The Future of Joint Repair | Hot Enzymes | Computer Counterinsurgency

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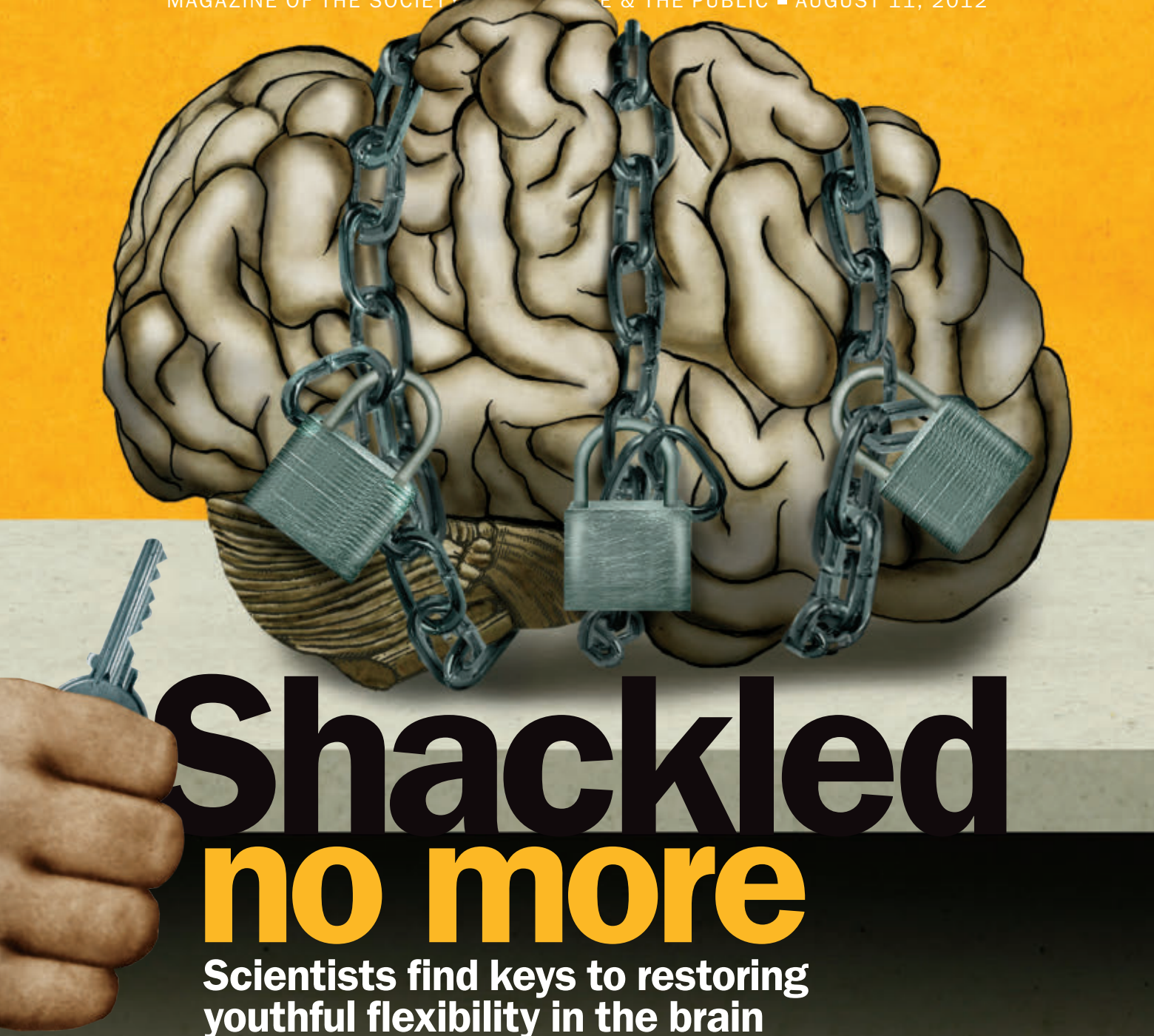
ScienceNews

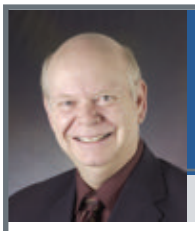
MAGAZINE OF THE SOCIETY [illegible]CE & THE PUBLIC ■ AUGUST 11, 2012



Shackled no more

Scientists find keys to restoring youthful flexibility in the brain

Lunar Swirls Demystified | Maya King Saw Beyond 2012 | Fruit Fly Cannibalism



First Federal

Special Market Opportunity

Your Expert Guide to the World's Finest Coins

Nicholas J. Bruyer, CEO, First Federal Coin Corp.
ANA Life Member Since 1974

How can two $5 Gold Eagles have a 470% difference in value?

If you're not interested in the answer, give this to your best friend.

We can't tell you how many times we're approached by very successful, very sophisticated business people who are interested in the collectible gold coin market, but don't have the first notion of where to begin.

Here's what we tell them: We think the $5 Gold American Eagle MS70 First Strike is the ideal coin to own.

Why?

There are few gold coins that collectors want more than the American Gold Eagle, especially when they find the absolutely finest quality specimens. Because for coins, value is driven by quality. The higher a coin's grade, the higher its value. It's that simple.

The highest grade possible is the exalted, absolutely flawless grade of MS70 (MS stands for "Mint State") First Releases. It's referred to as the perfect coin.

This is the pinnacle of a gold coin's state of quality. These gold coins are in demand. They've earned the highest grade possible, and for gold coin collectors, it just doesn't get any better.

Consider this example: a 1995 $5 Gold Eagle in average ungraded condition is worth about $200 today. But that same 1995 coin in perfect MS70 grade is valued at $940. That's a 470% difference!

Of course, the population of these MS70 coins is smaller, because they're examples of the finest coins.

What can make a perfect coin even better?
A "First Releases" pedigree!

Only a very small percentage of the first 2012 coins struck qualify for the additional First Releases certification from highly-respected independent grading service Numismatic Guaranty Corporation (NGC).

The 2012 $5 Gold American Eagle MS70 First Releases coin is an absolutely perfect example of the gold coin that's been an American icon for 25 years.

Actual size is 16.5 mm

You'd expect to pay a premium for such a unique combination of quality factors. But we have a limited opportunity for you:

You can now own what gold collectors compete over for only $249

No one can predict the future value of this coin, of course. Markets and prices go up and down. But the 2012 $5 Gold American Eagle MS70 First Releases is at the top of the gold coin collector's "Most Wanted" list.

Hurry! This is a first come, first served offer!

Call First Federal Toll-FREE today

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to Reserve Your 2012 MS70 NGC First Release $5 Gold Eagles for only $249 each (plus s/h)

Offer Code FDE149
Please mention this code when you call.

Prices and availability subject to change without notice. Past performance is not a predictor of future performance. NOTE: First Federal Coin Corp. is a private distributor of worldwide government coin and currency issues and other collectible numismatic products, and is not affiliated with the United States government. Facts and figures deemed accurate as of April 2012.

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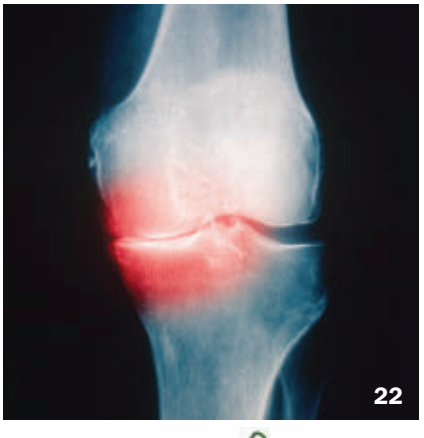

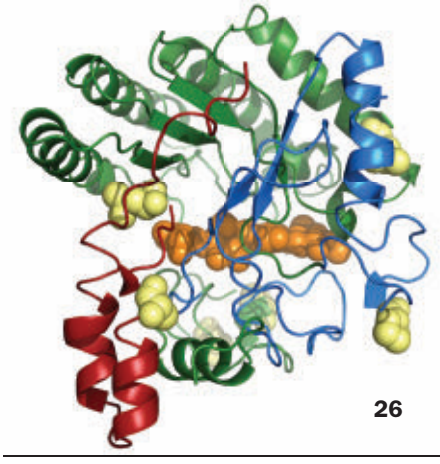

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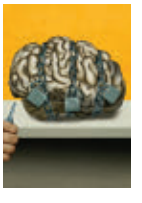

COVER Scientists have found ways to help the adult brain throw off its chains, restoring the elasticity of infancy and childhood.
Michael Morgenstern

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FROM THE EDITOR

For news about science, there's one best place

In the aftermath of Higgs boson media frenzy, it's worth noting that for a few days the world of journalism was focused on the world of science. An esoteric experiment involving scales of space, time, energy and money far beyond the usual scientific enterprise commanded the attention of media of all stripes.

In the days leading up to the report of the new particle's discovery, the Twittersphere was full of rumors, guesses and forecasts. But the actual news came first on the *Science News* website, a day before the official announcement. Officials at CERN, the European laboratory in charge of the Higgs search, had mistakenly posted a video on the Web with one experiment's spokesman describing the discovery of the new particle. In preparing for our coverage of the Higgs announcement, associate editor Kate Travis spotted the video. Soon after we reported its contents, other media scrambled to follow up the story of the "leaked" video. But it hadn't been leaked to anyone. It was just found through journalistic diligence.

Such diligence has been a hallmark of journalism at *Science News* all the time, not just for the brief moments when the rest of the media's spotlights are focused on science. For anyone who wants to know what scientists are finding out, *Science News* has always been the magazine to read. Big stories like the Higgs get covered everywhere, but it's hard to find the rest of the never-ending flow of science news anywhere but here.

In this issue, for instance, you'll find out about the newest moon of Pluto, a "sighting" of invisible dark matter in space and an explanation for an old mystery about swirls of dust on Earth's moon. You'll learn about new insights into the relationship between long-term climate change and short-term weather events. You'll get real archaeological science related to the Maya apocalypse nonsense and get the latest about early humans in North America. If your taste is biology, you'll want to check out the life span of spacefaring worms and the genetic catalog of the banana. On the medical front, you can read about a protein's key role in lung cancer, recent work on the brain's role in chronic pain and a fascinating twist in understanding amnesia, where memory loss may be tied to having too many memories. And don't forget the intersection of science and society, where mathematical analysis of the war in Afghanistan has led to robust predictions of where the most fighting will be.

It's a great magazine, isn't it?

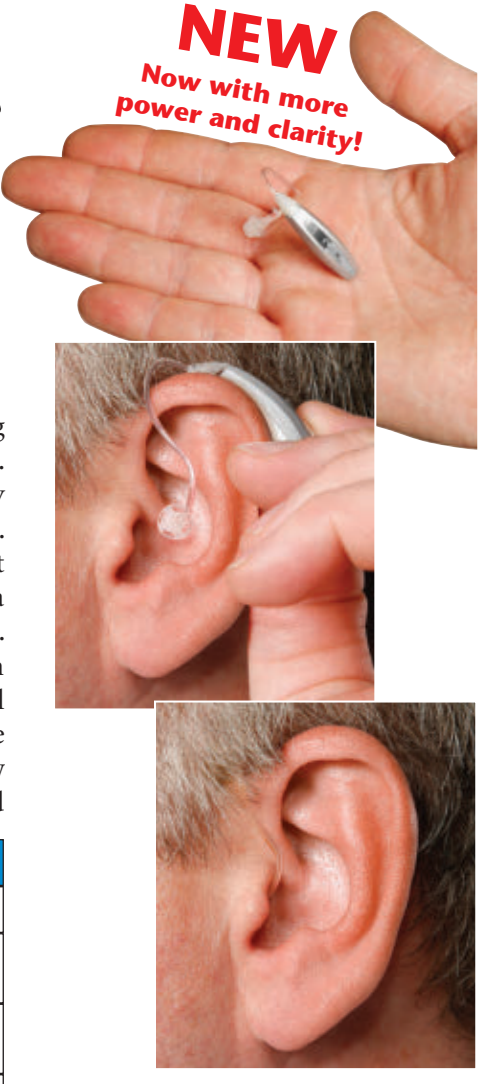
—Tom Siegfried, Editor in Chief

JIM WEBB

The invention of the year is great news for your ears

Perfect Choice HD™ is easy to use, hard to see and costs far less… it's like reading glasses for your ears™!

New Personal Sound Amplification Product is an affordable alternative



Over the years, technology has made the way we live easier, safer and more convenient. In many cases, it's even made many products more affordable… (remember how much the first VCR used to cost?). Unfortunately, the cost of hearing aids never seemed to come down. Now, a new alternative has been invented… it's called Perfect Choice HD™.

"Reading glasses for your ears"

Perfect Choice HD is NOT a hearing aid. Hearing aids can only be sold by an audiologist or a licensed hearing instrument specialist. In order to get a hearing aid, you had to go to the doctor's office for a battery of tests and numerous fitting appointments. Once they had you tested and fitted, you would have to pay as much as $5000 for the product. Now, thanks to the efforts of the doctor who leads a renowned hearing institute, there is Perfect Choice HD. It's designed to accurately amplify sounds and deliver them to your ear. Because we've developed an efficient production process, we can make a great product at an affordable price. The unit has been designed to have an easily accessible battery, but it is small and lightweight enough to hide behind your ear… only you'll know you have it on. It's comfortable and won't make you feel like you have something stuck in your ear. It provides high quality audio so sounds and conversations will be easier to hear and understand.

Perfect Choice HD feature comparison

	Perfect Choice HD	Others
Lightweight and Inconspicuous	YES	Some
Easy Toggle Switch Adjustment	YES	Few
Setting Memory	YES	Few
Tests and Fittings Required	NO	Most
Affordable	YES	as much as $5000
Friendly Return Policy	**YES**	**Rarely**

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…and other times where you need to turn up the volume

Try it for yourself with our exclusive home trial. Some people need hearing aids but many just need the extra boost in volume that a PSAP gives them. We want you to be happy with Perfect Choice HD, so we are offering to let you try it for yourself. If you are not totally satisfied with this product, simply return it within 60 days for a refund of the full product purchase price. Don't wait… don't miss out on another conversation… call now!

Affordable, Simple to use, Virtually impossible to see

Perfect Choice HD™

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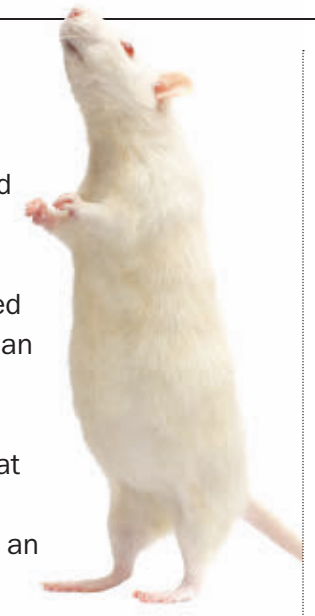
Perfect Choice HD is not a hearing aid. If you believe you need a hearing aid, please consult a physician.

80329

Say What?

Kairomone *\KYE-roh-mone\ n.*

A chemical signal produced by one species that benefits another species. Some prey animals can avoid becoming a snack, for example, by eavesdropping on the pheromones that predators use to communicate with each other. Researchers in Australia and the United States found that hungry lab rats hesitated for more than a minute and a half when approaching food dishes if a cloth infused with the marking scent of a male cat was placed nearby, even though the rats had never met a cat before. Rats still acted skittish around the dishes 24 hours after the cloth was removed, the team reports in an upcoming *Physiology & Behavior*. —*Allison Bohac*



Science Past | FROM THE ISSUE OF AUGUST 11, 1962

ONE-WAY SPACE MISSION TO THE MOON POSSIBLE — The feasibility, from a technical standpoint, of sending a man on a one-way mission to the moon without the propulsion to bring him back to earth was explored by two Bell Aerosystems Company scientists. John M. Cord, project engineer in Aerospace Preliminary Design, and Leonard M . Seale, chief of the Human Factors Section, at Textron's Bell Aerosystems Company, Buffalo, N.Y., emphasized that they do not advocate such a mission although they believe it will be possible to provide a means of returning the lunar explorer or explorers to earth at some later date.... In fact, they asserted, the man can be kept alive indefinitely to do valuable scientific work.

Science Future

August 23
Christof Koch discusses his book *Consciousness: Confessions of a Romantic Reductionist* at the Aspen Brain Forum in Colorado. See bit.ly/SFkoch

August 31
Nominations due for induction into the Space Technology Hall of Fame. See bit.ly/SFfame

September 3
Last day of Summer of Irresponsible Science at the Maryland Science Center. See bit.ly/SFSIS

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DELETED SCENES BLOG

The Higgs boson deserves all the hype it has received — and then some. Read about the particle's hidden talents in "Higgs hysteria."

Two new studies support the idea that an odd microbe cannot swap arsenic for phosphorus. Read "Arsenic-based life gets even more toxic."

LIFE

Scientists electronically tag ants (below) to watch the insects' foraging strategies. Read "Skinny searchers keep fat ants full."

ATOM & COSMOS

A new lithium source won't help researchers find more of it. See "Black hole theory deepens lithium crisis."

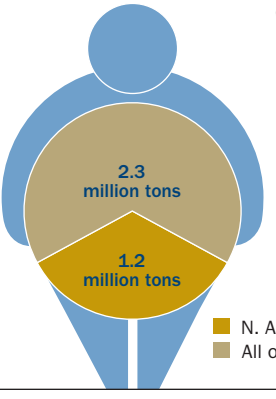
First | FOSSILS IN FLAGRANTE

Nine preserved pairs of turtles from 47 million years ago have revealed the first fossil snapshots of vertebrate sex. Dug up at a fossil-rich pit near Messel, Germany, the lovers were poisoned in the toxic depths of a volcanic lake, researchers from Germany and Switzerland report online June 20 in *Biology Letters*. The *Allaeochelys crassesculpta* couples (one pair shown) probably began mating in clean surface waters, the researchers say, but froze their position during mating and sank into a toxic layer, challenging the idea that sips from the lake's surface waters killed other creatures previously unearthed at the site. —*Devin Powell*

Science Stats | FAT ADDED UP

An estimate of the total biomass of adult humans on the planet finds that North America has just 6 percent of the world's population yet is responsible for 34 percent of global obesity tonnage.

SOURCE: S.C. WALPOLE *ET AL/BMC PUBLIC HEALTH* 2012



Obesity tonnage worldwide

Total human biomass in 2005	=	287 million metric tons
Portion from being overweight	=	15 million metric tons
Portion from being obese	=	3.5 million metric tons

CLOCKWISE FROM TOP: FLIBUSTIER/ISTOCKPHOTO; COURTESY OF NIGEL FRANKS; STEPHEN EGTS; SENCKENBERG NATURMUSEUM FRANKFURT

“People have been looking at these strange, mysterious structures since the invention of the telescope. Now we know exactly how they are made.” —RUTH BAMFORD, PAGE 8

In the News

STORY ONE

Simulation helps forecast fighting in Afghanistan

WikiLeaks data plus math predict insurgent activity

By Rachel Ehrenberg

A new advanced weapon may offer guidance on strategy for the war in Afghanistan: math. Using secret U.S. military logs made public by WikiLeaks, scientists have created a mathematical simulation that may help predict the intensity and whereabouts of future insurgent activity.

The simulation also evaluates its own predictions, acknowledging that some events may be impossible to foresee. Such an approach might help decision makers better weigh their options, the researchers say online July 16 in the *Proceedings of the National Academy of Sciences*.

“If the model says there’s a lot of uncertainty about what’s going to happen in an area, then you might act differently than if you were more certain that you were going to see an increase in activity,” says computer scientist Guido Sanguinetti of the University of Edinburgh, coauthor of the new study.

Sanguinetti and his colleagues took a mathematical approach that’s typically used by epidemiologists to predict the spread of a virus or disease outbreak. But instead of using data on the where and when of newly infected individuals, the researchers used details from the Afghan War Diary, a blow-by-blow of the conflict in Afghanistan that was published by the whistle-blower website WikiLeaks in 2010. The documents contain more than 75,000 logs of military actions, from routine searches to major gunfights.

STR/ASSOCIATED PRESS

Smoke billows from an Afghan police compound after a 2011 attack by suicide bombers and gunmen in Kandahar, southwest of Kabul. A new study offers a way to predict the locations and magnitude of insurgent activity.

Data fed to the computer simulation are represented as coordinates on a map, generating a visual representation of how and where in Afghanistan the conflict escalated from 2004 through 2009. After training the model on the War Diary data, the team asked it to predict the likelihood and whereabouts of armed opposition group activity in 2010.

The program fared pretty well: In Baghlan province, for instance, the simulation predicted a median increase of 128 percent in armed opposition group activity from 2009 to 2010. The Afghanistan NGO Safety Office, a nonprofit group dedicated to protecting aid workers in dangerous regions, reported that activity in Baghlan actually rose by 122 percent, from 100 incidents in 2009 to 222 incidents in 2010. The group reported a 19 percent decrease in insurgent activity in Badakhshan province in the northeast; the simulation predicted a median decrease of 23 percent. It also gave an 80 percent probability that there would be between 15 and 85 insurgent attacks in Badakhshan in 2010; there were 35.

“We can’t say exactly what’s going to happen,” says Andrew Zammit-Mangion, who wrote thousands of lines of computer code for the program as part of his dissertation at the University of Edinburgh. “But we can give accurate probabilities of what can happen.”

The program also allowed the

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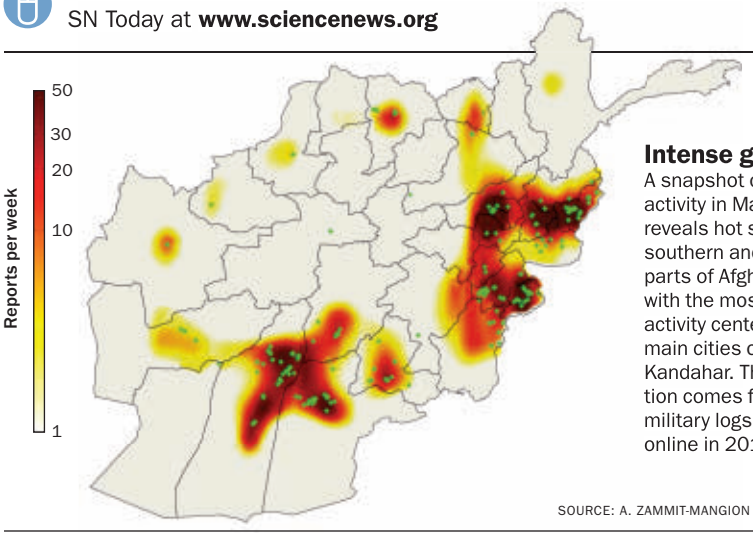


Intense geography
A snapshot of insurgent activity in March 2008 reveals hot spots in the southern and eastern parts of Afghanistan, with the most intense activity centering on the main cities of Kabul and Kandahar. The information comes from secret military logs leaked online in 2010.

SOURCE: A. ZAMMIT-MANGION *ET AL/PNAS* 2012

researchers to probe whether particular fixed variables, such as proximity to the Pakistan border, could be used to strengthen the computer program's predictive power. Both population density and closeness to a major city were associated strongly enough with an uptick in conflict intensity that knowing those variables improved the program's predictions. Terrain type and elevation, on the other hand, weren't especially linked to activity. And while insurgents are known to seek refuge in Pakistan, considering the proximity of activity to the Pakistan border didn't help refine the program's estimates overall. Such a border effect may have been canceled out by low levels of activity in some bordering provinces, such as the relatively quiet Nimruz in the southwest, compared with others with more activity.

Even though the incidents reported in the War Diary logs were extremely variable and in many cases nonviolent, the computer program succeeded at tapping into and making something of the data, a feat that even the scientists didn't expect.

"I was very surprised, actually," says Zammit-Mangion. He had considered breaking the incidents down into different types, but that would have led to fewer data points per type, compared with one enormous dataset. "I was thinking of refining it later but found we didn't need to."

Including additional data, such as information on U.S. military troop movements, could make the simulation perform even better, Sanguinetti says.

The real strength of the simulation is that it highlights regions where it's difficult to predict future activity, says statistical epidemiologist Peter Diggle of Lancaster University and the University of Liverpool, both in England.

"It delivers best guesses with honest estimates of how good those guesses are so you don't have to be dogmatic," says Diggle. "This is a very nice and imaginative application of this modeling approach. It's a good piece of work."

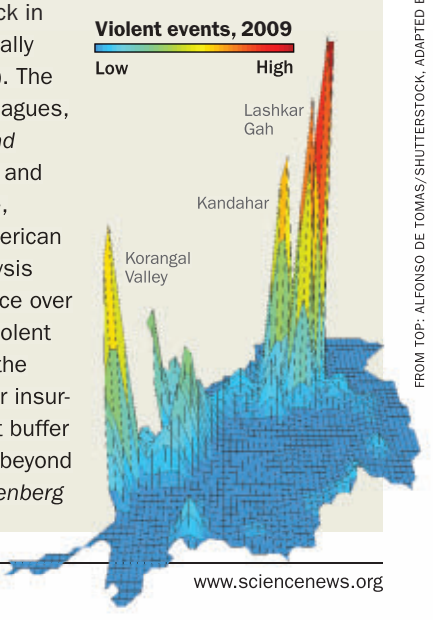
Such simulations nicely complement approaches that look at the social dynamics of a conflict and the demographics and history of the people involved, says John O'Loughlin, an expert in the geography of conflict at the University of Colorado Boulder.

Research by O'Loughlin and others that also assessed the WikiLeaks logs paints a similar picture of the conflict's escalation. The number of violent events skyrocketed in early 2008, for example. While the predictions generated by the new computer program are at the province level and not very fine-grained, they are still informative, he says, especially given how wars are fought today.

"During the Cold War it was tit-for-tat modeling at the international level," he says. "Almost all wars are irregular these days. It's not armies lining up against each other with front lines." ■

Back Story | PUTTING WIKILEAKS ON THE MAP

While the secret U.S. military logs released by WikiLeaks in 2010 contain little in the way of intelligence, the data are still revealing, says political geographer John O'Loughlin of the University of Colorado Boulder. Three-dimensional mapping of more than 46,000 violent events recorded in the leaked documents, for example, shows a hefty uptick in violence after 2006 that increases dramatically in 2009 (pictured, as viewed from the north). The fine-grained analysis by O'Loughlin and colleagues, published in 2010 in *Eurasian Geography and Economics*, pinpoints the cities of Kandahar and Lashkar Gah as growing hotbeds of violence, along with the Korangal Valley (known to American troops as "The Valley of Death"). Their analysis also reveals the spread of insurgency violence over time. Until mid-2009, about 46 percent of violent events took place within 100 kilometers of the border with Pakistan, an important refuge for insurgents. The violence then creeps beyond that buffer zone, illustrating the diffusion of insurgents beyond standard Taliban strongholds. —*Rachel Ehrenberg*



FROM TOP: ALFONSO DE TOMAS/SHUTTERSTOCK, ADAPTED BY E. FELICIANO; J. O'LOUGHLIN *ET AL/EURASIAN GEOGRAPHY AND ECONOMICS* 2010

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Get Rid of Hard Water Problems Without Expensive Water Softeners

Made with Pride in North Canton Ohio

Eliminate unsightly limescale and actually prevent it from forming

By: B. Michael John, Media Services

Are you tired of hard water problems: **unsightly scale** on fixtures, toilet bowls and showerheads; **scale build-up** in pipes; **poor lather** from detergents and shampoos; **clothes that look dingy** and feel harsh after washing; **hard-to-remove film** on glass shower doors, walls and bathtubs; hair that feels sticky and looks dull; **dry, itchy skin**; **soap scum** on sinks and appliances?

If you answered "yes" to any of these questions, you are the perfect candidate to try the ScaleRID Electronic Hard Water Treatment System in your home for the next 120 days.

If you don't get RID of your hard water problems, you will receive a complete refund.

But that won't happen because ScaleRID is one of the most advanced, effective electronic hard water treatment systems you will find for such a low cost. It's also the easiest to install.

Q. But how exactly does it work?

A. The ScaleRID, which is about 4" x 6" and weighs less than 3 pounds, treats hard water with a patented technology that induces variable electric fields with a continuously changing frequency. This field causes the dissolved minerals of calcium and magnesium to crystalize and remain in suspension, instead of adhering to the walls of piping, faucets or heating elements.

These minerals, now in suspension, flow with the water and are discharged down the drain (see diagram and our ScaleRID white paper at www.usascalerid.com for more details).

Q. How is ScaleRID different from salt-based water softeners?

A. In a salt-based water softener, an ion exchange takes place and the hard ions in calcium and magnesium trade places with the soft ions from the salt poured into the tank used during regeneration.

Most of the salt used for regeneration gets flushed out of the system. The process can be damaging to the environment, not to mention wasting 80-150 gallons of water a week or month and adding unhealthy salt to your water.

Q. What makes ScaleRID so special compared to other electronic water treatment systems?

A. International Home Shopping (IHS) just completed an upgrade of ScaleRID to make it: 1) **the easiest to install;** with our new exclusive Quick Connect Coil you can have your ScaleRID up and running in about 10 minutes, 2) **one of the most advanced;** new computer chips replace integrated circuits to make the ScaleRID the most efficient device for its cost, 3) **the least expensive;** even with all our improvements, we lowered our prices so that everyone with hard water problems can get RID of them simply, safely and inexpensively.

Q. Without regeneration, how will I know the ScaleRID is working?

A. You will get the most important benefit: you will not be bothered by hard water problems. Within the first week, you will see the elimination of new scale deposits and loosening of existing scale. Soap will lather easier; your skin will feel softer and your hair cleaner and more manageable. You will notice the reduced amounts of soap, shampoo, detergents and cleaners you need to use to get jobs done. Cleaning of surfaces, especially chrome, baths and sinks will be easier. Your water pressure may improve without scale clogging your pipes.

Q. This sounds too good to be true; does ScaleRID really work?

A. The science behind ScaleRID has been around for years. There are hundreds of thousands of units operating in 40 countries. But thanks to IHS, everyone can now utilize this science because ScaleRID is so easy to install, so efficient and effective and so reasonably priced.

Pipe Before ScaleRID — Pipe 4-5 weeks of ScaleRID use

Q. What is the difference between the SR-1000 and the SR-2000?

The big difference is that the ScaleRID SR-1000 is for moderate to hard water and comes with one Quick Connect Coil. The ScaleRID SR-2000 is for very hard water or for homes/duplexes with large water usage and comes with two Quick Connect Coils.

You cannot afford not to try ScaleRID in your home for 120 days. If it's not for you, return it for a complete refund. YOU RISK NOTHING.

Just think how you'll enjoy not lugging those salt bags around, but still getting rid of hard water problems.

How To Order

The ScaleRID SR-2000 for very hard water is regularly $497 plus $10 shipping. For the next 10 days you receive a $250 discount, over 50% off, plus FREE shipping and pay $247 delivered.

The ScaleRID SR-1000 for hard water is regularly $197 plus $10 shipping. Over the same time period you receive a $50 discount and FREE shipping and pay only $147 delivered.

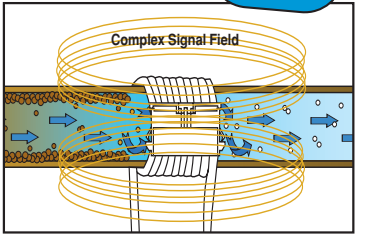
For more information or to start your 120-Day ScaleRID No Risk Challenge call toll-free or go online at **www.usascalerid.com.**

Quick Connect Coil attaches in seconds around water pipe. Works on copper, PVC, galvanized pipe.

ScaleRID How It Works

100% Satisfaction Guarantee 120-Day Home Trial

- Install in 10 Minutes - **No Plumbers**
- **No Maintenance**
- The system consists of the energizing unit and a **Quick Connect Coil** that simply snaps around the pipe of the incoming water supply.
- The 21st century way to **eliminate limescale and hard water problems** in city water and well water.
- Unit **weighs less than 3 lbs.** No more big tanks and heavy salt bags to lug around.



Incoming water saturated with calcium and other mineral ions in solution.

Solution less saturated with calcium and able to dissolve existing scale.

ScaleRID vs. Salt-Based Water Softener Comparison		
	ScaleRID	Salt-Based Water Softener
Cost	under $150	$400 - $1,800.00
Maintenance	none	constant maintenance
Treatment Method	electrical field	salt-based ion exchange
Installation	10 minute do-it-yourself	professional
Scale Removal	YES	NO
Scale Prevention	YES	NO
Back Flush	NO	YES 80-100 gallons per week
Skin Test	makes skin softer	makes skin dry
Soap Test	soap suds wash away	soap suds difficult to remove
Health Benefits	leaves in essential minerals	exchanges minerals with sodium

Non-Paid ScaleRID User Testimonials

The ScaleRID has helped my plumbing tremendously. It has cleaned out the build-up on all my faucets. There is no more residue on my sink... and most importantly, not in my coffee pots. I am a very satisfied customer.

Mrs. Wagner, WY

The product is very wonderful. There is no more build-up on my shower heads. They are no longer plugging up ...from all the years of build-up. I have only had the product for several months, however I am very satisfied.

Mr. Finch, WA

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Moon patterns finally explained

Solar wind–deflecting fields make swirling lunar features

By Meghan Rosen

Scientists have charged up an old moon mystery. New research suggests that swirling designs on the dusty lunar surface might be the product of electric fields generated by magnetic bubbles.

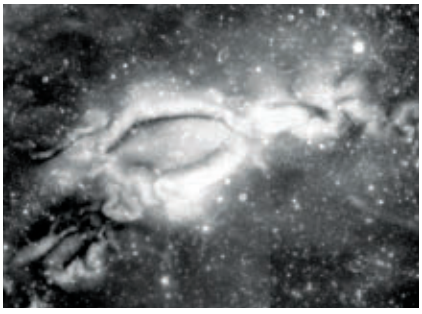
"People have been looking at these strange, mysterious structures since the invention of the telescope," says physicist Ruth Bamford of the Rutherford Appleton Laboratory in Didcot, England. "Now we know exactly how they are made."

The milky patterns stand out like pale flesh against darkly tanned skin. It's as if you used sunblock to paint whorls on your arm and then spent the day outside, says planetary geologist Georgiana Kramer of the Lunar and Planetary Institute in Houston. The sun would color everything but the protected skin, leaving the whorls a lighter shade.

Scientists have long suspected that weak magnetic fields near the moon's surface might shape the looping patterns. The moon doesn't have a dynamo-driven magnetic field like Earth's, but researchers have found patchy magnetic bubbles scattered throughout the lunar crust.

Data from the Apollo missions fed a theory that the moon's magnetic bubbles act like a solar wind sunblock. The solar wind — a steady stream of charged particles from the sun — constantly buffets the moon, turning pale lunar dust dark. But magnetic bubbles might protect the moon's crust, keeping silvery soil fresh and young-looking.

The mystery, Bamford says, was how such puny fields could deflect the raging solar wind. The answer is the bubbles' electric field, Bamford and her colleagues suggest in an upcoming *Physical Review Letters*.



Bright designs called lunar swirls dot the moon's surface. This one spans about 60 kilometers of the nearside.

Usually, the solar wind's charged particles travel together. But when the wind smacks into the moon's magnetic bubbles, flimsy negatively charged particles skirt around the bubble and hefty positive ones try to penetrate it. Splitting apart these oppositely charged particles whips up a heavy-duty electric field.

Bamford's team created a scaled-down laboratory version to find out if human-made magnetic bubbles could deflect rushing rivers of particles.

The researchers used a device called a solar wind tunnel to shoot a jet of blazing particles down a tube. The searing stream toasted any object in its path, except, the team discovered, a magnet. The scientists showed that a thin electric field formed around the magnet, shielding it — and anything behind it — from the scorching flow. "It works incredibly well," Bamford says. Even a marshmallow placed in the magnet's wake would escape melting, she says.

"The work ties a bunch of ideas together," says planetary scientist Ian Garrick-Bethell of the University of California, Santa Cruz. "And the lab model is really cool." ■

Hubble spots a fifth Pluto moon

Dwarf planet's celestial retinue continues to expand

By Nadia Drake

Pluto, the popular dwarf formerly known as a planet, has another little friend: a fifth moon, first reported early the morning of July 11 on Twitter.

"Just announced: Pluto has some company — We've discovered a 5th moon using the Hubble Space Telescope!" tweeted Alan Stern of the Southwest Research Institute in Boulder, Colo.

On July 7, Mark Showalter of the SETI Institute in Mountain View, Calif., spotted the fifth moon, referred to as P5 for now, in images captured by Hubble.

P5 revealed itself in 14 sets of images, each containing around a dozen three-minute exposures. Hubble has been peering at the Pluto system since June, tasked with helping astronomers detect any potential hazards to NASA's New Horizons spacecraft as it flies by the dwarf planet in 2015. The team is hoping to have enough time to plan an alternate route through the system if the probe's current trajectory points toward trouble.

Just a tiny little thing, P5 measures between 10 and 24 kilometers in diameter, putting it in the running to be Pluto's smallest moon. It joins a moony menagerie that includes (comparatively) enormous Charon, the satellites Nix and Hydra, and the moon still known as P4, discovered last year by Showalter.

"We fully expect to discover still more moons," says Stern, the principal investigator of the New Horizons mission. "Every time we look harder, we find another."

P5's orbit lies between the orbits of Nix and Charon, making it the most interior of the smaller moons. That explains why P5 has been hard to see. "We're looking right next to Pluto," Stern says. "So there's this bright searchlight that we have to deal with."

NASA

"It's a new way to probe the universe when it was very young." —ZOLTAN HAIMAN

Dark matter filament illuminated

Astronomers image one strand in a shadowy cosmic web

By Devin Powell

An invisible web thought to span the cosmos has now revealed one of its strands.

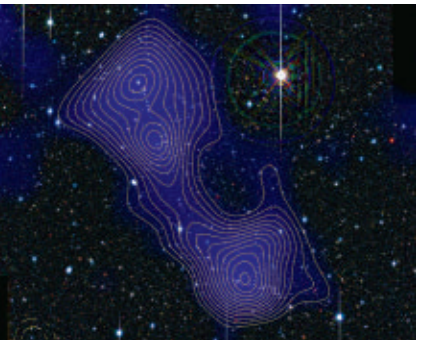
That thread is spun of dark matter and connects two titanic clusters of galaxies. Its discovery, reported July 12 in *Nature*, supports the idea that galaxy clusters grow at the intersections of dark matter filaments, and its heft backs the claim that such filaments hide more than half of all matter.

As the name suggests, dark matter is difficult to detect because it gives off no detectable radiation. The material's presence is typically inferred by observing its gravitational pull on stars and galaxies.

But dark matter's gravity also warps spacetime and bends light passing nearby, so that more distant galaxies beyond the intervening dark matter appear distorted.

Jörg Dietrich, an astronomer at the University Observatory Munich, and his colleagues focused on Abell 222/223, a pair of galaxy clusters that are close together and thus should be connected by a relatively massive filament. Using the Subaru telescope in Hawaii, the researchers looked at light from distant galaxies passing through the space between the clusters.

Sure enough, the distorted shapes of the galaxies revealed a thick cord of matter with a mass comparable to that of a small galaxy cluster. Gas can account for only about 9 percent of that mass. Dark matter seems to make up the rest.



Contour lines trace an invisible dark matter filament connecting the galaxy clusters Abell 222 (bottom) and Abell 223 (top) in the night sky.

"In the future I expect we will extend this and see more of these filaments," says Meghan Gray, an astronomer at the University of Nottingham in England who wasn't involved in the study.

Early stars made sight yet unseen

Signature could someday be detected by radio telescopes

By Nadia Drake

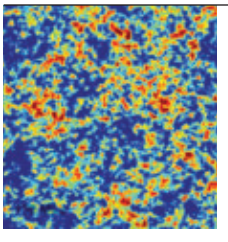
A 3-D simulation of the early universe suggests that the first stars left a cosmic signature large enough to be read by radio telescopes.

"It's a new way to probe the universe when it was very young," says Zoltan Haiman, a cosmologist at Columbia University, who was not involved in the new work. "We have very few ways to do that."

Studying early star formation is challenging because the first galaxies were so small and, because of the universe's expansion, are now exceedingly distant.

But the new simulation, described online June 20 in *Nature*, suggests that a stellar signature exists in the form of fluctuating radio waves, oscillations produced when young stars and galaxies warm and excite surrounding hydrogen gas. Simulated when the universe was 180 million years old, the stars and galaxies are distributed in a distinct, detectable pattern.

Eli Visbal, a graduate student at Harvard University, and his colleagues simulated a cube of space measuring 1.3 billion light-years across. They filled it with hydrogen gas and dark matter, the invisible counterpart to normal matter, and accounted for the recent observation that the two kinds of matter travel at different speeds. These different rates, when combined with varying densities of each substance, affect star formation by stunting growth in some places and promoting it in others. "The dark matter collapses into clumps," Visbal says. "And the gas, due to the force of gravity, falls into these clumps and forms stars and galaxies."



A computer simulation of the early universe depicts regions of intense star formation (red) separated by voids (blue). Radio telescopes might be able to detect a similar pattern.

But not where the gas is moving too quickly relative to the dark matter clumps, which then have to tug harder to get the gas to come inside. A paucity of gas produces a star-forming void, while dense gas congeals to form clusters of stars and galaxies. Those clusters then heat up and excite the surrounding sea of neutral hydrogen atoms, which emit radiation detectable by radio telescopes.

But such telescopes would have to scan the sky at a frequency lower than the band typically used by today's most powerful radio detectors. Future instruments, such as the enormous Square Kilometer Array now under development on two continents, could do the job. Another option, says UCLA astrophysicist Steven Furlanetto, would be a proposed project called the Dark Ages Radio Explorer, a lunar satellite that would use the moon as a shield against interference from technologies like television and radio.

FROM TOP: J. DIETRICH/UNIV. OBSERVATORY MUNICH; E. VISBAL *ET AL*/*NATURE* 2012

Body & Brain

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Protein goes rogue in cancer

Rac1b could be target for treatment in lung malignancy

By Nathan Seppa

A defective protein might be a key go-between in the string of terrible molecular events that lead to lung cancer. The protein, Rac1b, gets activated by other compounds and launches cells toward malignant behavior, experiments in human cells and mice suggest.

The findings open the door for lung cancer researchers to investigate the molecular chain reaction in which Rac1b is involved. Since Rac1b seems to show up early in lung cancer, it might also make a target for diagnosis or early-stage treatment, researchers report in the July 11 *Science Translational Medicine*.

"This is really comprehensive work," says Farrah Kheradmand, a pulmonologist at Baylor College of Medicine in Houston who wasn't part of the study. "This gives us ammunition to go after Rac1b, an inconspicuous molecule, to try to inhibit it."

Rac1b is a variant of Rac1, a protein involved in cell proliferation. But while the gene that encodes Rac1 routinely turns itself off after producing its protein, the variant gene making Rac1b doesn't. "It's not meant to be on all the time," says study coauthor Derek Radisky, a cell biologist at the Mayo Clinic in Jacksonville, Fla. So the aberrant version of the protein gets made in excess.

> "This gives us ammunition to go after Rac1b, an inconspicuous molecule, to try to inhibit it."
> FARRAH KHERADMAND

Because earlier work hinted that Rac1b might play a role in some cancers, Radisky and his colleagues examined lung tissue from smokers with lung cancer and found consistently high concentrations of the rogue protein in cancer cells.

Tests in mice revealed that Rac1b gets activated by an enzyme called MMP3, which is involved in the breakdown and rebuilding of connective tissues such as collagen and elastin.

The mouse experiments indicated that MMP3 activation of Rac1b triggers a process by which lung-lining epithelial cells abandon their posts and change behavior. While the process helps in wound healing — when cells are called upon to take on emergency roles — it has also been linked to cancer. Some work suggests it is a way for tumors to create space for themselves, Radisky says.

But the process, especially when it involves Rac1b, still remains hazy. For instance, it's not clear how MMP3 activates Rac1b, he says. Nevertheless, says Sandra McAllister, a cancer biologist at Harvard Medical School, Rac1b does help to fill in one of the gaps in the understanding of lung cancer progression.

Some brains may be primed for pain

Addiction-like process may extend hurt long after injury heals

By Laura Sanders

A signal in the brain can predict who will continue to suffer back pain more than a year after an initial injury. This early warning sign could reveal new ways to reverse or prevent pain that lingers long after an injury heals, scientists report online July 1 in *Nature Neuroscience*.

"We're very excited about these results," says coauthor A. Vania Apkarian of Northwestern University in Chicago. "We think they open up a whole new way of looking at chronic pain."

The study included 39 people with newish back pain, about half of whom still suffered a full year later. These people's pain had turned chronic, morphing from the pain associated with the original problem to something more devastating. At the start, pain intensity was similar in people with chronic pain and in those who recovered.

But people whose pain turned chronic had an unusually strong connection between two parts of their brains: the nucleus accumbens and the prefrontal cortex. These two regions behaved in tandem, brain scans revealed, so that when one was busy, the other was too. The strength of this connection predicted which participants would have lingering pain a full year later: The stronger the connection, the more susceptible a person was to chronic pain.

"This is something we can study," says neuroscientist Laura Stone of McGill University in Montreal. "We can figure out how to target this to prevent that transition."

Earlier studies have cataloged brain differences in people with chronic pain and healthy controls, but researchers never knew whether such differences were the cause of chronic pain or an effect of living with it. This study is the first to uncover a signal that's present before pain becomes chronic, Stone says.

The study may also link chronic pain development to the brain's addiction machinery, which includes the nucleus accumbens. "This is certainly part of the addiction pathway," Apkarian says. Though the idea hasn't been tested, he says, chronic pain may stem from the brain essentially becoming addicted to pain.

Stone says the concept of pain co-opting the addiction circuitry in the brain makes a lot of sense, but it's too early to say whether that idea is right. ■

"I find this so exciting because of the cognitive rehabilitation possibilities."—MARK BAXTER

Memories clutter brain in amnesia

Information overload may clog up access to relevant data

By Laura Sanders

In a paradoxical twist, people with amnesia can get bogged down by too many memories. Unwanted, irrelevant information crowds in and prevents amnesiac patients from recognizing objects, scientists report in the July 12 *Neuron*. The finding suggests that amnesia isn't strictly a memory problem, and may even point out ways to help people with the disorder live more normally.

Most people consider amnesia a breakdown of memory that leaves sufferers unable to recall a conversation they had minutes earlier, says study coauthor Morgan Barense of the University of Toronto. While it's true that people with amnesia have striking memory deficits, "the real picture is more complicated," she says.

People with amnesia caused by damage to a brain region near the ears called the perirhinal cortex also have problems recognizing objects, Barense and colleagues found. In the study, two people with this form of amnesia assessed a series of pictures of two objects — squiggly blobs with distinctive patterns of lines. The objects, shown at different rotations, were either identical or slightly different.

At first, people with amnesia were just as good as people with functioning recall at deciding whether the two objects were the same. But as the experiment wore on, amnesiac participants' performance started to crash.

"They're doing fine, they're doing fine — and then all of a sudden, it was like a switch flipped," says Barense.

After ruling out other possibilities, the researchers landed on what Barense calls a "wildly paradoxical conclusion" to explain the crash: too many memories. As the participants saw more and more objects, memories of irrelevant features of objects from previous pictures started to clog up the works, interfering with the present task.

These interfering memories weren't for whole objects. Instead, small features common to many of the objects, such as the tilt of a line or the precise shading pattern, seemed to cause the problem.

When researchers removed some of these common features, amnesiac participants' performance shot right back up, suggesting that memories of the objects' elements were indeed hindering performance.

For people with amnesia, these fine details may not coalesce in their minds as a coherent object, a deficit that could explain both the memory problems and the object perception problems.

"These amnesiac patients have fragments of memories, but they're not bound together," says Barense.

People who fail to form such a representation in their minds won't be able to recognize that object or remember it later. And the perirhinal cortex, once thought to be used mainly for memory, seems to be a place where this coalescing happens, the researchers propose.

The results blur the line between memory and perception in the brain, says neuroscientist Mark Baxter of Mount Sinai School of Medicine in New York City. "The idea that perception and memory are different is folk psychology," he says. This new study and others like it show that the brain's memory system and perception system may be one and the same, he says.

Streamlined changes to the environment, such as exchanging multiple complicated remote controls for one simple, distinctive one, might help some people with amnesia.

"I find this so exciting because of the cognitive rehabilitation possibilities," Baxter says. ■

NEWS BRIEFS

Deaf people see and touch differently

People who are born deaf process touch and visual input in what would be the brain's hearing system, Christina Karns of the University of Oregon and colleagues report in the July 11 *Journal of Neuroscience*. Figuring out how that happens may help deaf people who get cochlear implants, which require the auditory cortex to go back to processing sounds. — *Laura Sanders*

Rare gene tweak confers Alzheimer's immunity

A rare genetic variant seems to protect against Alzheimer's disease. The change blocks production of the amyloid-beta protein that builds up in the brains of people with Alzheimer's, scientists from deCODE genetics in Reykjavik, Iceland, report online July 11 in *Nature*. The variant also protects against brain decline among elderly people without Alzheimer's, suggesting that A-beta might have a role in normal aging. —*Laura Sanders*

To serve and protect

Cells called oligodendroglia are known to wrap insulating myelin around neural extensions called axons. As they swaddle axons, oligodendroglia also feed them energy-boosting lactate, researchers at the Johns Hopkins University School of Medicine and colleagues report online July 11 in *Nature*. This feeding process could be what goes wrong in amyotrophic lateral sclerosis, or Lou Gehrig's disease, the scientists suggest. — *Laura Sanders*

Life

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Insulin may fuel overgrown organs

Hormone seems to boost growth of outsized body parts

By Susan Milius

Some of the animal kingdom's showiest extremes, from deers' antlers to the outsized horns of male rhinoceros beetles, may be natural insulin meters.

As an animal grows, the nubbins of tissue that will form its big weapons or displays may be more sensitive to insulin than other developing body parts, Douglas Emlen of the University of Montana said July 10.

The tissue forming the horn on the rhinoceros beetle *Trypoxylus dichotomus* is more sensitive to insulin than tissue elsewhere, offering a possible explanation for how some animals' outsized body parts evolved.

The proposal "potentially narrows the range of explanations for the evolution of ornaments and weapons," said Bob Montgomerie of Queen's University in Kingston, Ontario, who studies courtship-related features in birds.

Insulin orchestrates growth in tune with how much food a young animal gets, Emlen said. A well-fed youngster flush with insulin will presumably grow the most spectacular horns or other paraphernalia, while underfed rivals remain stunted. If the growing antlers or other extreme structures are supersensitive to insulin, they will supersize out of proportion to less sensitive tissue.

That's the case for the horns of the rhinoceros beetle *Trypoxylus dichotomus*. Males grow horns about two-thirds as long as the rest of their bodies. They use these weapons to knock rivals away from sap-oozing wounds on trees where females feed. The horns are eight times more responsive to insulin or insulin-like growth factors than other body parts.

That sensitivity fits with reports from other researchers that insulin or related signals affect development of antlers in red deer and the outsized male claws in some crustaceans. For those animals, though, researchers haven't yet explored how the weaponry tissues' sensitivity compares with that of other body parts.

Differences in insulin sensitivity could provide an underlying mechanism for some of the outrageous traits that evolve through sexual selection, a quirky force that favors weaponry for trouncing rivals or flashy structures for winning the attention of mates.

"If you don't know how something works, you can't really be certain that you understand how it could have evolved," Montgomerie said.

Young fruit flies go cannibalistic

Surprising actions may aid future studies of predation

By Susan Milius

Larval fruit flies, supposedly relentless devourers of rotting fruit, at times leave their regular laboratory food to stalk, kill and group-cannibalize some of their older, fatter fellows, scientists report.

This predatory cannibalism shows up in *Drosophila melanogaster*, the fly species that generations of biologists have grown in untold numbers, Roshan Vijendravarma of the University of Lausanne in Switzerland reported July 7. He and Lausanne colleagues documented the behavior in both Canton S fruit flies, a strain raised in labs for more than six decades, and the Valais strain, brought into labs only in the last two years.

"I have never read or heard about this, and I was absolutely stunned that nobody has ever noticed this before," said Thomas Flatt of the University of Veterinary Medicine in Vienna. "This story is to my mind of great biological interest, and it shows very clearly that there are many surprises left, even in a well-studied model organism."

Because fruit fly genetics is known in such detail, Vijendravarma said his discovery may allow researchers to study the evolution of predatory cannibalism at the molecular level.

The closest reports Vijendravarma has found to what he's witnessed describe larvae of fruit fly *D. hydei* dining on already dead larvae of other insects. What Vijendravarma reported is not just feeding on a happenstance free lunch, but hunting as well. He showed close-up videos of the dark, pronged mouthparts of a smaller larva scraping again and again against the wide, cream-colored body of a larger one. Finally the big larva's body rips open, exposing softer flesh. He also showed photographs of clusters of small larvae side-by-side with their mouths against the flesh of a much larger one.

Those larger larvae represent the final stage for fruit flies before metamorphosis. After a voracious race to eat and grow as much as possible, the blobby almost-adults lumber off to go through metamorphosis, which turns a pale wormy cylinder of a larva into a winged adult. That last larval phase appears to be a food bonus worth stalking, Vijendravarma said. In a feeding test, more than a third of the younger larvae survived by eating nothing but the older ones. ■

NORMAN CHAN/SHUTTERSTOCK

"If you don't know how something works, you can't really be certain that you understand how it could have evolved." —**BOB MONTGOMERIE**

Trout nose cells sniff magnetism

Tissue in fish's snout could explain animals' compasses

By Devin Powell

Cells plucked from a trout's snout can swivel like tiny compasses to line up with a nearby magnet. That sensitivity, credited to iron inside the cells, could explain how fish, birds and other animals sense Earth's magnetic field — a long-standing mystery among biologists.

"For decades scientists have been searching for the cells responsible for magnetosensation," says neuroscientist David Keays of the Research Institute of Molecular Pathology in Vienna. "They're the biological equivalent of the elusive Higgs boson."

The first demonstration of an animal's internal compass dates to nearly half a century ago, when experiments showed that caged robins turn when exposed to rotating magnetic fields. Other birds, as well as sea turtles and some fish and amphibians, share this ability.

But the specific body structures behind the sense — which humans either lack or aren't aware of — have remained elusive. Magnetic fields easily penetrate flesh, so receptors that respond to them could be hidden anywhere in the body.

Recent clues have pointed to nose tissue as the place to look. In fish, magnetic fields can stimulate brain cells that connect to the nasal cavity, as neuroscientist Michael Walker of the University of Auckland in New Zealand and colleagues have demonstrated. His team also found crystals of the magnetic mineral magnetite in nasal tissue from yellowfin tuna.

In the new study, reported online July 9 in the *Proceedings of the National Academy of Sciences*, Michael Winklhofer of the University of Munich and colleagues broke apart olfactory tissue from rainbow trout and bombarded free-floating cells with magnetic fields.

About one to four of every 10,000 cells responded, spinning in a tight embrace with the rotating magnetic fields. A closer look revealed chains of magnetite glued inside each cell's membrane. Like a magnetized compass needle, the iron-rich mineral guided the cell around.

In living tissue, cells aren't free to spin in this fashion. But the magnetite's push could open up pores in a cell's membrane. Charged particles moving in and out could set off electrical impulses, stimulating the brain. To support this theory, the researchers are looking for the movement of charged calcium in living cells.

Meanwhile, Keays plans to search for magnetic cells in pigeons.

Environment

"People may very well remember this as a year of extreme weather and climate." —JESSICA BLUNDEN

Recent extreme weather attributed to human-caused climate warming

Probability of some droughts, heat waves now much greater

By Janet Raloff

Texans sweltered through the hottest, driest spring and summer on record last year. Much of the blame can be attributed to a recurring climate pattern known as La Niña, which emerges every few years as surface waters chill in the eastern equatorial Pacific. But Earth's steadily warming climate contributed as well, a new analysis concludes.

Since the 1960s, the likelihood of Texas seeing extremely hot, dry weather in a La Niña year has mushroomed 20-fold due to human-induced global warming, David Rupp of Oregon State University in Corvallis and his colleagues calculate.

The researchers were one team among six international groups probing climate's link to extreme events in late 2010 through 2011. The collected findings appear July 10 in the *Bulletin of the American Meteorological Society*.

"People may very well remember this as a year of extreme weather and climate," says Jessica Blunden of the National Climatic Data Center and an editor of *State of the Climate in 2011*, a report published as a supplement to the July 10 *Bulletin of the American Meteorological Society*.

Severe food shortages gripped the Horn of Africa last year after drought left the land parched from winter 2010 through the following spring. La Niña played a role there, too. And computer analyses of global climate conditions since 1979 find that a recent warming of surface waters in the Indian and Pacific oceans has destabilized La Niña weather patterns. Chris Funk of the U.S. Geological Survey in Santa Barbara, Calif., concludes that this process probably intensified 2011's drought in East Africa.

Other teams pointed to global warming as a likely contributor to excessive heat in central Europe last summer and to unusually balmy temperatures in central England in November 2011. In the British case, that kind of heat could be expected to recur every 20 years now — a 62-fold increase over the 1960s.

Yet global warming can't be blamed for all monster weather. Unprecedented flooding that submerged large tracts of northern Thailand, including its capital, for up to two months last year resulted from rainfall at an intensity the region had encountered before. But water management practices and heavy industrialization of a floodplain slowed drainage last year.

These new analyses are pioneering efforts to get near real-time assessments of climate's role in extreme weather events, says climatologist Thomas Peterson of the National Climatic Data Center, in Asheville, N.C.

Benbrook Lake near Fort Worth, Texas, dried up last year during the state's warmest August on record. A new analysis links global warming with increases in extreme weather.

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For years, he says, climate scientists have argued that although global warming can increase the frequency of extreme weather, they couldn't pin any particular event on human-caused climate change. That appears to be changing, Peterson and his colleagues argue.

Using a developing field known as "attribution science," researchers are beginning to apply massive computing capacity to explore how global temperatures, surface reflectivity and moisture patterns can affect the odds of localized extreme weather events.

In 2011, droughts beyond Africa and Texas brought billions of dollars in crop losses, Blunden says. The North Atlantic saw above-average hurricane activity (19 named storms, compared with an average of 12), and seven separate U.S. tornado outbreaks that each wreaked more than $1 billion in damage.

Polar regions racked up their own extremes, says Martin Jeffries of the University of Alaska Fairbanks. Barrow, Alaska, sustained a record 86 days in a row when the minimum air temperature failed to dip below freezing.

Understanding global warming's role in extreme events extends well beyond blaming rights. Peterson notes that water managers may need to change policies if evidence begins pointing to persistent changes in the recurrence rates and lengths of droughts or the frequency of heavy rains.

Right now, linking these events is difficult, usually works only for events lasting longer than a month and can take a year to complete. Peterson's team hopes to see the science mature to the point that assessments might be turned around more quickly and to tackle events lasting mere days. ■

Humans

"The ancient Maya used their calendar to promote continuity and stability rather than predict apocalypse." —MARCELLO CANUTO

Apocalypse not written in stone

Newfound Maya glyphs don't treat 2012 as an end year

By Bruce Bower

Hieroglyphs carved on this 1,300-year-old Maya stairstep mention December 21, 2012, apparently as part of a nearby king's efforts to shore up his waning power.

Although hieroglyphs previously found at an ancient Maya site may or may not mention December 21, 2012, as the end of time, don't cancel any New Year's Eve plans. Scientists working at the remains of another Maya city have uncovered another reference to the same 2012 date, and the writing on the wall — make that the staircase — concerns political turmoil back then, not apocalypse now.

Anthropologists who discovered the 2012 reference among carvings on 22 stone steps at Guatemala's La Corona site announced the find June 28 in Guatemala City.

"The ancient Maya used their calendar to promote continuity and stability rather than predict apocalypse," says excavation codirector Marcello Canuto of Tulane University in New Orleans.

On one staircase block, anthropologist David Stuart of the University of Texas at Austin, who led the decipherment of the text, recognized a commemoration of a visit in 696 to La Corona by the ruler of Calakmul, a Maya site in what's now southern Mexico. Long thought to have been killed or captured in a 695 battle lost to a rival kingdom, the Calakmul king apparently weathered that defeat and visited his allies at La Corona to convince them that he remained a strong ruler.

In the commemoration, the Calakmul king refers to himself with a title signifying that he presided over and celebrated the end of a key Maya calendar cycle in 692. To attribute special status to his weakened reign, Stuart says, the king also connects himself to a future date when the next calendar cycle would conclude — December 21, 2012.

To a Maya king stung by a military setback, "the reference to 2012 might even have provided a comforting sense of inevitability" in his continued rule, remarks anthropologist Stephen Houston of Brown University in Providence, R.I.

Earliest American tools not all alike

Oregon finds hint Clovis hunters weren't alone in New World

By Bruce Bower

Ancient residents of four caves in south-central Oregon may deserve bragging rights as the earliest known North Americans. New finds in the caves unveil a population that reached the New World around the same time or shortly before the famed Clovis hunters who roamed the Great Plains and the Southeast at the end of the Ice Age.

Regardless of who first set foot in North America, populations practicing at least two spear-making styles colonized the New World, say archaeologist Dennis Jenkins of the University of Oregon in Eugene and his colleagues.

"The emerging picture is of multiple movements from Asia into the Americas by populations with multiple tool traditions," says archaeologist James Adovasio of Mercyhurst College in Erie, Pa.

Excavations at Oregon's Paisley Caves find that four distinctively shaped stone spearheads representing a toolmaking style called the Western Stemmed Tradition date to between 14,000 and 13,000 years ago, Jenkins and his colleagues report in the July 13 *Science*.

"We seem to have two different toolmaking traditions that coexisted in North America and did not blend for hundreds of years," Jenkins says.

Some investigators have held that Clovis-style implements were precursors of Western Stemmed points. In support of that scenario, a Wyoming site previously yielded Clovis points in deposits below Western Stemmed points.

But Western Stemmed points at the Paisley Caves are at least as old as Clovis points that have been found elsewhere in Oregon, Jenkins says.

At the Paisley Caves, Jenkins' team obtained 121 new radiocarbon dates, in addition to 69 previous radiocarbon dates, for sagebrush twigs, dried feces and animal bones unearthed above and below stone artifacts. The radiocarbon measures obtained by Jenkins' team provide "reliable ages that put Western Stemmed points contemporaneous with Clovis points," says anthropologist David Meltzer of Southern Methodist University in Dallas.

D. STUART

Genes & Cells

36,542 Number of protein-coding banana genes

22,333 Number of protein-coding human genes

Space trek cuts signs of aging

Worm study shows gene changes after 11-day orbit

By Tina Hesman Saey

In space they can barely see you age — if you're a worm.

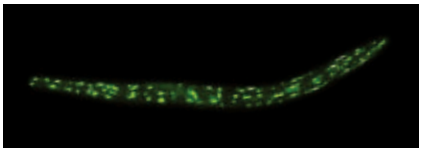
Tiny, transparent nematodes that spent a gravity-free 11 days — equivalent to about 16 years for a person — en route to and aboard the International Space Station appeared to age much more slowly than earthbound worms, Yoko Honda of the Tokyo Metropolitan Institute of Gerontology and colleagues report online July 5 in *Scientific Reports*.

The result is the opposite of what some scientists expected, based on experience with human spaceflight and studies of other animals. Mammals, including people, are under physiological stress in the microgravity of space, says D. Marshall Porterfield, director of NASA's Space Life and Physical Sciences Research and Applications Division in Washington, D.C. In low gravity, muscles atrophy and aging accelerates.

While the space station worms, from the species *Caenorhabditis elegans*, may have been under stress, they didn't have those side effects. Their muscles did not degrade, and clumps of aging-related proteins known as Q35 aggregates did not build up in them as much as in worms on the ground, indicating that worms don't age as fast in space as on Earth. Worms that visited the space station were frozen immediately after returning to Earth, so the researchers weren't able to test whether time in space enabled the critters to live longer.

The researchers also discovered that relative to ground-based nematodes, the spacefaring worms had lower activity of 199 genes, including 11 genes involved in transmitting information through the nervous or endocrine systems. For seven of the 11 genes, mutations that lowered the genes' activity also caused ground-based worms in a separate experiment to live longer.

Reduced activity of three of the life-extending genes — called *gar-3*, *cha-1* and *shk-1* — also lowered the number of Q35 clumps that built up in aging worms. Those genes encode proteins that are produced in the nervous system, and two of them encode proteins that are also made in muscles.



Tiny worms that spent time in space (like the one shown here) have fewer clumps of aging-related proteins (green) than worms that stayed on the ground. That could mean that worms live longer in microgravity.

Lowering the levels of those proteins during spaceflight might affect how worms perceive their environment, leading the nematodes to reduce their metabolism and extend their life spans, says Catharine Conley, NASA's planetary protection officer. Conley helped develop the system the worms lived in while in space.

Studying worms in space may help scientists learn more about how low gravity affects organisms, regardless of the impact on life span, Porterfield says. "It doesn't really matter what the outcome is if we learn about the biophysical environment," he says. That knowledge may help engineers design ways of better protecting the health of astronauts. ■

Genome of a fruit besieged

The banana genome has been unpeeled. The genetic makeup of *Musa acuminata* (top), a fertile banana species that gave rise to the seedless Cavendish (bottom) and other clonal varieties people eat today, sheds light on the plant's evolutionary history and ripening process. This information may also help researchers boost the crop's resistance to fungal and viral pathogens threatening its survival. *M. acuminata* has 36,542 protein-encoding genes and 235 genes that make small snippets of protein-regulating RNA, called microRNAs, an international team of researchers reports online July 11 in *Nature*. The team also found the genetic remains of a pathogen called the banana streak virus scattered on 10 of the banana's 11 chromosomes. "It's probably a footprint of an attack that was successful, but the banana somehow overcame it," says Thomas Givnish, a plant ecologist and evolutionary biologist at the University of Wisconsin–Madison. —*Tina Hesman Saey*

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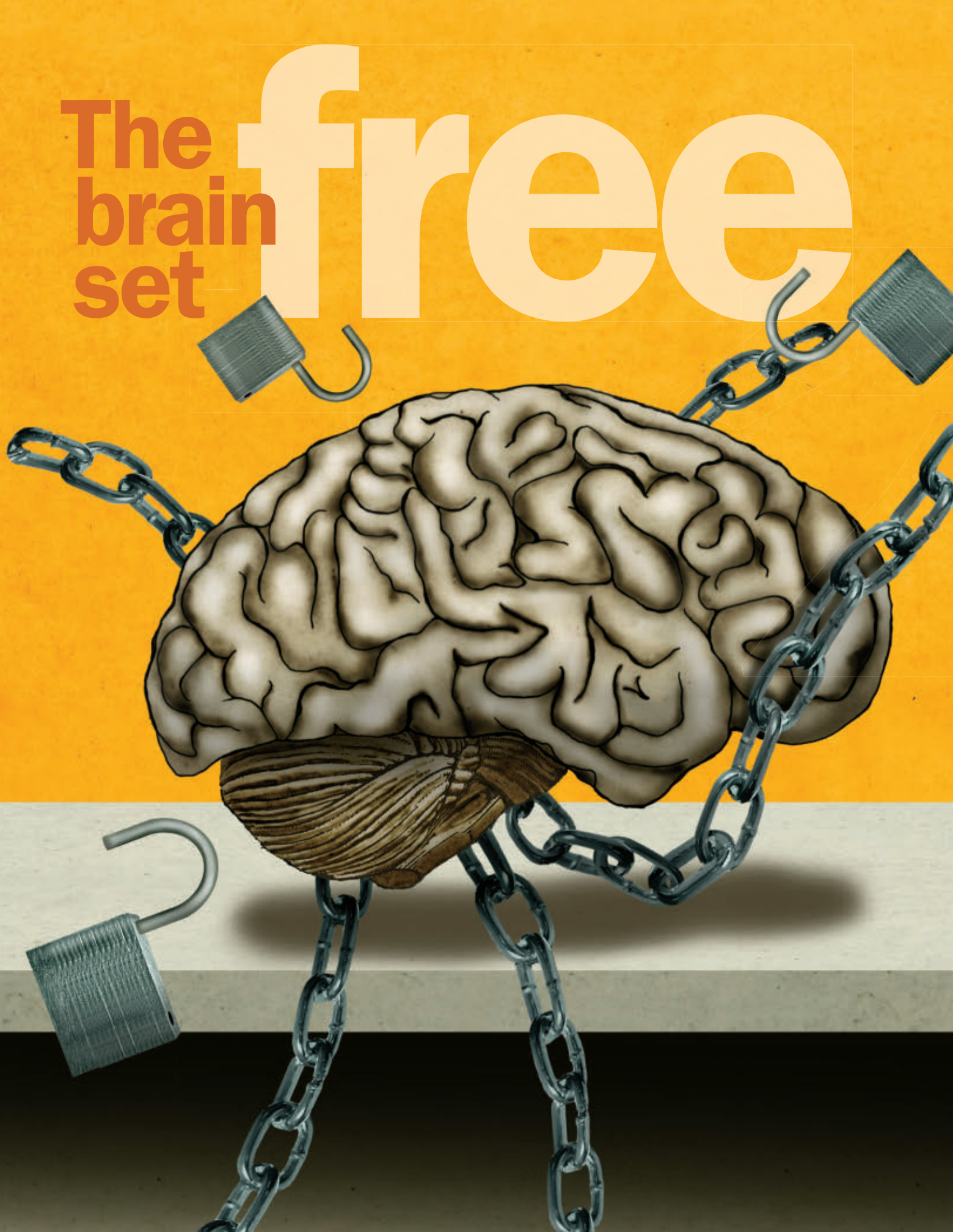


Lifting neural constraints could turn back time, making way for youthful flexibility

By Laura Sanders

A baby's brain is a thirsty sponge, slurping up words, figuring out faces and learning which foods are good and bad to eat. Information about the world flooding into a young brain begins to carve out traces, like rushing water over soft limestone. As the outside world sculpts the growing brain, important connections between nerve cells become strong rivers, while smaller unused tributaries quietly disappear.

In time, these brain connections crystallize, forming indelible patterns etched into marble. Impressionable brain systems that allowed a child to easily learn a language, for instance, go away, abandoned for the speed and strength that come with rigidity. In a fully set brain, signals fly around effortlessly, making commonplace tasks short work. A master of efficiency, the adult brain loses the exuberance of childhood.

But the adult brain need not remain in this petrified state. In a feat of neural alchemy, the brain can morph from marble back to limestone.

The potential for this metamorphosis has galvanized scientists, who now talk about a mind with the power to remake itself. In the last few years, researchers have found ways to soften the stone, recapturing some of the lost magic of a young brain.

"There's been a very, very significant change," says Richard Davidson of the University of Wisconsin–Madison. "I don't think the import of that basic fact has fully expressed itself."

Though this research is still in its early stages, studies suggest techniques that dissolve structures that pin brain cells in place, interrupt molecular stop signals and tweak the rush of nerve cell activity can restore the brain's youthful glow. Scientists are already attempting to reverse brain rigidity, boosting what's known as "plasticity" in people with a vision disorder once thought to be irreversible in adults.

These efforts are not an exercise in neural vanity. A malleable brain, researchers hope, can heal after a stroke, combat the decline in vision that comes with old age and perhaps even repair a severed spinal cord. An end to childhood — and the prodigal learning that comes with it — does not need to eliminate the brain's capacity for change. "There are still windows of opportunity out there," says neuroscientist Daphné Bavelier of the University of Rochester in New York. "It may require a little more work to open them, though."

Prying at windows

Research aimed at restoring the brain's youthful flexibility is leading to a more nuanced view of findings from the 1960s. In experiments that won them the Nobel Prize in physiology or medicine, David Hubel and Torsten Wiesel discovered that sealing shut a kitten's eye for a period during the early stage of life would leave the animal unable to see normally out of that eye. If the opposite eye were then patched, forcing the underdeveloped eye to work, the kitten could recover, scientists later discovered. This patch fix worked only on a young animal, suggesting there was a finite window of time during which the brain could rewire itself.

Humans have this window of opportunity, too. During what scientists call a "critical period," nerve cells in the brain can forge new connections, sprouting tendrils that carry messages to other cells. Children with amblyopia, a condition in which one eye is weaker than the other, can be cured with a patch over the strong eye, which forces the brain to rewire incoming information from the stunted eye. In adulthood, the exact same treatment is useless. By figuring out why this period ends, and why other forms of flexibility are also lost, researchers think they might be able to bring the brain's healing power back.

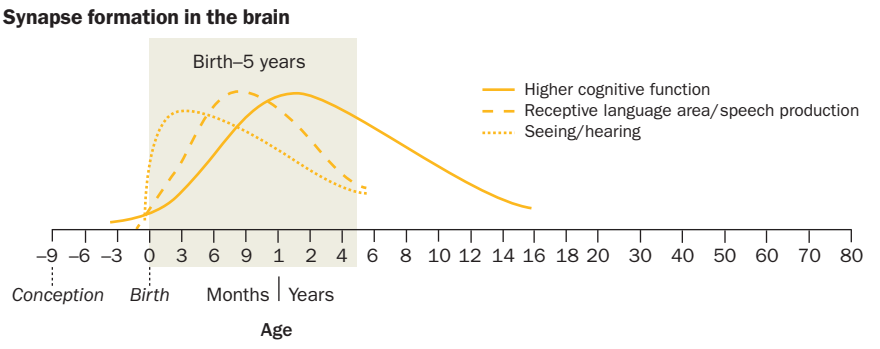
Some of the more obvious players ushering in an end to brain plasticity are structures that literally pin nerve cells in place. One is a tight mesh straitjacket — with the texture of cartilage — that surrounds a nerve cell and restricts the formation of new connections in the brain. Called perineuronal nets, these webs show up early in life to stabilize most nerve cells in the brain and spinal cord.

Manipulating these perineuronal nets might be a way to "return people to a childlike state," in which rapid learning or recovery can happen, says neuroscientist James Fawcett of the University of Cambridge in England.

Unlike in Hubel and Wiesel's animals, whose visual deficits were locked in once

Periods with potential Different functions in the brain, such as thinking and seeing, develop during varying time windows, as nerve cells form new connections called synapses. Though scientists used to think such development had clear peaks and then waned with age (shown), evidence now suggests substantial flexibility can be restored in adulthood.

SOURCE: CENTER ON THE DEVELOPING CHILD/HARVARD UNIV.



FACING PAGE: MICHAEL MORGENSTERN; THIS PAGE: T. DUBÉ

adulthood arrived, the visual systems of mice with abnormal or missing perineuronal nets retained the ability to be sculpted, Fawcett and colleagues have found. These nets may hem in neurons by calling in particular molecules, perhaps ones that stymie new nerve cell connections, Fawcett and Difei Wang, also at Cambridge, wrote in the July *Cell and Tissue Research*.

Another impediment to malleability comes from a fatty substance called myelin, which winds around neurons' information-sending axons like insulation around an electrical wire, speeding messages along. With this speed comes less flexibility, as the myelin holds nerve cell fibers in place. Wresting myelin off of nerve cells restores plasticity in mice, neuroscientist Takao Hensch at Boston Children's Hospital and colleagues have shown.

Besides its physical constraints, myelin also releases repressive signals. One, a protein called ephrin-B3, holds axons back, Stephen Strittmatter of Yale School of Medicine and colleagues reported in the March 27 *Proceedings of the National Academy of Sciences*. Removing ephrin-B3 allowed axons to grow much more than those in normal mice after an injury.

Other myelin-related proteins are known to squash new nerve cell connections. One is the downer protein NoGo. When a neuron detects NoGo, it kicks off a series of changes that prevent the growth of new connections. If NoGo detector proteins are eliminated, nerve cells become extra active and primed for growth.

Already, results from various studies have pushed scientists to stop talking about cut-and-dry "critical" periods, but rather, "sensitive" ones. The brain can be coaxed into changing, even in adulthood.

Behavior revisited

While some researchers are overcoming physical barriers that swaddle nerve cells and stunt new growth, others recognize an easier path to malleability: manipulating nerve cell behavior to make cells more or less likely to fire off messages. "Changing the structure is hard, but changing function is possible," says vision scientist Dennis Levi of the University of California, Berkeley.

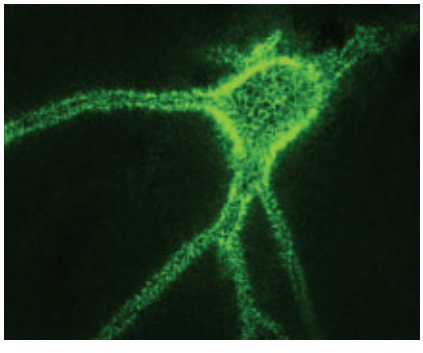
Rather than relocating the concrete walls of a stone canal, the functional approach alters the speed of water moving through that canal. One architecture can sustain either a rushing stream or a trickle.

Hensch and his colleagues started with a hunt for substances in the brain that were scarce during early life but abundant as brain wiring windows closed. The protein lynx1 popped out. (Its molecular makeup resembles an active molecule in snake venom.) Mice genetically engineered to lack lynx1 spontaneously recover from early vision problems, retaining a malleable brain long into adulthood, Hensch and his team reported in *Science* in 2010.

Normally, lynx1 puts a damper on certain nerve cells' excitability, a job that helps the brain maintain the proper flow rate of nerve cell activity. In a balancing act, the brain is poised between too much activity (excited) and too little (inhibited). By muffling certain cells, lynx1 holds the adult brain to a status quo, called the excitatory/inhibitory balance. But lose lynx1, and the brain shifts toward a more excited, and more malleable, state.

"You can pursue all of the molecules," Hensch says. "What's exciting is that they all converge on the excitatory/inhibitory balance."

A cartilage-like net (green) envelops a nerve cell, restricting the formation of new connections. Loosening the grip could boost brain flexibility.



A. DITYATEV AND M. SCHACHNER/*NATURE REVIEWS NEUROSCIENCE* 2003

Neuroscientist Alessandro Sale of the Institute of Neuroscience CNR in Italy thinks that this balancing act may explain many of his team's results in adult rats. Over the last several years, Sale and colleagues have reported a growing number of situations that can repattern the adult rat's visual system: Exercise, living in a stimulating environment, starvation and even doses of Prozac, which caused certain nerve cells to become more active, all reinstated a brain with more youthful behavior.

"At the very beginning, I was surprised that many different noninvasive strategies were able to elicit plasticity in the adult brain in such a powerful way," Sale says. But after looking closely, his team believes that the procedures all alter the flood of messages that nerve cells send.

Although many of these detailed experiments in animals test the visual system, the same general principles might underlie other brain systems, Sale says. Of course, the real goal of this work is not to make blind rats see again, but to help people retrain their brains.

Help for humans

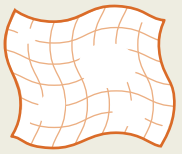
Preliminary studies in people hint that the excitatory/inhibitory balance is important for many aspects of a healthy brain. People with Down syndrome, Alzheimer's disease and even spinal cord injuries may have out-of-whack balances, studies suggest. Though there's no really good way to see how nerve cells in a live human brain behave, some training techniques (like those used by Sale in rats) do appear to resculpt the adult mind.

In some ways, the idea that experiences shape the brain is obvious to anyone who has ever learned anything. Playing the guitar, leisurely swinging golf clubs and driving a taxi in London for years all mold the adult brain, some more dramatically than others. Just two hours of playing a racing video game changed the structure of volunteers' brains, researchers reported in the March 22 *Neuron*. Similar processes are at the core of products that promise to boost cognitive powers (though many of

Roads to resculpting

A variety of strategies have been shown to reverse the eye condition known as amblyopia in adult rats. Scientists hope some of these same approaches could work in human adults, reinstating a youthful, flexible state that allows for healing.

Dark exposure
Paradoxically, animals kept in complete darkness are able to regain some sight during adulthood, presumably through a shift of nerve cell behavior.



Net breakdown
Using a compound called chondroitinase ABC to dissolve components of perineuronal nets, which hold nerve cells in place, improves visual acuity.

Calorie restriction
Although it's not clear how it works, a short-term bout of severe calorie-cutting seems to reset the brain's clock and allow recovery from amblyopia.

Drugs
An SSRI used to treat depression, fluoxetine (brand name Prozac) can reactivate adult brain plasticity in rodents, leading to a full recovery of vision.

Environment enrichment
Living in a place with ample time for exercise, toys and friends boosts the brain's malleability in rats.

these brain-training regimes have yet to be validated).

"We are continuously being exposed to the environment, and those things that impinge upon us are continuously shaping our brains," Davidson says. So the question isn't so much whether something can change the brain, but rather, how people can take charge of the process for a desired outcome.

Studies by Levi and his collaborators have found that a certain kind of vision practice can actually help adults see better. Hours of difficult vision training, in which people had to discern hazy lines, for instance, improved vision in people with amblyopia, normal vision and even normal age-related vision decline. People could see sharper images, detect contrasts better and even read small letters faster.

Older people's eyes still sent blurry information to their brain, but the brains were better equipped to handle it, Levi and colleagues wrote in *Scientific Reports*. Levi believes that the improvement comes from a training-enhanced ability to pay attention to relevant information and ignore the blurred distractions.

There may be a more fun way. Data from Bavelier support a counterintuitive idea: Mindless video games actually make the brain sharper. Action-packed video games like *Call of Duty 2* seem to enhance people's perception, and not just in ways required to get a high score. "You get benefits that ripple," Bavelier says.

After logging hours at the games, people were better at nimbly switching between two demanding jobs and quickly determining whether a number is even or odd. The results, reported in May in *Computers in Human Behavior*, suggest that this kind of brain training reopens a window. "What really changes is the ability to locate resources and ignore distractions," Bavelier says.

Flexibility too far

Efforts toward restoring plasticity hold great promise, but only when targeted to hit particular brain systems. If not, the potential benefits come with some strong caveats. "I'm very scared of taking off the brakes," Hensch says. "They're there for a reason."

Myelin insulation on nerve cells, for instance, is damaged in patients with multiple sclerosis. That damage affects the conduction of nerve impulses, leading to the hallmark symptoms of the disease.

In Hensch's lab, the mice genetically engineered to lack lynx1 protein show dementia-like damage in their brains earlier than their normal counterparts. "After nine months, you see holes in cortex," Hensch says. Parts of the brain that are damaged earliest are the most malleable throughout life. "Maybe the price we pay for plasticity is the susceptibility to neurodegeneration."

Brakes may also help the brain know what not to learn. Extremely proficient musicians have reported a rare condition in which neural pathways become so shaped by playing music that the performers' brains fuse the control regions for separate fingers, and the ability to cleanly pluck a single string is lost.

Likewise, too much malleability could play a role in inappropriate fear or anxiety. It would be bad if people learned to associate a strong emotional response with every negative situation encountered in daily life. "If our brains exhibited plasticity for all of those kinds of cues, it would likely elicit anxiety disorders in the entire population," says Davidson.

Researchers attribute such concerns to what they call the double-edged sword of plasticity: "A system capable of such flexible reorganization harbors the risk of unwanted change," Alvaro Pascual-Leone of Harvard Medical School and colleagues wrote in 2005 in *Annual Reviews of Neuroscience*.

For now, scientists are proceeding cautiously, still in the discovery phase. But their studies may ultimately lead to ways to let the right signals rush in and sculpt a readily accepting brain. For the first time, Davidson says, "we can actually take more responsibility for the shaping of our own brains." ■

Explore more

- D. Maurer and T. Hensch. "Amblyopia: Background to the special issue on stroke recovery." *Developmental Psychobiology*. April 2012.

RAT IMAGE: BASEL101658/SHUTTERSTOCK; ICONS: T. DUBÉ

Glassy, resilient bone-capping cartilage has long eluded tissue engineers trying to grow it in the lab.

BIOPHOTO ASSOC./GETTY IMAGES

CARTILAGE CREATION

New joint tissue could keep people moving, reducing need for knee or hip replacements

By Nathan Seppa

Cartilage, the shock absorber of the body, has been bearing the brunt of a modern lifestyle.

This nerveless connective tissue allows bone to glide over bone without any repercussions — most of the time. But human cartilage evolved in an earlier age, in ancestors who lived shorter lives, carried less body weight and roamed an unpaved world. Nowadays, cartilage takes a constant and prolonged beating from which it has poor capacity to bounce back.

It's not a good scenario for an aging population. As the go-between tissue in joints, cartilage can handle only so many jolts and jars before something has to give. When the daily grind wears the tissue down, or it gets damaged by more abrupt injury, the bones' nerve cells become exposed. Movement can lead to a painful zing, the hallmark of osteoarthritis, which now affects more than 27 million people in the United States. In addition to pain, osteoarthritis shows up as stiff joints, cracking sounds, inflammation and bone spurs.

What's worse, when it comes to cartilage damage, there is no safety net. Cartilage manufacture depends entirely on one type of cell, chondrocytes, and these tiny cartilage factories multiply less and less with advancing age. They fall behind in making new cartilage to repair defects and sometimes respond to injury or long-term

pounding by giving up and dying off.

No wonder some orthopedists consider cartilage regeneration the holy grail of their field. Confronted with hordes of gimpy people living longer lives, orthopedists have used surgery to clean out damaged joints, braces to stabilize a wobbly gait and artificial knees and hips to replace damaged bone ends, a last resort against osteoarthritis. Everything short of new cartilage.

27 million
Number of American adults affected by osteoarthritis

50 percent
Chance of getting osteoarthritis during your lifetime

But now, with the help of stem cells, a new generation of bioengineers are coming close to cracking the code for cartilage regrowth. Stem cells have yet to choose a career path, a characteristic that makes them attractive future cartilage-creators. The blue sky version of the stem cell approach goes like this: Stem cells are extracted from a patient, geared up to become chondrocytes, wrapped in a favorable mix of compounds and then inserted into damaged joints. The cells take it from there. Voilà: neo-cartilage.

Scientists have recently pinpointed prominent proteins needed to keep stem cells on track to becoming cartilage-making chondrocytes and have even devised nanosized polymer scaffolds on which these stem cells can start growing cartilage. The cells seem to behave better if surrounded by molecules found naturally in healthy cartilage, and some research suggests scaffolding derived from cartilage itself might deliver much-needed biochemical prompts.

While still in the experimental phase, the stem cell strategy is gaining ground: More than a dozen clinical trials using stem cells as cartilage regenerators are under way or planned in Norway, Spain, Iran, Malaysia, France and elsewhere.

Tissue engineering and cartilage regeneration were at "point zero" in the 1970s, says Wan-Ju Li, a tissue engineer at the University of Wisconsin–Madison. "Now the technology is more mature. All together, the field is getting very rich and very interesting."

Getting glassy

Researchers aren't interested in making just any cartilage. They want the kind that caps the ends of long bones, such as those in the legs. This type, called hyaline cartilage, is distinct from the bendable kind in the ears or the fibrocartilage found between vertebrae. Hyaline cartilage is slippery, glassy, elastic and smooth. Picture the tough gristle at the rounded end of a ham bone. It is everything humans would want to cap a bone in a weight-bearing joint, allowing them to move about like pain-free machines.

But past attempts to repair hyaline cartilage through regeneration have come up short. For decades, the surgical approach for a damaged joint has been to clean out frayed cartilage and, sometimes, drill tiny holes into the worn ends of the bone. The holes allow blood and stem cells from bone marrow to leak out and patch the injury.

In theory that should work, but the stem cells seeping through the holes lack focus. "Those stem cells that come out are confused," says John Sandy, a biochemist at Rush University Medical Center in Chicago. "They're not getting the right signals.... So they hit the middle road." They make fibrocartilage, a poor substitute for hyaline. In a recent study, only two-thirds of athletes receiving this "microfracture surgery" following injury showed good results, and only half maintained their original level of play for several years.

Doctors have also transplanted living chondrocytes onto worn-out bone ends. Genzyme, a biotech company based in Cambridge, Mass., offers an off-the-shelf kit for this procedure. It requires taking thousands of live chondrocytes from healthy cartilage elsewhere in the body, culturing the cells in a dish to expand their numbers and packaging them with other products for insertion into the trouble spot. Called autologous chondrocyte implantation, the procedure has outperformed microfracture surgery in some studies, but some patients need follow-up surgery and a nine-year study of implantation patients found that 30 percent didn't improve. The trouble may arise because mature chondrocytes lose their ability to produce cartilage if expanded through too many generations, Li says.

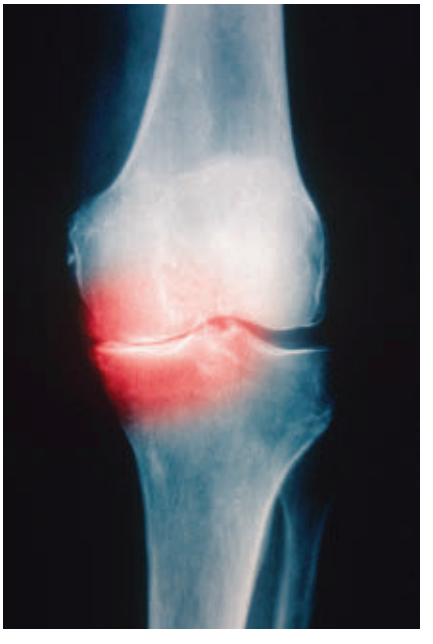
Stem cells have an advantage there. Like newly hired employees, they should have plenty of productive years in store. Key for scientists is finding a reliable way to teach these blank slate cells to become hyaline-producing chondrocytes.

Cellular prompting

Scientists can round up starter stem cells from all over the adult body. Those found in connective tissues such as cartilage, tendons and the synovial membrane that forms a sac enveloping joints have the potential to make good chondrocytes. But so might some less-obvious choices, such as stem cells from bone marrow, fat tissue and discarded umbilical cords.

Before sending them into the fray,

When cartilage wears away, bone rubs on bone (as shown in this color-enhanced X-ray of a 76-year-old man's knee). Such rubbing is a sure sign of budding osteoarthritis.



SCOTT CAMAZINE/PHOTO RESEARCHERS, INC.

though, scientists need to give the cells every possible advantage, nudging them toward chondrocytic behavior. In the lab, the cells are typically mixed with a brew of natural chondrocyte-promoting compounds and then seeded onto a scaffold that encourages growth and cartilage production. The seeded scaffolding is later inserted into a cartilage defect in a patient.

An optimal recipe for the cell-directing brew is still a mystery, but scientists have identified several possible ingredients.

Essential to the mix are compounds called growth factors. The best-studied is TGF-beta, which is good at jump-starting a stem cell to act like a chondrocyte and produce cartilage, Sandy says. But TGF-beta can't work alone; relying too heavily on TGF-beta, for example, can steer a stem cell toward making fibrous tissue, rather than the resilient hyaline cartilage, he says.

Recent research has focused on another growth factor called FGF-2. Li and Wisconsin colleague Andrew Handorf reported last year in *PLoS ONE* that treating stem cells with FGF-2 primed them to become hyaline-making chondrocytes.

FGF-2 activates a compound called Sox9 in the stem cell, which in turn switches on the production of two main components of cartilage, type 2 collagen and aggrecan, Li says. FGF-2 might be best used before cell differentiation, the point at which a stem cell becomes a mature cell with a specific role, he says. Then other growth factors, including TGF-beta, could push the cartilage-making process along. A 2010 review lists a dozen growth factors that affect stem cells' ability to differentiate into chondrocytes.

But these growth factors must be wielded carefully. Ming Pei, an orthopedic surgeon and cell biologist at West Virginia University in Morgantown, says even FGF-2 can cause the resulting chondrocytes to swell in size and swing toward bone making. His team has found that combining FGF-2 with certain booster proteins, as well as cartilage tissue with its own chondrocytes removed, helps increase the numbers of stem cells in the lab and, later, keeps the cells on task.

Another booster compound called kartogenin can steer stem cells directly toward making components of hyaline cartilage, researchers report in the May 11 *Science* (*SN: 5/5/12, p. 10*). Kartogenin inhibits a protein called filamin A in the stem cells, an action that unleashes other compounds that switch on genes that ultimately trigger cartilage creation.

This molecule shows promise, say Joan Marini of the National Institutes of Health in Bethesda, Md., and Antonella Forlino of the University of Pavia in Italy. "Stimulating the differentiation of one's own stem cells by means of an easily deliverable chemical compound would be more advantageous than using conventional drilling and microfracture techniques," they write in the June 28 *New England Journal of Medicine*.

A protein called vimentin takes a different route to reach the same objective. To differentiate into a chondrocyte, a stem cell must take on a round shape, says Rocky Tuan, a tissue engineer at the University of Pittsburgh. He and his colleagues found that vimentin nudges bone marrow stem cells toward becoming rounded like chondrocytes. Extra vimentin also boosts genes instrumental in making type 2 collagen, Tuan's team reported in 2010 in the *Journal of Cellular Biochemistry*.

The blend of compounds required to create a good hyaline-making chondrocyte may ultimately hinge on the choice of stem cell itself, Pei says. He proposes that stem cells derived from the synovial membrane have an advantage by already coming from a joint. In fact, synovial stem cells manufacture a substance, a type of matrix, that seems particularly valuable.

Pei's team mixed the matrix made by synovial stem cells with FGF-2 in a low-oxygen environment. That combination, when added to other synovial stem cells, enabled those cells to ramp up their numbers. It also provided them with a favorable "niche," a microenvironment amenable to chondrocyte formation, the researchers reported last year in *Tissue Engineering, Part A*.

Optimizing these conditions improves the niche and helps the stem cells thrive, in part because they are free of stress. Pei's approach limits reactive oxygen species, unstable molecules that damage tissues, keeping the stem cells comfortable. Growing outside of niches, Pei says, "stem cells bear the stresses of the environment and lose their proliferation capacity. They become old."

A place to reside

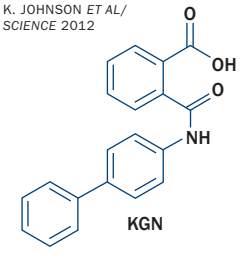
Efforts to expand stem cell numbers and steer them to become chondrocytes go for naught if the cells can't hold together long enough to form a cartilage patch. A stable home would be a replica of what scientists call chondrocytes' extracellular matrix, the elastic web of cartilage naturally surrounding them in the body.

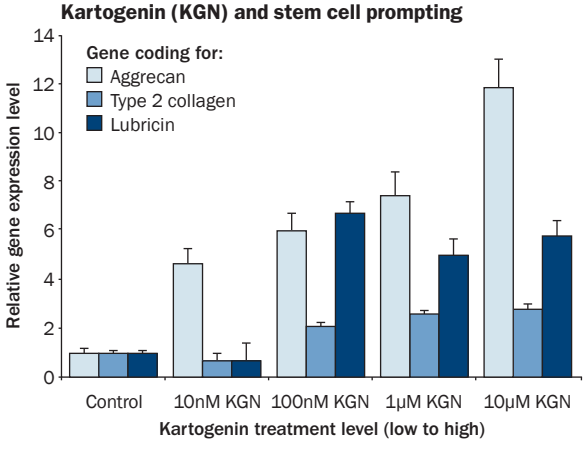
About a decade ago, Li and Tuan

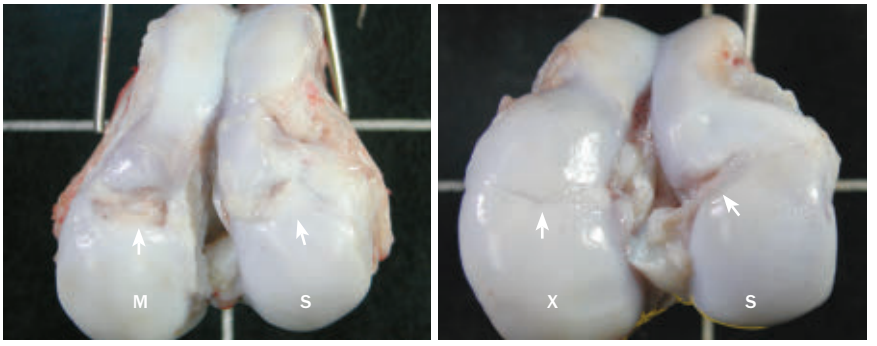
Giving directions

The molecule kartogenin (shown below) revs up genes that code for cartilage proteins, notably aggrecan, type 2 collagen and lubricin (right).

SOURCE: K. JOHNSON *ET AL*/ *SCIENCE* 2012







For the pigs By implanting biodegradable polymer scaffolding seeded with stem cells, researchers were able to regrow lost cartilage in the knee of a pig. Six months after the damage (arrows), the joint treated with the scaffold and stem cell combination (X) showed better cartilage regrowth than did a joint treated with scaffolding seeded with mature chondrocytes (M) or joints with scaffolding alone (S).

became interested in building a synthetic scaffold resembling the naturally occurring one, onto which they could seed stem cells. While still at the National Institutes of Health, they used a process called electrospinning to cast nanofibers of polymer into a structure resembling cartilaginous matrix. Those first webs have since been improved and made into a biodegradable cartilage scaffold.

"It's easy to fabricate and there's no batch-to-batch difference," Pei says.

Li notes that the polymer, built to degrade after six to 12 months in the body, has strong mechanical properties that keep stem cells together, giving the cells time to weave their own matrix of cartilage.

In 2009, Tuan and Li tested their scaffold, seeding it with human stem cells to create a patch that was then inserted into pigs with cartilage damage in their knees. Some pigs received scaffolding seeded with mature chondrocytes. The researchers allowed the pigs, with some restrictions, to put weight on the knees almost immediately, since routine compression is the norm for cartilage.

After six months, the stem cells had grown into chondrocytes that made hyaline cartilage that outperformed the fibrocartilage made by the mature chondrocytes.

"It was glassy cartilage," Li says, "with good mechanical properties."

In an alternative approach, other researchers are testing the scaffold potential of a cartilage matrix obtained from cadavers. The scientists remove the cells and use the rest. Though that work is in its early stages, Pei says the natural matrix offers some useful biological cues for stem cells that a polymer doesn't.

Still other teams are instead using a natural adhesive called fibrin glue as scaffolding material. Doctors at Cairo University combined bone marrow stem cells with fibrin glue and blood platelets — a healing aid that produces the growth factor TGF-beta — and placed the mix in the cartilage-damaged knees of five patients. A year later, all reported improvement.

Regardless of exactly which blend of boosters and scaffolding work best, Li is optimistic that a stem-cell strategy, once fully researched, will succeed in making cartilage where aging chondrocytes have failed. "We believe we can do better," he says.

Early adapters

Once that goal is achieved, one of the big challenges will be to determine which patients would benefit most from the treatment. Much osteoarthritis seems to unfold over a lifetime, but many patients develop it from hyaline cartilage damage traceable to an injury. If cartilage regeneration reaches the clinic, treating these patients early could prevent further loss of cartilage and prevent osteoarthritis from overtaking the joint, says David Felson, a rheumatologist at Boston University School of Medicine.

Cartilage is made to handle stress and compression, but every tissue has its limits, and knee injury increases by sixfold the likelihood that a person will develop osteoarthritis. Felson says his team has found evidence that injuries to the knee structures "probably account for a great majority of osteoarthritis."

But early detection isn't easy since many people ignore or underestimate their injuries. Years can pass before X-rays and other scans show two bones rubbing together, a sign of painful cartilage loss and budding osteoarthritis.

Carla Scanzello, a rheumatologist at Rush University Medical Center, and colleagues reported last year that inflammatory molecules that gradually destroy cartilage flood an injured joint and leave a telltale signature long before symptoms of osteoarthritis arise. Understanding these markers of inflammation "might help us target patients," she says.

For the moment, that targeting would involve anti-inflammatory drugs to limit the cartilage degradation. But in the future, a stem cell treatment might dramatically reduce the number of people who end up needing a joint replacement, says Dobrila Nesic, a molecular biologist at the University of Bern in Switzerland.

An artificial knee or hip can last 10 to 15 years, Nesic says, and a person can get two (per joint) in a lifetime. "If you're 60, no problem," she says. "If you're 40, your lifetime before wheelchair is 20 to 30 years."

Researchers agree more work is needed to bring stem cell–based cartilage regeneration to the clinic. Li suspects the technical problems might be solved in the next five years or so, with another five years needed to sort out regulatory and insurance issues.

He has students who are surgeons, still busy learning how to replace knees and hips. "I was joking with them," Li says, "saying, 'You guys are going to have to find a new job soon.'" ■

Explore more

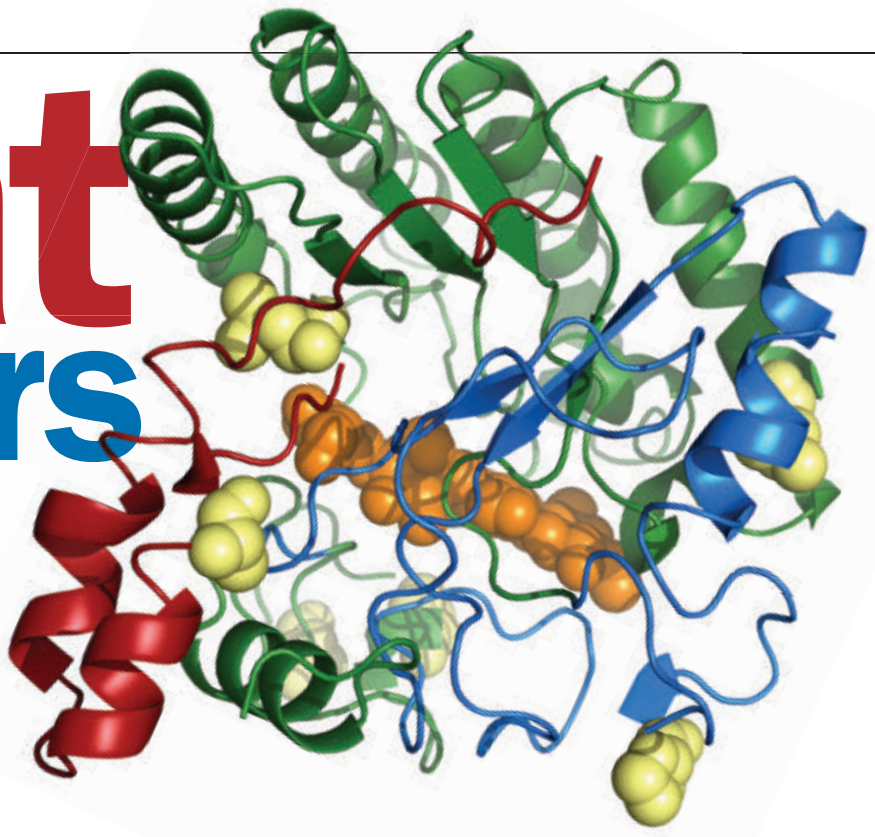
- R.F. Loeser *et al*. "Osteoarthritis: A disease of the joint as an organ." *Arthritis & Rheumatism*. June 2012.

W.-J. LI *ET AL./ TISSUE ENG REGEN MED* 2009

Heat Beaters

Scientists seek enzymes that don't mind working at high temperatures

By Rebecca Cheung



Even when the heat is on, an enzyme designed and created in the lab (ribbon structure shown) can break down plant material (sugar in orange).

INDIRA WU

In the middle of a cattle ranch near Gerlach, Nev., enclosed by a corrugated metal fence, are small pools of steaming water. Close to the surface of these pools, water temperatures reach about 90° Celsius; deeper down, it's even hotter. Landowners have sectioned off the area around the pools and installed an overflow pipe to keep the water from seeping out and harming livestock or people.

Despite the dangers involved, one September day Joel Graham leaned over the barrier and plunged in his gloved hand to scoop up sediment and water samples.

At this locale, the biologist from the University of Maryland, Baltimore was fishing for microorganisms that can thrive at very high temperatures. Such extremophiles contain reaction-driving proteins called enzymes unlike any found in less harrowing environs.

Enzymes show up in all living things — from the simplest microbes to humans — and are essential for survival. They do jobs such as digesting food and helping to make sure genetic material gets properly copied. While a lot of known enzymes live and work best around the temperature of the human body, Graham and colleagues are among a cadre of scientists looking for new enzymes that can function when conditions get superheated.

The collective hope? That such enzymes could be co-opted to perform industrial processes. In many of the world's factories, setting the thermostat on high keeps reactions moving quickly and prevents bacteria and other microbes from contaminating or eating up desired products. Papermaking, textile manufacturing and some food processing might all benefit from the help of heat-loving enzymes.

Graham's team is particularly interested in a class of enzymes that help break down the grassy parts of plants, an area where researchers have made recent progress. Finding such enzymes, and employing them in large quantities, could lead to biofuel production on an industrial scale, helping society reduce its dependence on fossil fuels.

Some scientists are hoping a newly discovered enzyme pulled from the field may be an ideal high-temperature grass muncher. Other researchers, instead of getting their hands dirty in hot pools, are designing never-before-seen heat-loving enzymes in the lab. Many lab enzymes are in some way inspired by versions already found in nature.

"The environment contains a lot of solutions to a lot of human problems," Graham says. "If you find a directed way to go after what you're looking for, it's actually not that hard to find something new and useful."

Enzyme-palooza

People have been using enzymes to perform chemical reactions outside the body for millennia. Food processing with enzymes, for example, dates back at least 4,000 years, when people stored milk in the excised stomachs of animals. Digestive enzymes in an animal's gut caused milk to curdle, forming other dairy products like curds and whey.

Today, a cocktail of various proteins help coagulate milk to make solid curds for cheese. Other enzymes soften cotton during textile manufacturing or treat paper in the pulp industry. Enzymes

even lend a hand in the synthesis of drugs and supplements such as vitamin E.

But enzyme-driven processes these days can get much more involved than just throwing starting materials into an animal gut. Often materials need to undergo additional reactions, treatment with a solvent or dyeing with a chemical, before or after an enzyme can do its job. For many of these reactions, heat is required as a catalyst.

The problem: Most known enzymes work only at conditions matching those of the organism from which they came. So temperatures have to be repeatedly ramped up and lowered at various stages of a multistep process. "You have to cool it down for the biological part and heat it back up for the next step," says Vicki Thompson of Idaho National Laboratory in Idaho Falls. "That makes the process more expensive, and it's wasteful of energy."

During low-temperature stages, materials can also be vulnerable to attack from a plethora of microbes.

The breakdown of long chains of glucose molecules from plants, or cellulose, faces such troubles. Cellulose stored in corn stover — the grassy part of corn that people don't eat and a potential biofuel source — is tied up in complex chemical arrangements, making it hard for enzymes working alone to get access.

So the biomass has to be mashed up a bit first. It's heated and treated with corrosive chemicals like acids or salty solutions, which expose the cellulose. But the plant products have to be neutralized and cooled before a collection of room-temperature enzymes can digest the material into usable sugars.

Cellulose-breaking enzymes, called cellulases, made by organisms that normally thrive under extreme conditions, in places like hot springs or hydrothermal vents, could offer a solution.

"You can take those enzymes and use them under the industrial conditions that you are interested in," explains Thompson, who has been studying organisms that live in extreme environments and the proteins they make for more than a decade.

Pulled from a pool

Biologist Thomas Brock unearthed one of the first known extremophiles from hot pools in Yellowstone National Park in Wyoming in the 1960s. Later named *Thermus aquaticus*, the bacterium thrived best at temperatures around 70° C. This finding suggested life could exist in all kinds of places that were previously thought of as dead zones.

Explorations in volcanic soil, hot vents, deep seas and salt deserts have turned up thousands of extremophiles since, says Thompson. A sizable portion love the heat. Notably, a heat-loving, DNA-building enzyme from *T. aquaticus* has been a major boon for genetic engineering and forensics. To analyze DNA from collected samples, scientists need large amounts of uncontaminated copies. By simply adding the enzyme, Taq polymerase, to a starting strand of DNA and other genetic ingredients, scientists can make lots of DNA copies with no contamination to worry about.

Over the last two decades, Frank Robb of the University of Maryland, Baltimore has scoured some of the hottest corners of the Earth — from the hot springs at Yellowstone to the deep-sea vents of the Okinawa Trough — in search of interesting heat-loving enzymes. An ability to break down cellulose is one sought-after skill in the "help wanted" ads.

Robb and Graham want cellulases that can do their thing at temperatures of 100° C or even higher. The more types the better, since some enzymes specialize in cutting the cellulose into chunks and some break it down even further into glucose.

Recently, after examining genetic material from samples collected at the hot springs in Nevada, Robb and Graham turned up an enzyme called EBI-244 that can perform one of the early cellulose breakdown steps at temperatures as high as 109° C. Though other heat-loving cellulases have previously been found, none do the job as well as EBI-244 at such high temperatures.

"Nobody ever thought or had seen an enzyme that worked at those temperatures, and it's so stable at those temperatures," says Isaac Cann of the University of Illinois at Urbana-Champaign, who also studies cellulases and their role in biofuels production.

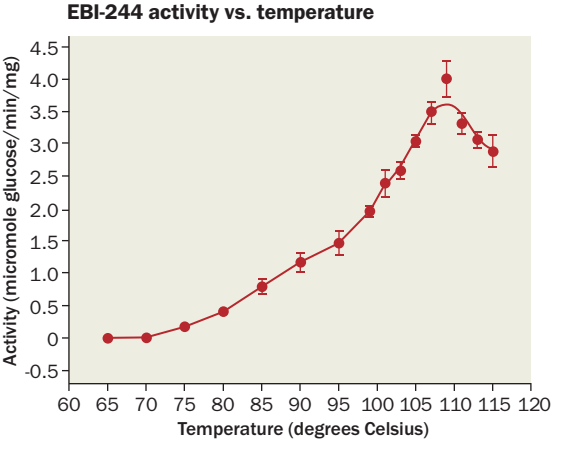
And the enzyme, described in *Nature Communications* last year, can also survive in acidic conditions and put up with harsh solvents.

Researchers can't collect nearly enough heat-loving cellulases in the field to run a cellulose-degrading factory (kilogram amounts are needed). Instead, scientists typically hook up the genetic material that codes for a specific enzyme to a bacterium's or yeast's molecular machinery, tricking the organism into producing the protein. But for many enzymes, EBI-244 included, it has been a challenge.

"It's just a painstaking and tedious process to get suitable quantities," says Douglas Clark, a biochemical engineer at the University of California, Berkeley

J. GRAHAM *ET AL*/*NATURE COMMUNICATIONS* 2011

Fueled by heat From a hot pool in Nevada (above), researchers pulled a cellulose-busting enzyme called EBI-244. The enzyme shows its maximum breakdown activity above 100° Celsius (right), making it a good candidate for use in biofuel production.



SOURCE: J. GRAHAM *ET AL*/*NATURE COMMUNICATIONS* 2011

who collaborated with Robb and Graham in identifying EBI-244.

Despite the barriers, the discovery of EBI-244 is exciting because of what scientists can learn from it. The DNA that codes for the enzyme is unlike the DNA for any known cellulase; it was traced back to a never-before-seen extremophile. "It's very strange to find one that doesn't fit into a well-defined category," Graham says. "That was the most exciting part."

The next challenge will be to study the enzyme's physical structure, to unlock its heat-loving secrets.

"We would love to know more about it," says Clark. "Could we begin to use the information gained from studying this enzyme to engineer not only other cellulases, but other proteins?"

Into the lab

Rather than hunt for enzymes that can function under extreme conditions in the field, chemical engineer Frances Arnold of Caltech makes her own. She brings the principles of breeding to the lab: guiding the development of new and better proteins using known proteins as parents.

Arnold's lab starts off by using a computer program to put together stretches of amino acids, the building blocks of proteins, from different enzymes. By mixing and matching chunks from each parent, the team's computer software figures out what types of children would emerge if these enzymes could "breed." The idea is to pick parents with different features — like ones that thrive in high heat, or can grow up in large quantities — to produce a child with the best of all the qualities.

> "No natural enzyme will fulfill all the requirements of your desired applications."
> FRANCES ARNOLD

Arnold and colleagues mine the results, looking for prospective children with the most promising futures — that is, with proteins likely to perform interesting functions and assemble into a desired shape in the lab. The children can then undergo small-scale changes that make them even better.

"You're better off making it in the laboratory, because no natural enzyme will fulfill all the requirements of your desired applications," says Arnold.

Arnold produces her computer-based cellulases with the help of simple organisms in the lab. She doesn't try to grow huge loads of her heat-loving cellulases, but she does start with proteins from filamentous fungi, which are known to grow up easily.

After a recent cellulase "breeding," Arnold's team reported making new enzymes that could work at about 70° C. Some of the parent enzymes could break down cellulose only at temperatures around 57° C. Since that report, Arnold has been able to coax more proteins to work at similar temperatures.

She has since sold licenses for making these cellulose-breaking prodigies to private companies, though she doesn't yet know whether the enzymes are being used for large-scale plant munching.

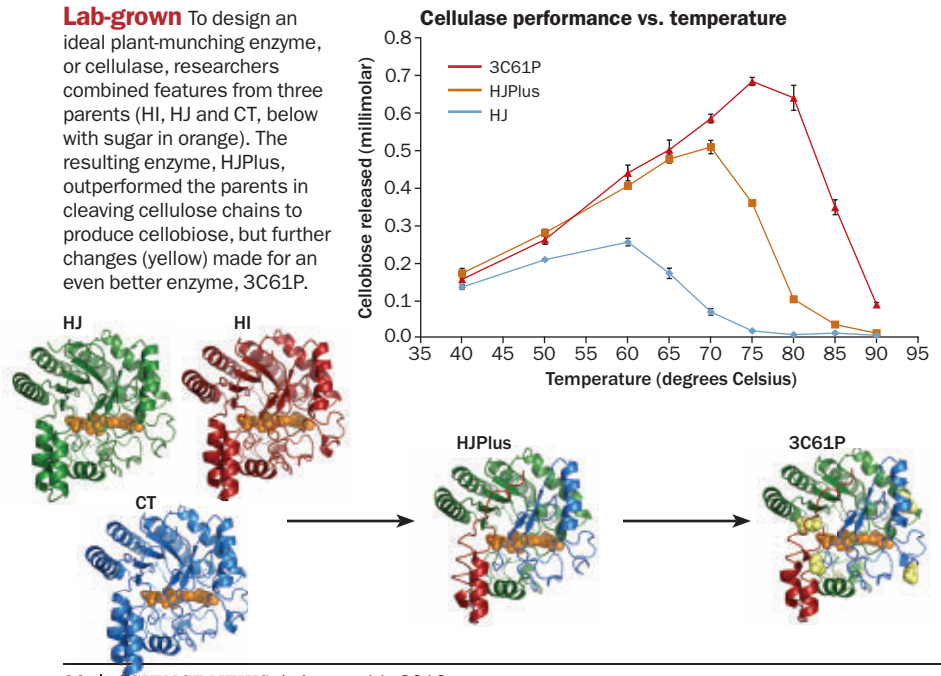
Despite the recent breakthroughs, there's a lot of work to be done before heat-loving enzymes can usher in an era of cheap biofuels.

Individual proteins, whether unearthed from hot zones or made in the lab, will have to come together as members of a cellulose-converting team. Once many different heat-loving enzymes are found, scientists need to get them to work well as a group.

"Cellulases are a particularly hard problem because it's not just one enzyme. It's a whole family of enzymes. If you improve one component, it does not mean you have a better product," Arnold says. "You have to improve many components and then find the right mixture of those components. It's a challenging thing."

Once the right team is found, scientists still have to overcome the hurdle of inexpensively making a lot of proteins in the right ratios. The same hurdle will probably hold for enzymes in other heat-dependent processes.

"We haven't yet figured out all the rules for making highly stable enzymes that also do what they need to do and can be produced in an economical fashion," Arnold says. "We're trying." ■



Lab-grown To design an ideal plant-munching enzyme, or cellulase, researchers combined features from three parents (HI, HJ and CT, below with sugar in orange). The resulting enzyme, HJPlus, outperformed the parents in cleaving cellulose chains to produce cellobiose, but further changes (yellow) made for an even better enzyme, 3C61P.

Explore more

■ Visit the Arnold group's website: bit.ly/FArnold

Rebecca Cheung is a former intern at Science News.

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Rabid: A Cultural History of the World's Most Diabolical Virus

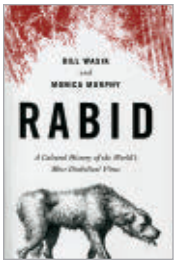
Bill Wasik and Monica Murphy

A modern list of most-hated viruses might include ones that have triggered recent pandemics: HIV, or the lethal H1N1 swine flu. Wasik and Murphy are here to say such a list shows people today have far too short a memory.

Historically, no other virus can hold a candle to the cultural and medical terror of rabies, this husband-and-wife team argues. And it's hard to disagree with the litany of horrors they roll out in their blood-flecked history: the infections, the symptoms, the deaths.

Wasik and Murphy don't go into the science of rabies in depth. But they excel at chronicling the bizarre, especially when it comes to rabid links with man's best friend. One blue-blooded lady, they report, contracted rabies and died after her infected lapdog licked her on the chin. Pliny the Elder thought a maggot from any dead canine, inserted into a wound, would cure the disease. A 19th century quack doctor suggested that dogs spontaneously generated rabies if they didn't have enough sex — a concept that took off in 1830s England and seriously set back attempts to develop modern treatments.

Not until the 1880s did Louis Pasteur's research team develop a vaccine that lifted what had been a death sentence for anyone infected. Today,



more than 55,000 people still die from rabies each year, most in Africa and Asia, but the disease holds far less sway over popular and medical imaginations.

Still, the authors close their book with a well-timed reminder that deadly viruses can pop up among unsuspecting populations. On Bali, an island once deemed rabies-free, a single infected dog brought by a sailor in 2008 triggered a regional epidemic that the government struggled to control.

HIV, swine flu, rabies: Killers all that won't go away. — *Alexandra Witze*
Viking, 2012, 275 p., $25.95

The Everett Interpretation of Quantum Mechanics

Jeffrey A. Barrett and Peter Byrne, eds.

A collection of original documents, many hard to find, relating to one of the most controversial of the many interpretations of quantum mechanics. *Princeton Univ., 2012, 389 p., $75*

What a Plant Knows

Daniel Chamovitz

Plants have senses too, a biologist shows. Though they can't hear Chopin they do have ways to essentially touch, see and taste the world around them. *Scientific American/Farrar, Straus and Giroux, 2012, 173 p., $23*

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SCIENCE BOOKS FOR YOUNG READERS

A Black Hole is Not a Hole

Carolyn Cinami DeCristofano

There's plenty for both kids and adults to learn in this colorful look at the discovery of black holes and what scientists know about them today. *Charlesbridge, 2012, 74 p., $18.95, ages 9–12*

The Book of Blood

H.P. Newquist

Stories about blood — from ancient bloodletting to modern medicine — take advantage of kids' fascination for the gross and explain science at the same time. *Houghton Mifflin Harcourt, 2012, 152 p., $17.99, ages 10–14*

The Mighty Mars Rovers

Elizabeth Rusch

As part of a series called Scientists in the Field, this book about the rovers Spirit and Opportunity is told through the eyes of rover lead scientist Steven Squyres. *Houghton Mifflin Harcourt, 2012, 80 p., $18.99, ages 10–14*

Where Do Mountains Come From, Momma?

Catherine Weyerhaeuser Morley

Get a little help answering an age-old question of kids, plus read about volcanoes, erosion and more in this book for younger readers. *Mountain Press, 2012, 32 p., $12, ages 4–8*

It's Raining Fish and Spiders

Bill Evans

An Emmy-winning meteorologist explains weather mysteries through experiments, stories and real-world weather data. *Forge, 2012, 240 p., $18.99, grades 2–5*

The Ultimate Book of Saturday Science

Neil A. Downie

A scientist updates the home-experiment genre with original projects chosen because they are both interesting and "spectacular," including hovercraft and electric sundials. *Princeton Univ., 2012, 546 p., $29.95, ages 8 and up*

Galactic collisions explained

Perhaps you can explain why Andromeda and the Milky Way are going to collide ("Milky Way will be hit head-on," *SN: 7/14/12, p. 10*). Galaxies, as is always written, are rushing away from each other at ever-increasing speeds. How do things collide when there is never anything to collide with? Either galaxies are rushing outward in all directions with the same impetus from the point of the Big Bang, or they are not.
Bruce Smith, via e-mail

Galaxies do indeed collide with one another even though the universe is expanding at an ever-accelerating rate. In the case of the Milky Way and Andromeda, the two galaxies are massive enough and close enough that they are gravitationally attracted to one another. So they are headed for a collision even as both galaxies continue to be carried along on the flow of the expanding universe. You can think of it like two boats running into one another in the middle of a river; they collide even though both are also being carried downstream by the current. —Alexandra Witze

Clarifications

The high energy (100 billion billion electron volts) of a particle mentioned in "Chasing a cosmic engine" (*SN: 7/14/12, p. 16*) refers to the particle's energy when it hit the atmosphere, not when it reached detectors on the ground. The article should have said that in 1962, the particle slammed into the atmosphere, creating a shower of particles that rained down on an array of detectors in Volcano Ranch, N.M.

The image of human papillomavirus shown in "Catching a cancer" (*SN: 7/28/12, p. 22*) is a photo illustration based on a micrograph and shows a single virus particle at multiple magnifications.

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

To see some of Rosemary Mosco's science comics, visit **www.sciencenews.org/comics**

Rosemary Mosco's comic strips feature her favorite inspirations from nature and science.

Comic strip science

"I am so awesome." [Smug grin.]

So goes the final frame in a humorous comic called "Birds are Gross," in which artist and field naturalist Rosemary Mosco highlights the virtues of the turkey vulture. The bird, speaking throughout ("I am a turkey vulture. Yes indeed."), reaches this conclusion after announcing its proclivities for things like projectile vomiting and poop-mediated temperature control.

"I like showing people animals that aren't especially appealing, and then highlighting what's really neat about them," says Mosco, 31, of her "Bird and Moon" comic strip series. "Lately, I'm really into herpetology. I live skinks and salamanders. Salamanders, I think, are really, really unappreciated."

Mosco's interests sometimes follow her home, which is currently in Boston. "We have a little rescued corn snake," she says, in a nod to the resident whose diet has turned her freezer into a scary place: "Half of it is frozen vegetables, half of it is frozen mice."

A native of Ottawa, Mosco has been living a half-and-half life for as long as she can remember, tugged by both art and science. As a kid, she would venture outdoors for some quality snake-seeking, then return home and "write stories and terrible kids' books." In college, Mosco recalls marching into her adviser's office and asking if she could major in both science and art. "He looked at me like I was crazy and said 'No.' So I wound up doing anthropology."

Now, though, the schism is resolved. Mosco is blending her science background — she has a master's degree from the University of Vermont's field naturalist program — with her artistic endeavors. She's worked for nonprofit organizations, the U.S. National Park Service and public radio, helping to explain climate change and other issues. But explaining science through art, especially in just a few words, can be nerve-racking. "The science can be really complicated," she says.

Mosco says it's worth it, though, and enjoys knowing that her projects can make a difference. Once, a nonprofit printed her vulture comic on a T-shirt and sent Mosco a photo of a woman wearing the shirt and holding a huge turkey vulture. "It had a cute name, like Ed or something," Mosco says. "I got choked up." — *Nadia Drake*

A classic method for remembering birdcalls using similar-sounding words gets a new twist in Rosemary Mosco's guide to the songs and calls of eastern North American birds.

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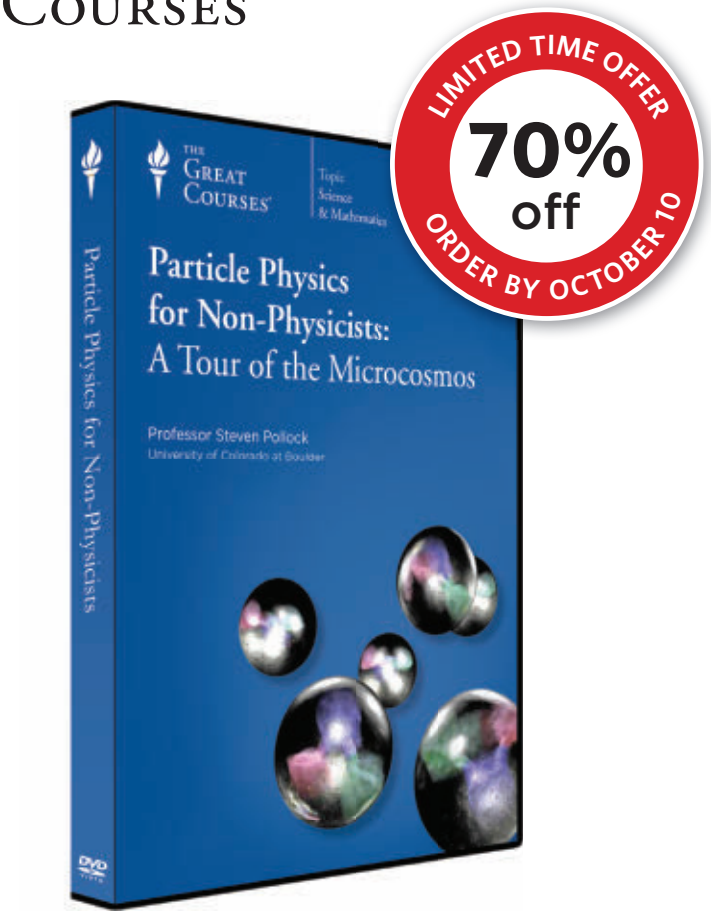

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