind by a retreating glacier.

"It's starting to get to the point where this grass is beyond control in Antarctica," says Kevin Hughes, an environmental scientist with the British Antarctic Survey.

International law forbids the introduction of new species to Antarctica. But the rules are fuzzier about what steps should be taken to deal with species that have become established in the wild. Nothing has been done to get rid of the grass intruder, Hughes says.

What impact the plant will have on native life isn't yet known. But the same species has already overrun islands near — but not part of — Antarctica. On the island of South Georgia, the grass has replaced indigenous vegetation in some spots and stunts the growth of beetles that have trouble digesting the grass.

Tens of thousands of foreign seeds may make their way to Antarctica each summer, a recent analysis finds. Most of the potentially troublesome seeds collected as part of the analysis (some shown) were carried by scientists and expedition staff.

What's bugging Antarctica

Plants aren't the only potential invaders. Research stations have become beachheads for microbes, fungi, insects and worms. In 2005, construction vehicles brought by ship to a British outpost came with attached soil teeming with life. Produce imported to feed visitors can also carry infested dirt.

"About 12 percent of all fresh food items traveling to Antarctica are contaminated," says Dana Bergstrom, a polar ecologist with the Australian Antarctic Division in Kingston. Bergstrom and colleagues reported that statistic last year in *Biological Conservation* after examining more than 11,250 fruits and vegetables shipped to nine research stations.

Contaminated soil is supposed to be destroyed when discovered, but a few foreign agents have slipped through the defenses.

Flies made themselves at home in the sewage system of an Australian research station and in a British station's liquor store. Initial efforts to eradicate the insects failed at the Australian base. Their eggs endured, allowing the pests to spawn year after year. When reporting on the insects in 2005 in *Polar Biology*, Hughes and colleagues suggested that the bugs couldn't survive the bitter cold outside, meaning they probably wouldn't spread into the wild.

But another type of foreign fly with long legs has been sighted on King George Island. No one knows for sure how far *Trichocera maculipennis* has spread or how to get rid of it. Other interlopers now enjoying the fresh air include a worm and a species of midge that has multiplied in recent years.

With critters from abroad swooping in by land, by sea and by air, there's good reason to put caution first, says Mahlon "Chuck" Kennicutt II, an oceanographer at Texas A&M University in College Station and president of the Scientific Committee on Antarctic Research. Antarctica, he says, offers a natural laboratory like no other on Earth. Its largely untouched environment helps scientists understand not only the rugged flora and fauna that can live in such habitats but also the planetwide effects of climate change.

"Antarctica is one of our last true wilderness areas," says Kennicutt. "There are very few of those places left in the world. We'd like to keep them protected." ■

This fly, *Trichocera maculipennis*, has been seen in a new home — on King George Island, off Antarctica's coast.

Explore more

- Learn about the wildlife inhabiting Antarctica at the British Antarctic Survey's website: www.antarctica.ac.uk/about_antarctica/wildlife

FROM TOP: D. BERGSTROM; ODILE VOLONTARIO

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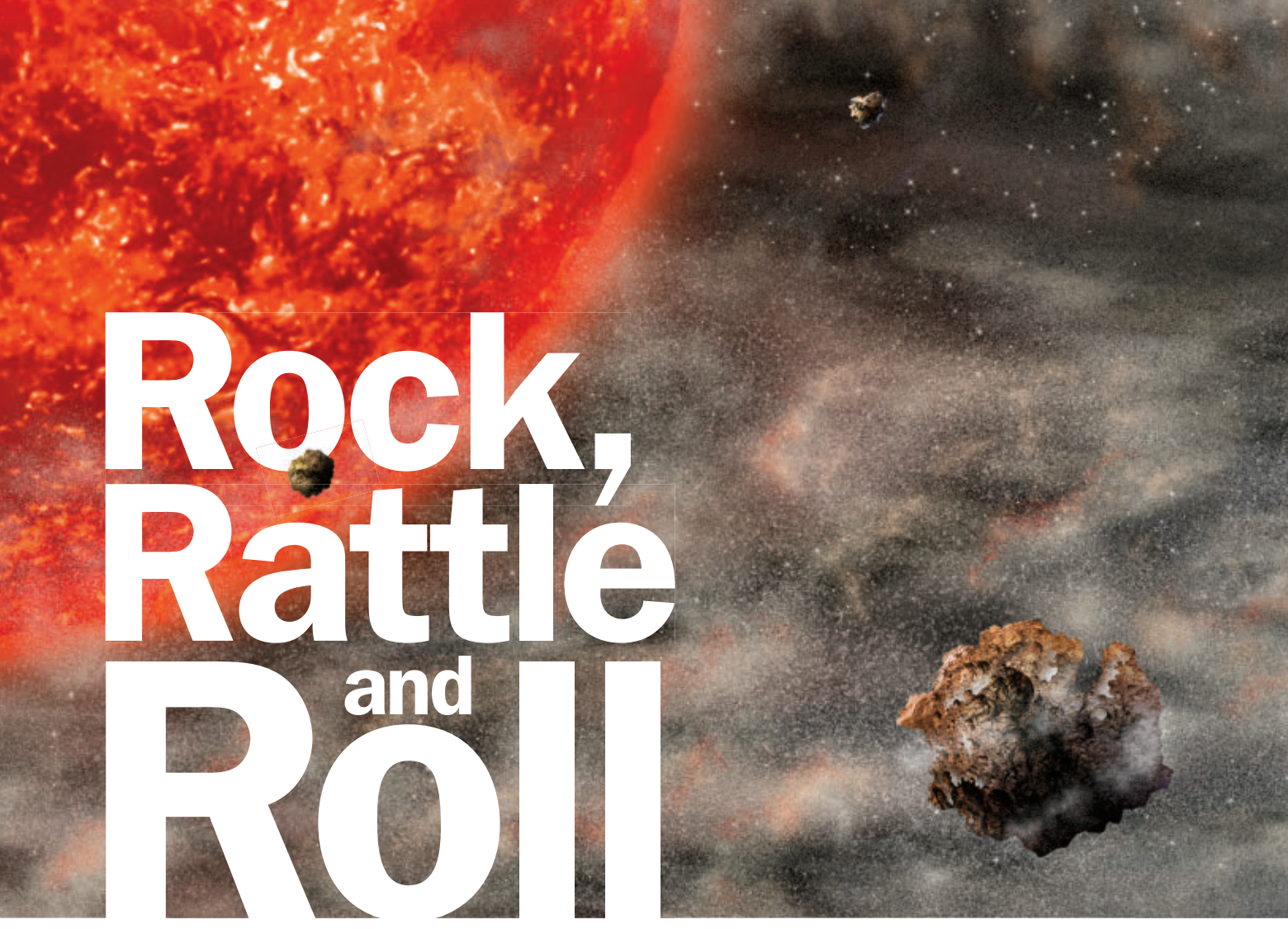


Rock, Rattle and Roll

Planetary scientists seek to fill in gaps in outer solar system's formative years

By Nadia Drake

Most scientists don't wear protective headgear while giving talks. Then again, most scientists aren't the target of pastry-wielding colleagues.

But last October, when astronomer Hal Levison presented what he called a "slightly radical" mechanism for building the solar system's giant planets, he was ready.

"I'm really a little intimidated about the reaction to this," Levison said, reaching behind the podium and retrieving a baseball catcher's mask, which he donned during his presentation, at a planetary science meeting in France.

Good thing, because at least one scientist in the audience was amused enough to deliver a good-natured French pastry toss.

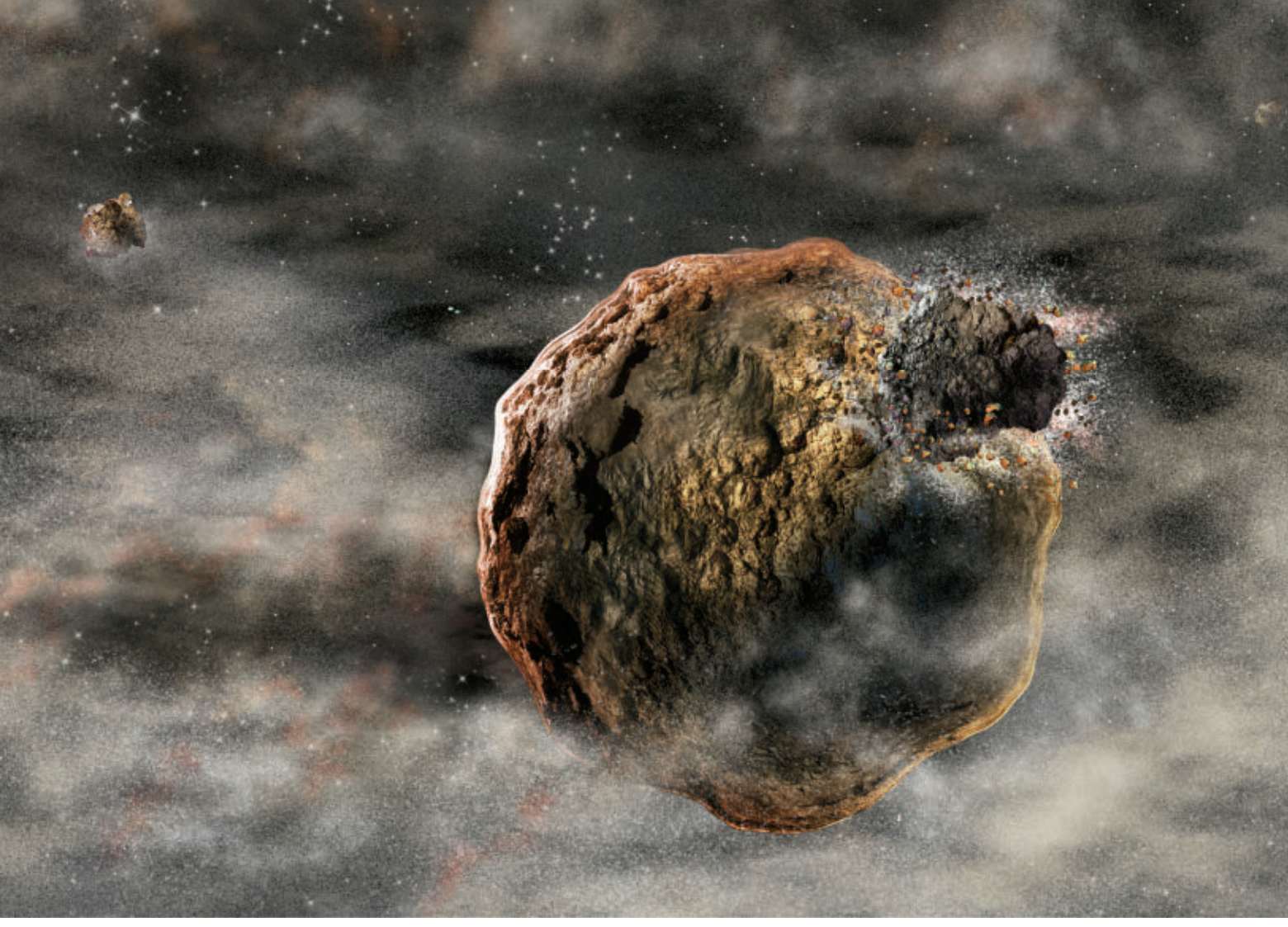
Levison, of the Southwest Research Institute in Boulder, Colo., proposed that the giant planets — Jupiter, Saturn, Uranus and Neptune — began forming near the distance at which the Earth currently orbits the sun. One by one, he said, the planet cores grew and shot outward, increasing in size like tumbling snowballs gathering material. Standard ideas describe a much more sedate embryonic environment, with the outer planets growing up simultaneously starting just after the birth of the solar system, roughly 4.5 billion years ago.

Levison's scenario is one of several recent proposals that seek to color in some gaps in the outer solar system's past. Some of the proposals complement a widely accepted theory that describes a good chunk of the solar system's formative years. Other ideas attempt to deal with some apparent snags in the theory, called the Nice model.

Named for the city in France where researchers pulled it together, the Nice story begins once the four giants are fully grown and plants them much closer together. Early chapters include a few million years of the giant siblings jostling and poking one another. The tale's middle is dominated by a period of extreme turbulence that eventually leads to a cataclysmic rearrangement of the solar system — a dissonance that resolves itself by the system's billionth birthday, with the giant planets moving into the orbits occupied today.

Though this story explains many observations about the solar system, it doesn't answer all the questions, such

NICOLLE RAGER FULLER



The giant planets may have tumbled outward from the sun during their formation, growing like rolling snowballs as material was collected along the way.

as what happened before the tale begins, how the terrestrial planets survived and why Mars is so small. Recent proposals, including Levison's tumbling planets idea and a second proposal from another Nice architect, are like a prologue, taking place in the roughly 5 million years after the birth of the solar system, long before the Nice model kicks off. Another tweak seeks to keep the Earth safe from the model's mid-tale turbulence by introducing a fifth giant planet — a long-lost sibling now wandering through interstellar space.

A Nice story

Reconstructions of the solar system's early years may benefit from observations of exoplanet systems, evidence contained in asteroids and other small bodies, and future spacecraft visits to the giant planets. But most studies probe this early epoch using the laws of physics to simulate the evolution of planetary orbits, given various starting conditions. "One thing that's nice about this branch of astronomy is that the physics are understood," says astronomer Greg Laughlin of the University of California, Santa Cruz, who studies how planetary systems form and evolve. "These guys are all just pulling at each other and interacting with Newtonian gravity."

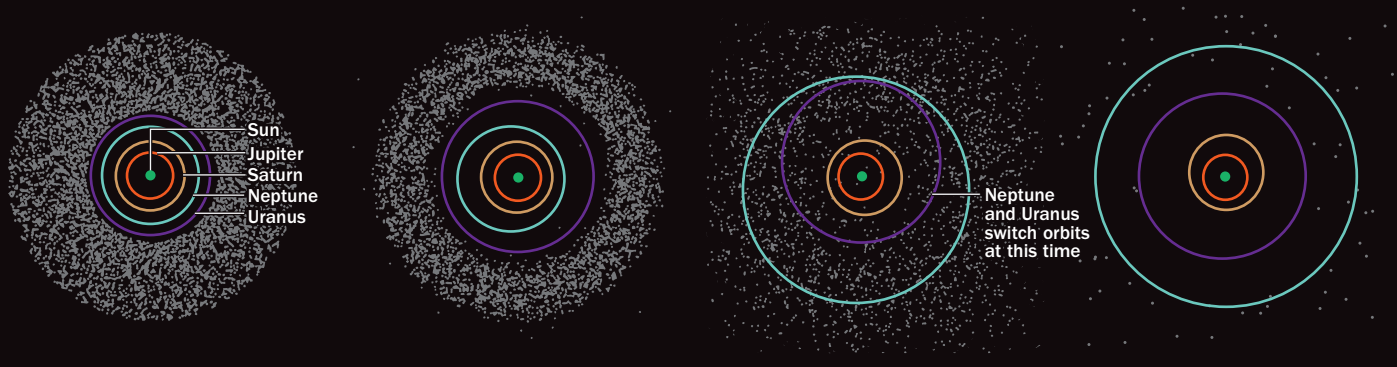
Scientists tend to accept a simulated scenario for the early solar system when the most common outcome matches the current planetary configuration. The Nice model — first reported by Levison and his colleagues in 2005 — does that well. It starts with the four giants in a compact configuration between 5.45 times the Earth's current orbiting distance from the sun, known as an astronomical unit, and 17 times Earth's orbiting distance.

After several million years, Jupiter's and Saturn's orbits evolve into a configuration that upends the adolescent solar system, flinging small bodies inward and scattering Uranus and Neptune out to their current distances. "Things start going all over the place," says Konstantin Batygin, a graduate student at Caltech who is basing his work on the Nice model.

Neptune ends up where it is now, out at 30 astronomical units. The material hurled toward the inner solar system explains what's known as the Late Heavy Bombardment, a period dominated by flying comets and asteroids and recorded in cratered scars on bodies such as the moon (*SN: 2/14/09, p. 26*).

Only after the solar system had been around for about 900 million years did

Arranging giants A well-accepted story of outer solar system evolution, called the Nice model, predicts many of the features seen in the solar system today. But some scientists, including those who proposed the model to begin with, have recently suggested some add-ons and tweaks.



4.5 billion years ago
Less than 5 million years after the solar system's birth, the outer planets are packed together following nearly circular orbits within a large disk of icy debris.

4.1 bya
The model kicks off when, as the planets spread out, Jupiter's and Saturn's orbits fall into lockstep. This alters the orbits of Neptune and Uranus; in some cases the two switch orbits.

4.1 bya–3.8 bya
Uranus and Neptune ram into the icy reservoir, sending debris into the inner solar system. This debris collides with the Earth and other planets during a period known as the Late Heavy Bombardment.

3.5 bya
The outer planets settle into their current arrangement, reproducing the configuration known today. By this point, the solar system has become a more sedate place.

its inhabitants settle into their current locations. The model ends with the planets achieving this stable configuration.

The Nice model also explains the Trojan asteroids orbiting near Jupiter and Neptune, swarms of outer solar system moons in irregular orbits and the icy Kuiper belt, a circle of small, frozen bodies that live beyond the orbit of Neptune.

"This is the only game in town," says planetary scientist Bill Bottke of the Southwest Research Institute. "There's no competing model that's even close to doing any of this."

Tumbling planets

But the Nice model doesn't address a crucial element: how to build planetary embryos, the solid seeds that form the cores of planets. Researchers just plunk in finished planets, Levison says of the model's starting point. "Everybody does this, and they all say this is clearly wrong," he says. "What happens when you try to do it right?"

Doing it right — that is, trying to capture the whole picture — includes starting ingredients called planetesimals. These asteroid-sized chunks of material float within the protoplanetary disk, knocking into each other and occasionally forming bigger chunks. When those chunks grow to embryos with 10 Earth masses, they begin to wrangle gas from the disk, eventually producing giant planets through what's known as the "core accretion" process.

Most scientists think that cooking a giant planet begins this way, from the inside out. But some say that growing a giant planet in less than 5 million years — before the gassy disk is known to have disappeared — is problematic.

A seed that starts developing in the thick of the disk, around where Jupiter sits when the Nice model picks up, can become a giant planet relatively quickly, in as little as 3 million years, says planetary scientist David Stevenson of Caltech. Still, the process will take much longer in the outer, colder parts of the disk where Uranus and Neptune live. "You run out of time," Stevenson says.

Levison's tumbleplanet theory can solve this timescale problem and get the planets where they need to be before the Nice model begins. Planet embryos forming at one astronomical unit from the sun and moving outward through the disk "grow like gangbusters," he says, noting that in his still-preliminary calculations, a lunar-sized seed can become a Neptune in about 800,000 years.

Among the more radical implications is that Neptune is the solar system's most senior citizen, moving outward first. And, contrary to many theories, Jupiter and Saturn did not form anywhere near their current locations.

"The general idea is really quite a way removed from what you will see in textbooks," Stevenson says.

Rolling planets do introduce some issues, such as how to stop the embryos from migrating beyond the point where the Nice model gets going. Levison and his colleagues are working on solving these issues and developing a full simulation for the idea, but they admit that they don't have all the answers yet. "I may end up being wrong," Levison says.

Wandering Jupiter

Levison isn't the only Nice model architect fiddling with a prologue to the main act. Planetary scientist Alessandro Morbidelli of the Observatoire de la Côte d'Azur in France, who helped construct the model, suggests that the nearly full-grown Jupiter made a little trek inward before settling into its Nice model starting position, 3 or 4 million years after the solar system's start.

ADAPTED FROM GOMES *ET AL*./*NATURE* 2005

In fact, Jupiter sidled all the way up to where Mars is now, about 1.5 astronomical units from the sun, according to this idea, reported last year in *Nature*. A big planet like Jupiter would sweep material from the disk at that location, with the leftovers eventually forming Mars. Such housecleaning could explain why Mars is smaller than researchers would expect.

"It explains why, among the terrestrial planets, there is a big difference between the Earth and Mars in terms of mass," Morbidelli says of the theory, which he calls the Grand Tack.

And the Grand Tack explains more than just a tiny Mars. Jupiter's inward wandering would have scattered the busted-up chunks sitting in a then-embryonic asteroid belt. When it moved back out, Jupiter would reassemble the rocky swath and drag in a second population of objects from farther away. The product? An asteroid belt between Mars and Jupiter with two distinct types of space rocks, a feature observed today but not explained by the Nice model.

Morbidelli's next step is to rewind the tape even further and really tackle the process of cooking a giant planet. "The Grand Tack makes some assumptions about where the planets are, which sequence they formed in, and we are not sure these assumptions are correct," he says. Morbidelli will also help test Levison's tumbleplanets idea, which may explain the small size of Mars via a different recipe than the Grand Tack (*SN Online: 10/4/10*).

Planetboot

Some parts of the main act have turned out to be a challenge for the Nice model, too. Over time, its architects discovered a complication. "We ran into a roadblock having to do with the survivability of the Earth," Levison says. "It ends up hitting Venus, mostly."

An evil encounter with Jupiter's gravity during the solar system's period of rebellion, when planets and asteroids are flying all over the place, sends Earth careening into its sister planet — an undesirable outcome for a model purporting to explain the current state of planetary affairs.

Scientists including David Nesvorny of the Southwest Research Institute proposed that the best way to save Earth was to offer an ice giant — Neptune or Uranus — to the Jovian bully. A larger planet interacting with Jupiter alters Jupiter's orbit almost instantaneously, causing it to skip over the deadly encounter with Earth.

But when Nesvorny simulated the interaction, he found that Jupiter and Saturn usually expelled the ice giant, leaving behind three giant planets instead of the four currently found in the outer solar system. So he added a fifth giant planet to the young solar system — a sibling who enters a deadly dance with Jupiter, spurring its own ejection and perturbing Jupiter's and Saturn's orbits.

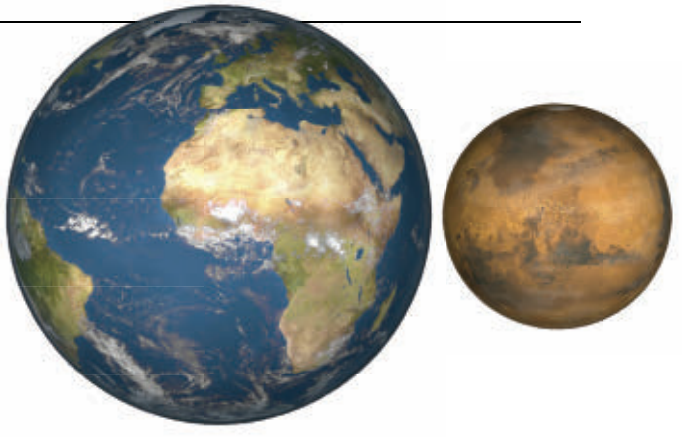
Simulations suggest that this ejected fifth planet preserves the solar system observed today when it resides for a short time between Saturn and the ice giants, Nesvorny reported in December in *Astrophysical Journal Letters*. "I'm able to match the current solar system, starting with five planets, up to 10 times more frequently than starting with just four planets," Nesvorny says. According to him, a young solar system populated by five giant planets is much more likely than one populated with just four.

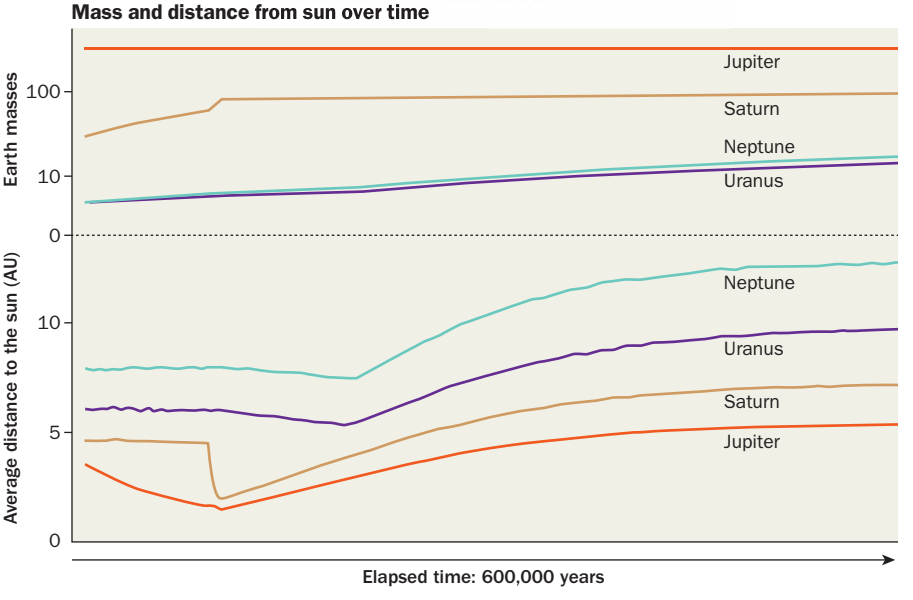
"Five-planet systems seem to work better," Bottke says. "But is there a way we can find a smoking gun telling us that this must be true?"

Batygin, who has also tried saving Earth with what he calls a "sacrificial goat," thinks such a find is unlikely, since an ejected planet probably wouldn't

Jupiter's journey

A few million years after the solar system was born, a young Jupiter may have journeyed inward, with an early Saturn following (chart shows masses and distances of outer planets from the sun over time). This trip could explain Mars' small size (far right, in comparison with Earth); as Jupiter moved, it swept away much of the material that would have otherwise come together to form Mars.



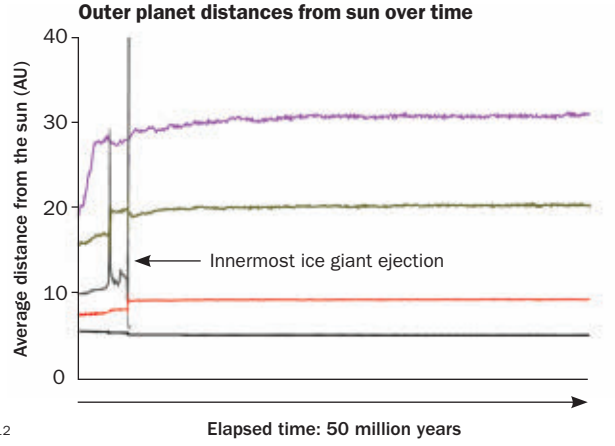


SOURCE: K.J. WALSH *ET AL*/*NATURE* 2011

WALTER MYERS/PHOTO RESEARCHERS, INC.

Bye-bye giant

After finding that a gravitational interaction with Jupiter could cause a young Earth to crash into Venus, researchers decided to offer Jupiter a separate partner. Models suggest the ejection of an extra ice giant (gray) could help the Earth survive. The extra giant also helps explain Jupiter's and Saturn's current orbits.



SOURCE: K. BATYGIN *ET AL*/ *ASTROPHYSICAL JOURNAL LETTERS* 2012

carry a detectable signature. In his study, published in *Astrophysical Journal Letters* in January, Batygin found that the fifth giant wasn't as necessary to save Earth as Nesvorny reports. "I'd say 50-50," Batygin says, of the likelihood of a fifth giant being required to save Earth in his model.

Various differences in the scientists' starting conditions — such as the amount of material the planets are swimming through — might produce the different outcomes. Even if the outcast isn't necessary to solve the Earth problem, Batygin thinks an extra giant would answer some important questions. "Without a close encounter, Jupiter's and Saturn's orbits very rarely end up being what they are today," Batygin says. "The reason for this fifth planet is to feed the big guys so they can toss it out and be happy."

As for that lost planet, Batygin doesn't know where it is today: "I'd like to say in a galaxy far, far away."

Exo-observations

Scientists observing faraway planetary systems around other stars are turning up data that may fill in the picture of how giant planets are made. "The extrasolar planetary systems are telling us that the basic idea of core accretion is consistent with our understanding," Laughlin says. Uranus- and Neptune-sized bodies are also turning out to be common, suggesting that they must be easy to make and providing an important clue about how outer planets grow.

Ultimately, the Nice model and scientists' other ideas will need to work for even Earth's most distant neighbors. Any story that seeks to explain how the planets are made should explain the ranges of sizes and compositions observed here and elsewhere. It is "dangerous to compare all these models with the solar system only," Nesvorny says.

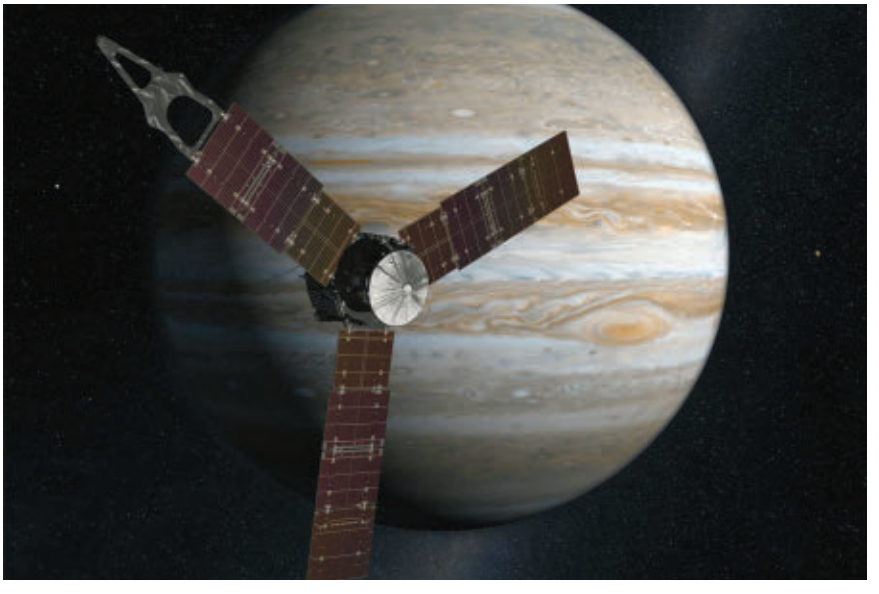
Observed exo-architectures have hinted at the ubiquity of planetary reorganizing, demonstrating that sibling scuffles are not hard to provoke. But the planetary configuration of Earth's nearest and dearest is still somewhat mysterious. So far, "It's not an enormously common outcome to have a Jupiter-mass planet in a Jupiter orbit," Laughlin says. But he notes, "We're just on the threshold of being able to reliably detect true Jupiter analogs around other stars."

Until more exodata come down from the sky, there are other ways of lending concrete support to simulations' suggestions. Levison's tumbleplanet idea makes some testable predictions. One is about the size of planet cores — something that NASA's Juno spacecraft could help determine when it arrives at Jupiter in 2016. Also, Levison predicts that cores will increase in size with distance from the Sun: Neptune's and Uranus' should be larger than Jupiter's. While it is unlikely to get off the ground anytime soon, a mission to Uranus recently prioritized by the Planetary Sciences Decadal Survey would be able to speak to this prediction.

Though the full recipe for cooking up an outer solar system might one day be written, Laughlin isn't convinced that humankind will ever really know what happened during the solar system's youth. The models contain quite a bit of truth, he says, but in the end, they're just models. Still, "there's no alternative that does a dramatically better job," he says. "The world may never know." ■

Explore more

■ For more on the outer solar system: http://1.usa.gov/outer_solar



The solar-powered Juno spacecraft (depicted in this artist's illustration) launched in August 2011. It is expected to reach and study Jupiter in 2016.

FROM TOP: ADAPTED WITH PERMISSION OF THE AAS; NASA

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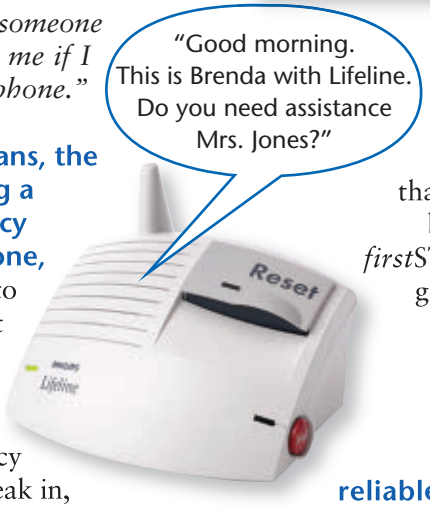

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Another Side to Statins

Heart-healthy drugs show promise against inflammation, cancer and the flu

By Nathan Seppa

It's been quite a ride for statins. Lipitor, Zocor, Crestor and their relatives are fast becoming household names. These cholesterol-lowering drugs have delivered a body blow to heart disease and stroke, two of the top four killers in Western society.

All success comes with a price: Some scientists express concerns over the widespread use and possible overprescription of statins. Still, there's no denying the evidence that they have cardiovascular benefits in many people. The drugs' track record has earned high praise for the research team that first devised them — and even higher rewards for the companies cranking them out.

Just when it seems that business can't get any better, another upside to statins is now emerging. Research suggests that the drugs have potent effects against diseases they weren't designed to fight. Statins might make people less susceptible to fatal pneumonia, better able to survive a head injury and more apt to dodge a nasty case of the flu.

No one is dancing in the streets just yet. These and many other findings arise from studies that track comparable populations but don't randomly assign participants to one treatment or another. While useful for picking up trends, such population analyses can't always be trusted, says Steven Lipkin, a physician and geneticist at Weill Cornell Medical College in New York City. These studies have been compared to economic models that have predicted "10 of the last four recessions," he says. Evidence is indirect and can be misleading.

Recently, though, laboratory research by Lipkin and others is adding weight to population findings. Cell-based work shows that aside from slowing cholesterol production, statins derail compounds that exacerbate inflammation — an all-around pain in the neck, and elsewhere. What's more, randomized trials in which people get statins or dummy pills, the gold standard for testing drugs, have shown a knockdown of rheumatoid arthritis and improvements in other immune-related ailments. Such findings are boosting enthusiasm among some researchers.

When statins first hit pharmacy shelves in the late 1980s, the tone was decidedly different. Many scientists worried that the drugs might cause cancer or have other unforgivable side effects. The discussion has come full circle now as scientists seek to sort out whether the drugs might *treat* cancer.

"It's definitely heartening. It's exciting," says Carol Prives, a molecular biologist at Columbia University. "But it's a large jump from [laboratory] data to any clinical changes."

Tackling inflammation

Statins have already made one giant leap, from the early days of drug development to today's broadening interest. Japanese biochemist Akira Endo and his colleagues devised the first rudimentary statin in the 1970s, building on earlier work that had deciphered how the body makes cholesterol. His team screened thousands of fungal components to see if any of them bogged down cholesterol manufacture in cells. The painstaking effort unearthed a compound that reduced LDL — the "bad" form of cholesterol. The compound works by neutralizing an enzyme called HMGCR (for 3-hydroxy-3-methylglutaryl coenzyme-A reductase), halting the chain reaction that leads to cholesterol production inside cells. Scientists at Merck later devised their own version of this first statin, and the rest is Big Pharma history.

But over time it became clear that lowering cholesterol was only part of the story. Although some patients still got heart attacks, statins consistently overachieved: The drugs prevented more heart attacks than would be attributable to cholesterol alone. Paul Ridker, a cardiologist at the Harvard School of Public Health and Brigham and Women's Hospital in Boston, thought statins' success might speak to a central puzzle

ICONS: DTERMINAL/ISTOCKPHOTO, ADAPTED BY E. FELICIANO

of heart disease: At least 15 to 20 percent of people who suffer a heart attack lack any apparent risk factor for the Big One, such as high cholesterol, hypertension, diabetes or a smoking habit.

Some researchers had already speculated that the hidden culprit might be chronic inflammation in the body, since plaques in the heart's coronary arteries contain inflammatory immune cells. Meanwhile, lab work and studies looking at statins' effects on the heart had suggested the drugs played a role against inflammation.

Ridker and colleagues decided to actually tackle the question: Did statins help prevent heart disease by reducing inflammation? His team assigned healthy people with normal LDL but elevated inflammation to get either Crestor or a placebo. The five-year trial was stopped early when it became clear that the statin recipients were having fewer heart attacks and strokes, the researchers reported in 2008 in the *New England Journal of Medicine*.

The following year, a second analysis of the data showed that roughly twice as many people getting placebos as receiving Crestor had experienced an episode of deep-vein thrombosis — a blood clot, often in the leg. Because clotting in veins is linked to inflammation and not plaque formation, the researchers concluded that statins' anticlotting effects in this case were independent of its benefits against heart attacks.

"Statins are twofers," Ridker says. "They both lower cholesterol and seem to inhibit inflammation."

Suddenly earlier studies hinting at an anti-inflammatory role for statins took on a new gloss, as did work by scientists who, in relative obscurity, had been looking into statins' effects on infections, trauma and other inflammation-related assaults on the body.

Multipronged switch

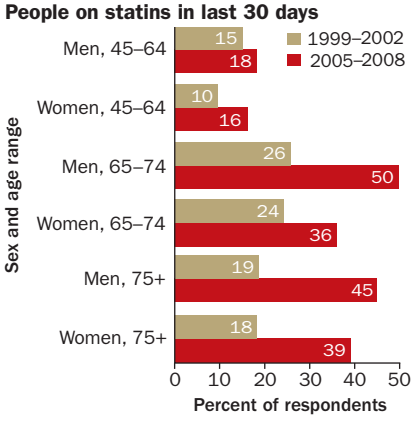
A steady stream of lab studies have since solidified the scientific basis for this inflammation-fighting effect. By attaching to HMGCR, statins flip a switch that does more than reduce cholesterol. The flip also knocks down production of compounds known as isoprenoids. Data now show that the knockdown influences basic immune function in a host of ways — a largely unexpected discovery.

"The whole history of science is filled with serendipity," Lipkin says.

James Liao, a vascular biologist and cardiologist at Harvard and Brigham and Women's Hospital, and others have tracked the fallout of statins' other career in recent years. The researchers have found that inhibiting isoprenoids prevents some immune cells from secreting inflammatory proteins and hampers other cells' ability to exacerbate existing inflammation. Statins even seem to tilt some immune cells away from an inflammatory bent toward a more quiescent role, and provide a bonus by maintaining good health in the all-important cells lining the insides of blood vessels. Such findings "have put into question whether cholesterol was entirely, or even mainly, the benefit of statin therapy," Liao says.

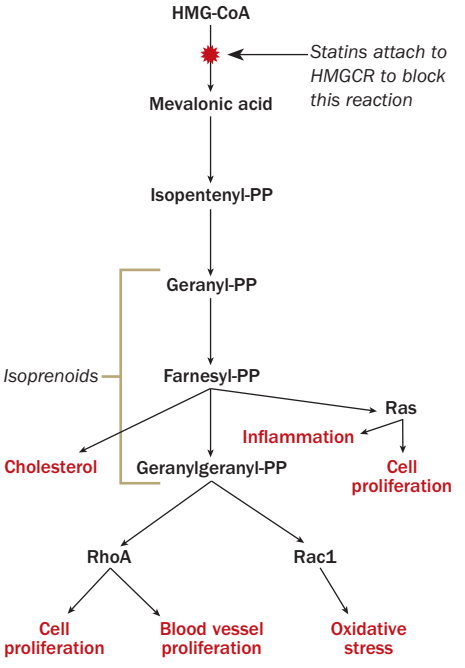
Make no mistake, inflammation is a helpful response to infection or injury and has served humans well over time, particularly in the rough-and-tumble Stone Age. But inflammatory cells and proteins can overstay their welcome, damaging healthy tissues in blood vessels, swollen joints and infected lungs.

Upped use The percentage of people in the United States reportedly taking statins has increased among most age groups in recent years. While some researchers are concerned about overprescription, others attribute the boost to the drugs' successful track record.



SOURCE: NCHS/CDC

Forked effects When statins attach to an enzyme called HMGCR, they prevent HMG-CoA from becoming mevalonic acid. This hinders a cholesterol-making reaction, as well as inhibiting a host of processes that promote inflammation and cancer.



SOURCE: M. BARDOU *ET AL/GUT* 2010

Statins' additional role "opens up a whole new therapeutic window," Liao says. There is plenty of work out there for an easy-to-take anti-inflammatory.

Randomized trials have shown that statins may ease rheumatoid arthritis and tame inflammation in a lethal condition called sepsis, a whole-body immune reaction to bacterial infection. Statins may also treat osteoarthritis, researchers reported in 2010: Statins prevent cartilage loss in mice and decrease enzymes known to abet inflammation and degrade cartilage in human cells. And then there's trauma. Anti-inflammatory effects are consistent with a 2011 study finding that older people taking statins were much less likely than nonusers to die in the hospital following a head injury.

Liao says statins' immune benefits go beyond fighting inflammation. The drugs don't appear to wipe out inflammation wholesale, as a steroid drug might, he says, but instead bring overwrought inflammation signalers "back to a normal state."

Some scientists even argue that the protection offered could extend to the inflammatory onslaught brought on by infections such as influenza. Severe flu strikes 35 million people annually. Patients already taking a statin are less likely to die of the disease than are nonusers, researchers at state health departments across the United States reported in January. Danish researchers tracking patients with pneumonia, a flu complication, reported that those on statins are less likely to die of the condition.

Despite this evidence, applying statins in the clinic could be tricky, says Edward Walsh, an infectious-disease physician at the University of Rochester in New York. "It's conceivable that you need to be on statins ahead of time, and I wouldn't advise anybody to take statins just because they might get the flu," he says. "But on the other hand, if we were faced with a really deadly flu pandemic and — good gosh — we didn't have a vaccine that worked? You could easily see how people might turn to anything that has potential."

David Fedson, a retired physician and flu researcher, has made such concerns his scientific mission. He argues that documented cases of bird flu jumping to humans in Southeast Asia make a deadly flu pandemic a plausible threat. Population studies and biological findings from the last several years suggest that statins could be lifesavers in such a scenario by blunting the flu's impact on the body. Annual stockpiles of flu vaccine and anti-flu drugs are nowhere near adequate to stop a 1918-level flu, particularly in poor countries, says Fedson, who formerly worked at the University of Virginia. The annual version of the flu vaccine might not even work against such a novel virus. "We need something else," he says.

Statins are widely available, easy to store and simple to take. Drug manufacturers in India already make a generic version of Zocor and ship it widely to developing countries at as little as 10 to 15 cents a dose, Fedson says. With appropriate planning and testing, he says, "on the first pandemic day, everyone in the world could be treated. What this means in terms of global equity is stunning. From Hyderabad to Houston, you give everyone an equal chance to live."

Not all the same

Whether statins will ascend to such wider use — and if so, which versions will work best — is still an open question. They are not all uniform. Some statins, such as Zocor, don't linger in the blood for long, Liao says. That's why doctors tell people to take them at night, when the liver actively makes cholesterol.

A short half-life might limit a statin's effectiveness against diseases not related solely to cholesterol. "If inflammation is happening around the clock," Liao says, "I'd like to have a statin that works 24 hours a day." Other statins are released over a longer time frame.

Zocor, though, might have an advantage in fighting disease more broadly, because it can gain entry into most cells. Zocor, Lipitor, Lescol and Mevacor are all lipophilic — they "like" fats — meaning they dissolve easily into most any cell's membrane, passing through it like a ghost through a wall. On the other end of the spectrum are Crestor and Pravachol. These lipophobic ("fat-fearing") statins are reluctant to dissolve into cell membranes. Makers of these drugs have by design equipped them to enter into liver cells preferentially.

A stark contrast emerges between lipophilic and lipophobic statins in studies of another menace that statins are now showing promise against: cancer.

At first blush, statins would seem to have little to offer against cancer. Yet many population studies have suggested that taking some statins can lessen a person's cancer risk. In an example reported in 2011, researchers tracked the fate of nearly every breast cancer patient diagnosed in Denmark over an eight-year span. Those taking simvastatin, the generic name for lipophilic Zocor, had 10 percent fewer cancer recurrences during a decade or so of follow-up than women not getting a statin. Women taking lipophobic statins didn't fare any better than nonusers. Similarly, lab tests have shown that Zocor and Mevacor readily kill ovarian, uterine and cervical cancer cells in lab dishes, but lipophobic Pravachol does not.

Recent research provides a biological explanation for how statins, if they can get into a cell, might fight certain cancers. Statins appear to sabotage pro-cancerous machinery within the cell by neutralizing the effect of a cancer double-agent called p53, Prives and colleague William Freed-Pastor reported in the Jan. 20 *Cell*.

The normally helpful p53 protein undergoes a Jekyll-to-Hyde transformation in roughly half of all cancers, including many aggressive ones. In breast tumors, mutant p53 ceases to check aberrant cell growth, instead frequently switching on 11 genes that encode

Statin selection Cholesterol-lowering drugs come in different varieties, some lasting longer and some liking fat (lipophilic) while others avoid it (lipophobic). The differences suggest that the best statin for the job depends on what disease needs to be treated.

Brand name	Generic name	First FDA approval	Half-life in the body	Lipophilic or lipophobic	Manufacturer and derivation
Mevacor	Lovastatin	1987	Less than 2 hours	Lipophilic	Merck, natural compounds
Zocor	Simvastatin*	1991	Less than 2 hours	Lipophilic	Merck, natural compounds
Pravachol	Pravastatin	1991	2 hours	Lipophobic	Bristol-Myers Squibb, natural compounds
Lescol	Fluvastatin	1993	Less than 3 hours	Lipophilic	Novartis, synthetic
Lipitor	Atorvastatin	1996	14 hours	Lipophilic	Pfizer, synthetic
Crestor	Rosuvastatin	2003	19 hours	Lipophobic	IPR Pharmaceuticals, synthetic

*Low-dose pill approved for over-the-counter sales in U.K.

RKIRKIMAGERY/ISTOCKPHOTO

proteins in the very chain reaction by which cholesterol and isoprenoids are produced. Prives and Freed-Pastor showed in cell-based experiments that adding a statin substantially impedes this disaster, limiting the ability of mutant p53 to switch on these genes. When the researchers treated breast cancer cells with Zocor in a lab, the cells died or showed slower growth.

There are other biological arguments in the case for statins against cancer. By thwarting isoprenoid production, Lipkin says, statins also disable a process that triggers activation of a proliferation molecule called Ras, which has been implicated in several cancers. Other evidence suggests that starving a tumor cell of cholesterol might slow runaway cell growth because cells use cholesterol as a building block for their fatty membranes.

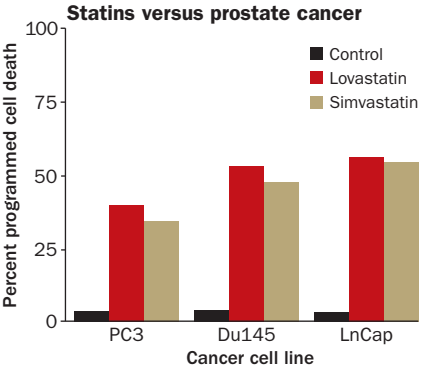
Several studies have linked high cholesterol with prostate cancer risk, and there is molecular evidence that bottling up HMGCR thwarts this cancer. A 2008 study showed that prostate cancer cell lines exposed to statins are more apt to undergo stalled growth or programmed cell suicide than unexposed cells. "The prostate cancer data actually look pretty strong," Lipkin says.

Men who underwent prostate removal for cancer were less likely to have a recurrence if they were on statins beforehand than men not getting the drugs, Johns Hopkins University researchers reported last year. While this population finding doesn't prove cause and effect, "what we're seeing does make sense in the biological data. It's nice that it hangs together," says Alison Mondul, an epidemiologist at the National Cancer Institute who coauthored the study while at Johns Hopkins.

Some say it ain't so

Inflammation and cancer aren't the only conditions beyond the heart that statins might address. Recent studies suggest that statins might counteract dementia, cataracts, multiple sclerosis, Parkinson's and depression. A National Institutes of Health website lists hundreds of clinical trials — finished, ongoing or still recruiting participants — that focus on statins' impact outside of heart disease or stroke. But some scientists remain unconvinced that statins hold more wonder drug potential.

Cancer blow A recent study shows that statins induced programmed cell death in three prostate cancer cell lines in the lab, suggesting a mechanism underlying the cancer-fighting effect seen in population studies.



SOURCE: A. HOQUE *ET AL/CANCER EPIDEMIOL. BIOMARKERS PREV.* 2008

Beatrice Golomb, a physician and neurobiologist at the University of California, San Diego, notes that studies have found some pretty bad side effects associated with statins, including muscle pain and liver damage, as well as perhaps memory loss and diabetes. Such side effects, combined with the widespread prescribing of such drugs, have led to a backlash against statins among some scientists.

Golomb says population studies aren't nearly adequate to outweigh these doubts. "This is a class of studies that should not be used for causal inferences of a drug and its outcomes," she says, citing the "healthy-user effect." People who obtain statins might have better insurance, more education and better access to health care than people who don't get the drugs, skewing the outcomes.

But physician and epidemiologist Reimar Thomsen of Aarhus University in Denmark says that while the United States does have wide disparities in health coverage, Denmark doesn't. His population study of statins and pneumonia included people covered by the same health system. "Everyone goes to the hospital for free — funded by taxes," he says.

Factors such as the variation in statins' ability to permeate cell membranes might explain some of the mixed results seen in recent studies. What's more, some patients might be hardwired to benefit more from statins than others, Lipkin says. He cites a 2005 population study that found that colorectal cancer appears to be vulnerable to statins in some people but not all.

Intrigued by that finding, Lipkin and his colleagues tested more than 4,000 people, half with colorectal cancer, looking for alterations in 40 genes known to play a role in cholesterol synthesis and metabolism in cells. The analysis showed that one-fifth of the people made a slightly altered form of the HMGCR enzyme, a form that statins didn't latch on to effectively.

"People with this variant don't respond well to statins," Lipkin says. These patients got poorer cholesterol-lowering effects and were less likely to share in statins' protection against cancer.

Pinning down these and other vagaries of statins' effects might clarify who would benefit from the drugs, and in what ways. In one trial, researchers are now randomly assigning patients who have undergone colorectal cancer surgery to get statins or a placebo along with other medications. The researchers will see which patients develop precancerous growths, or cancer itself. But results from this and other trials could be a long time coming.

Though some doctors still balk at the counterintuitive nature of giving a statin to someone with normal cholesterol, Thomsen believes the general thinking on statins is changing, if gradually.

"Five years ago, in an intensive care unit, doctors would stop statins, thinking there were other things to worry about," he says. "Now, I think the attitude is that you should keep a heavily inflamed patient on statins in the ICU. It might be dangerous to stop them." ■

Explore more

- C. Mihos and O. Santana. "Pleiotropic effects of the HMG-CoA reductase inhibitors." *International Journal of General Medicine*. April 2011.

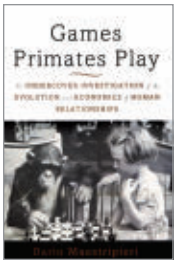
Games Primates Play

Dario Maestripieri

Even decked out in cultural finery, people make monkeys of themselves. Maestripieri, a veteran monkey investigator, builds a fascinating and occasionally disturbing case for fundamental similarities in the social shenanigans of people, apes and monkeys due to a shared evolutionary heritage.

Maestripieri spies unspoken primate customs lurking in mundane human encounters. In a crowded elevator, people instinctively stand still and avoid eye contact, keeping their distance when only two remain. An ingrained need to defuse potential aggression when confined with strangers drives this behavior, Maestripieri argues. He has observed similar behavior in pairs of female macaques put in a small cage. To break the ice, the monkeys bare their teeth to signal fear and friendliness before grooming each other. It's a short jump, he says, from caged macaques to two people in a high-rise elevator chatting nervously about the chance of rain.

Maestripieri also describes the evolutionarily deep appeal of nepotism. In female-run macaque societies, big shots' daughters are guaranteed privileged lives while daughters of bottom-feeders eke out a miserable existence. Maestripieri relates this behavior to his own run-ins with kin favoritism in Italy's military and universities.



Both people and macaques often hurt competitors if they can get away with it, Maestripieri says, but play nice in public. So it goes among scientists: Senior researchers attack rivals and young challengers in anonymous peer reviews. This would improve instantly with open review, he predicts.

Other research described in the book finds commonalities in primate cooperation and friendship, as well as in power plays, playing favorites and other dark social arts. In the end, Maestripieri's theme is hard to deny: Monkey business is everyone's business. — *Bruce Bower*

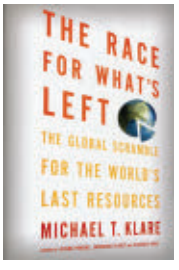
Basic Books, 2012, 336 p., $27.99

The Race for What's Left

Michael T. Klare

Had T.S. Eliot been around to read this book, he might have said: This is the way the world ends, not with a bang but a shortage.

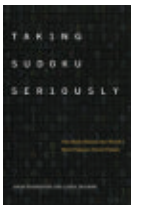
Klare offers some grim realities: "Because most of the world has already been scoured for readily accessible resource reserves, the only hope for finding more oil, natural gas, minerals and farmland will lie in extending the search to previously inaccessible or inhospitable areas."



Some might accuse Klare of underselling the ability of humans to manage these challenges. But the facts point to inescapable economic and environmental costs. The dangers of deepwater drilling, for example, were made clear by the 2010 BP oil spill. And Klare notes that an area the size of Nebraska has been leased for exploratory drilling off Greenland. Energy extraction on land isn't much easier. Tapping oil from tar sands and natural gas from shale rock is proving costly. Easy-to-get nickel and coal deposits are largely mined out, and many minerals are tucked away in remote or unstable areas — Bolivia (lithium), Niger (uranium), Afghanistan (copper) and, in West Africa, Guinea (bauxite).

Reading this book, it's hard not to think about postapocalyptic fiction in which resource scarcity leads to social disorder. Think Margaret Atwood, Cormac McCarthy and most recently Suzanne Collins' *The Hunger Games*. Yet novelists often skip over the messy parts along the road to dystopia. It's scary to think that Klare, far from crying wolf, might be providing the sordid details in real time. — *Nathan Seppa*

Metropolitan Books, 2012, 320 p., $27



Taking Sudoku Seriously

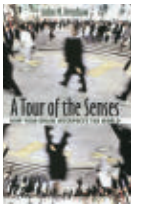
Jason Rosenhouse and Laura Taalman

A look at the popular puzzles reveals the fundamental mathematical concepts at play. *Oxford Univ., 2011, 226 p., $21.95*

Charles R. Knight

Richard Milner

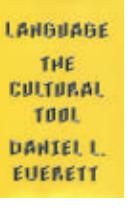
The wildlife artist and his classic illustrations of the ancient past come to life in this illustrated volume. *Abrams, 2012, 180 p., $40*



A Tour of the Senses

John M. Henshaw

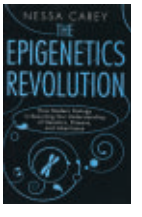
A blend of research findings and real-world anecdotes about people's sensory experiences enlivens this historical view of the science behind perception. *Johns Hopkins Univ., 2012, 272 p., $29.95*



Language: The Cultural Tool

Daniel L. Everett

A linguist who spent three decades among the Pirahã people of Amazonia presents language as a human tool that can be reinvented or lost over time. *Pantheon, 2012, 351 p., $27.95*



The Epigenetics Revolution

Nessa Carey

A look at the emerging field of epigenetics shows how chemical changes to DNA affect everything from cat color patterns to human health. *Columbia Univ., 2012, 352 p., $26.95*

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Happy 90th, *Science News*

My father has generously given a subscription of *Science News* to me since I was small. In the '60s I received a package in the mail each month containing science experiment materials and directions. So cool! We celebrated Dad's 90th birthday in April. He was an aeronautic engineer; I'm an architect. I am sure the magazine you deliver to us each month gives us the same joy. Thank you for making *Science News* a joy for so many of us for so many decades.
Becky Thompson, South Pasadena, Calif.

I'll add to what has probably been a flood of congratulations for 90 years of comprehensive coverage of all things science. I am 56 years old and a third-generation subscriber. If your records go back that far, my grandfather, Dr. Charles Wright MacMillan, was a devoted reader probably in the '20s, as was my mother from the time she was a child. My parents have been providing gift subscriptions to me and my siblings our entire adult lives, and I intend to continue that tradition for my grown children. Not sure if we win the prize for longest family subscription, but I do know that *Science News* has had a profound influence on our lives, inspiring curiosity and instilling great respect for the timeless pursuit of new discoveries.

It was a thrill to see that Janet Raloff got the plum assignment of combing through the archives. I've been a huge fan of Janet's writing for many years, and could recognize her clever, articulate style without looking at the byline.
Jack Connell, Raleigh, N.C.

Brain images questioned

Regarding the article "Cancer drug shows promise as treatment for Alzheimer's" (*SN: 3/10/12, p. 5*): Tom Siegfried (in his editorial from the same issue) talks about undue hype of breakthrough cures. Turning the page, I find two photos showing the great improvement that this wonder drug can do. Then, reading the caption I see that the second photo is "the brain of a similar mouse after three days of bexarotene treatment." These photos are unrelated! You are hyping a "soon to be" miracle drug for Alzheimer's with photos of two different mice.
Bob Clauson, via e-mail

The images in the story are indeed from different mice, but they illustrate a valid way to show that bexarotene reduced brain plaques. To spot A-beta plaques, brain tissue had to be removed from the skull, so multiple tests on the same animal were impossible. The scientists selected representative images from each group of animals (usually six or more) and combined data from individual mice to spot trends. — Laura Sanders

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

"It's a bunch of nerds making fun of nerds." —MAYIM BIALIK

Neuroscientist Mayim Bialik (left) slices a brain over dinner with costar Jim Parsons in *The Big Bang Theory*.

Scientific method acting

Mayim Bialik is a neuroscientist, and she plays one on TV. Bialik is neurobiologist Amy Farrah Fowler on CBS's *The Big Bang Theory*, a sitcom centered on the lives of four scientists at Caltech.

Bialik was an actor long before she became a scientist. As a teenager, she starred in the television show *Blossom* and the movie *Beaches*. On the set of *Blossom*, Bialik's love of science was kindled by one of her tutors, a predental student. Bialik went on to college at UCLA and finished her Ph.D. in neuroscience there in 2007.

After her first son was born, Bialik and her husband realized that the life of a research professor wouldn't provide the flexibility she wanted to spend time with her children, and she returned to acting. But she doesn't regret the time she spent earning her degree. "There's never a waste of study," she says.

The Big Bang Theory is a fictional look at scientists, but Bialik says the portrayals are true to her real-life experience. Part of the show's charm stems from the writers being as "brilliant, nerdy and geeky" as the characters they create, she says. "It's not a bunch of cool, attractive people making fun of nerds. It's a bunch of nerds making fun of nerds."

Originally, her character had no particular occupation, but when the producers saw "neuroscientist" on Bialik's resume, Amy Farrah Fowler became a neurobiologist. The decision means Bialik gets to offer pointers on how real biology labs work.

Her character always wears a lab coat and gloves in the lab, just like a good scientist should. Now and again, Bialik corrects scientific inaccuracies in the script, but sometimes science takes a backseat to gags. "I get a little twitch if something is wrong," she says, but preserving scientific accuracy is "often more complicated than it should be for a laugh."

Ongoing debates between Amy and her boyfriend Sheldon Cooper (played by Jim Parsons) about whose scientific discipline is better are right on the money. "I have for sure had that discussion, both sober and intoxicated, in graduate school," Bialik says. And like her character, she's got an argument-ending statement for any physicists who disdain brain research: "The very fact that you can *think* about which science is better means neuroscience rules." — *Tina Hesman Saey*

Scientists with an acting theory

Mayim Bialik is not the only actor with a background in science or medicine.

- **Hedy Lamarr** (shown), a celebrated actress of the 1940s and '50s, patented an idea for wireless communication.
- **Danica McKellar** (Winnie Cooper on *The Wonder Years*) contributed to the Chayes-McKellar-Winn mathematical theorem.
- Oscar winner **Natalie Portman** was a semifinalist in the 1999 Intel Science Talent Search and coauthored a brain-imaging study as a Harvard undergrad.
- Action star **Dolph Lundgren** won a Fulbright scholarship to study chemical engineering, but gave it up to become a bodyguard for actress Grace Jones.
- **Ken Jeong** (Señor Chang on *Community*) has a medical degree.
- **Masi Oka** (Hiro on *Heroes*) has a bachelor's degree in computer science and mathematics. He works as a digital effects artist when he is not acting.

FROM TOP: CBS; ASSOCIATED PRESS

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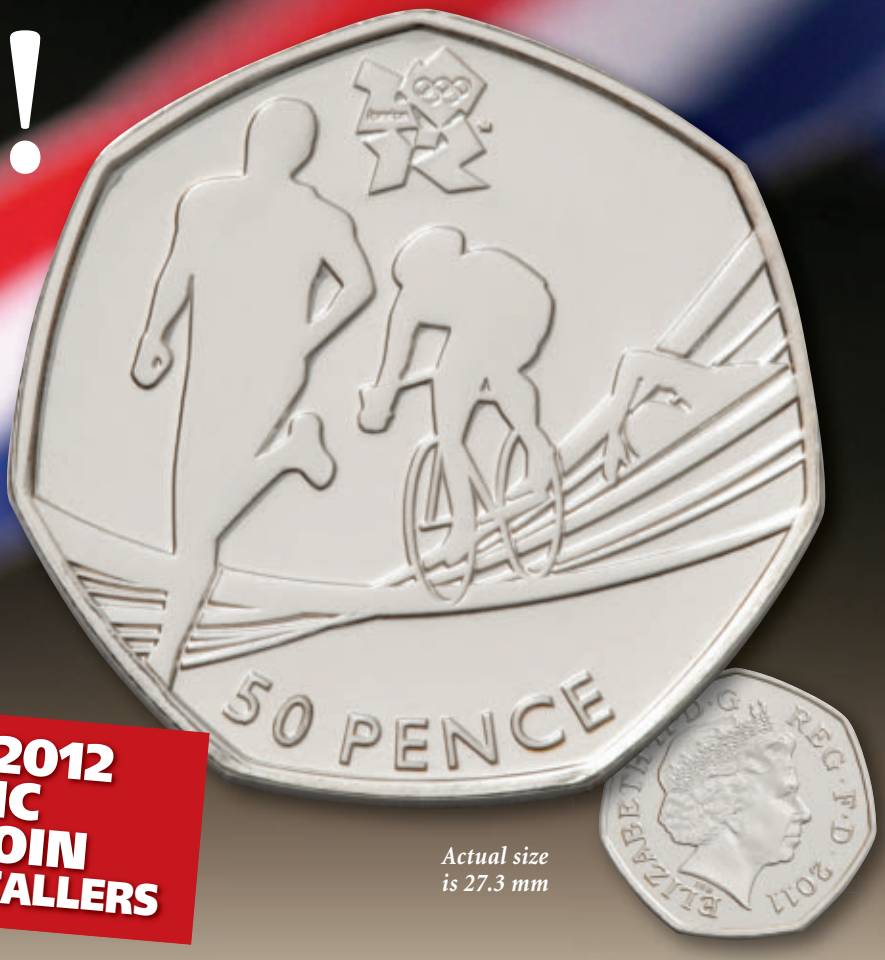

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