SN
SCIENCE NEWS MAGAZINE
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MAY 3, 2014

Mysterious Side of Antibiotics

A Step Toward Synthetic Cells

Subsurface Sea on Enceladus

Why Zebras Have Stripes

FUNGAL FIX

Mycologists close taxonomic loophole

Actual size is 40.6 mm

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Departments

COVER The outward form of a fungus, be it a cup, a toadstool or mere fuzz, can be deceptive to taxonomists. DNA is clarifying the identity of many species. *rujithai/Shutterstock*

New tools reveal new truths about fungi, flies, antibiotics

In the newsroom, any story about a new scientific method faces an uphill battle. Editors are likely to reject such a story; writers themselves often downplay these stories because they've learned that the answer is usually "no." To those of us who follow science, how scientists do what they do becomes important, and thus worth writing about, only once a new method reveals a novel truth about nature. We are after the "what," not necessarily the "how" (although we always make a point to summarize how a discovery is made).

In this issue, hidden in plain sight, are a number of stories that feature in one way or another new — or newly applied — methods, what the tools reveal about the world and, perhaps even more interesting to watchers of human nature, the many aftershocks new tools can create.

The study of fungal forms, for example, is an old science. But genetic tools have enabled new, more precise ways of seeing the organisms. As Susan Milius relates on Page 22, DNA studies can discern fungal species with more accuracy than a magnifying lens or microscope ever allowed. The tools have also created a challenge for mycologists, who are faced with the daunting prospect of changing their entire naming system, reclassifying many species and renaming thousands of fungal types.

A combination of genetic, computational and optical tools has enabled scientists to control select neurons in fruit flies, producing the advance described in the lead news story on Page 6. Laura Sanders writes that scientists have been able to link a suite of 29 behaviors in fly larvae to specific groups of nerve cells that produce each wiggle, roll and scooch. On Page 7, Tina Hesman Saey reports on how a new application of genetic engineering tools has created the first synthetic chromosome in a yeast cell. The lab-made chromosome seems to function as well as any of the yeast's own. And on Page 18, Beth Mole describes how the relatively young field of systems biology and its methods have created a ruckus by suggesting a new way that common antibiotics may kill microbes — an idea still hotly contested in the scientific literature.

Sometimes a new "how" does make for an exciting what.

— *Eva Emerson, Editor in Chief*

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Excerpt from the May 2, 1964, issue of *Science News Letter*

50 YEARS AGO

Animated Movies Made by Computer

A 17-minute animated movie has been produced, using a cathode ray tube and a movie camera, both controlled automatically by an electronic computer.... The film took two months of research and programming, four hours of computer time, and 2,000 hours of film processing, [at] a cost of about $600 per minute.... The picture is formed on a grid ... 184 spots long by 252 spots high. Each spot can be any of several degrees of brightness.... The computer might be told to draw certain objects. Then it would be told the laws controlling their behavior, and from these it would illustrate the objects' actions.... The use of color might be possible, though no research is being done in that area.

UPDATE: The 2013 computer-animated film *Frozen* cost about $1.5 million per minute to make. To simulate snow's properties for the movie, mathematicians helped develop a technique called a material point simulator that realistically accounts for snow's mix of fluid and solid properties.

THE SCIENCE LIFE

Cosmology's winter warrior

As the engineer in charge of maintaining the BICEP2 telescope, Steffen Richter made frequent trips to fill the instrument with helium to cool it to nearly absolute zero.

HOW BIZARRE

One giant leap for zit-causing microbes

Around 7,000 years ago, a bacterium that lives on humans and causes acne leaped to a very different host: domesticated grapevines. Since then, an essential DNA-repair gene in the microbe, *Propionibacterium acnes*, has mutated and no longer functions. Without the gene, the microbe is unable to function on its own and appears to rely on the grapevine for these DNA repairs. This is the first report of such a symbiosis between a plant and a microbe that's typically associated with animals, says microbiologist Andrea Campisano of Italy's Edmund Mach Foundation. The researchers named the grapevine version of the microbe *P. acnes* type Zappae, in a nod to both musician Frank Zappa and the microbe's agricultural significance; *zappa* means *hoe* in Italian. The work was reported in February in *Molecular Biology and Evolution*. While this may be the first recorded human-to-plant transfer of this type, it's not the first time Zappa has been honored Linnean-style. At least four organisms, including a spider and a mudskipper, bear his name. — *Annette Heist*

FROM TOP: S. RICHTER; ANDERS LJUNGBERG/FLICKR (CC BY 2.0)

Listen to an interview with Steffen Richter at **bit.ly/SNrichter**

The wind chill is −80° Celsius in Antarctica when Steffen Richter, donning a red parka, hops on a snowmobile and heads to work. Lighting his path are the stars, a sliver of moon and the faint green glow of the aurora australis, the southern lights.

It's the kind of day that might make him miss home — but Boston is nearly 15,000 kilometers away, and no pilot would dare fly anywhere near Richter's location for months. Plus, the Harvard engineer has a job to do. Hitched to Richter's snowmobile is a vat of liquid helium, the lifeblood of a telescope built to detect and dissect the universe's oldest light.

On March 17, scientists at the Harvard-Smithsonian Center for Astrophysics announced that the telescope, BICEP2, had detected ripples in spacetime dating back to a trillionth of a trillionth of a trillionth of a second after the Big Bang (*SN: 4/5/14, p. 6*). It's a potentially Nobel Prize–winning discovery, and it could not have been made without Richter. His daily maintenance checks and semiweekly helium deliveries during three consecutive Antarctic winters allowed BICEP2 to remain fixated on exposing the earliest moments of the universe.

Even in the South Pole's frigid temperatures, the telescope must be kept much colder to detect radiation, emitted just after the Big Bang, that hovers just a few degrees above absolute zero. Missing one delivery of liquid helium coolant could cripple the telescope for weeks. "You have to do it no matter what the weather is," he says. "Running out of liquid helium is not an option."

The Amundsen–Scott research station, where Richter works, is inaccessible by air for nearly nine months of the year. Once the sun dips below the horizon in March, it doesn't return until September, leaving a frigid, dry environment that's ideal for astronomy but abysmal for human habitation.

That doesn't bother Richter, an adventurer whose passions include riding motorbikes in remote parts of the world. In his nine winters at the polar station (which puts him in a tie for the most spent there), he has served as the only line of engineering defense for multimillion-dollar experiments such as BICEP and IceCube, which recently detected neutrinos from beyond the solar system (*SN: 12/28/13, p. 6*). Every day, he trekked out in temperatures averaging −58° Celsius to inspect the instruments and their data. In his free time, Richter became a proud member of the 300 Club, running outside naked in −100° Fahrenheit temperatures after roasting in a 200° sauna.

"Running out of liquid helium is not an option."
STEFFEN RICHTER

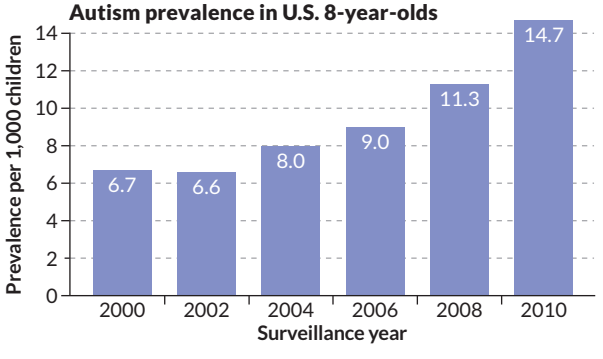
He's now back in Boston and plans to return to the South Pole later this year to install upgrades for the new and improved BICEP3 telescope. But he won't spend the winter — the team found another willing victim to take his place. "I have high confidence in him," Richter says of his stand-in. "Hopefully he'll be great so that I won't have to do it every year." — *Andrew Grant*

SCIENCE STATS

What's behind rising autism rates

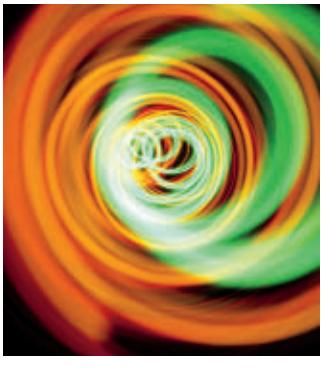
The number of 8-year-olds with an autism spectrum disorder rose from 1 in 88 (or 11.3 per 1,000) in 2008 to 1 in 68 (14.7 per 1,000) in 2010, the U.S. Centers for Disease Control and Prevention estimates. An uptick in diagnoses, perhaps due to better detection, may explain the increase and the regional differences seen. Alabama's autism rate is just 5.7 per 1,000, while New Jersey's is 22 per 1,000.

SOURCE: U.S. CDC



THE –EST

Fastest-spinning molecules



The powerful kick of a laser has spun molecules faster than they've ever been spun before: 10 trillion rotations per second, or 600 trillion RPM. A car with tires turning at that spin rate would travel the distance to the nearest star in half an hour. "There's definitely nothing macroscopic that can spin that fast," says Valery Milner, a molecular physicist at the University of British Columbia who led the study published March 19 in *Physical Review Letters*. The feat required a precisely sculpted, 100-trillionths-of-a-second laser pulse that coaxed a batch of neutral molecules of oxygen and nitrogen to polarize and spin, all in the same direction. Milner says the technique could allow scientists to use rotational stress to sever molecular bonds and create custom chemical reactions. — *Andrew Grant*

CLOCKWISE FROM TOP: CYNTHIA CHANG; CREATIVITY103/FLICKR (CC BY 2.0); E. OTWELL

BODY & BRAIN

Neurons linked to specific behaviors

New view of larval fly brain reveals functions of particular cells

BY LAURA SANDERS

By scrutinizing the twists, turns, wiggles and squirms of 37,780 fruit fly larvae, neuroscientists have created an unprecedented view of how brain cells create behavior. The results, published March 27 in *Science*, draw direct connections between neurons and specific movements.

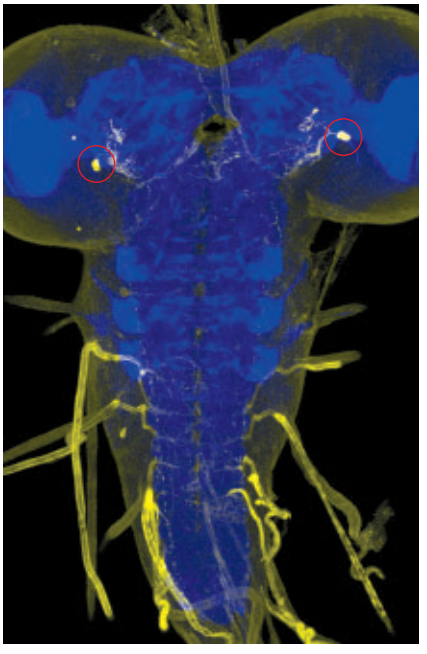
"Understanding how neural activity gives rise to behavior is the most important question in neuroscience," says MIT neuroscientist Kay Tye, who was not involved in the research. The new study provides a way for scientists to start answering that question, she says. "I think this is a really important approach that's going to be very influential."

Scientists led by Marta Zlatic of the Howard Hughes Medical Institute's Janelia Farm Research Campus in Ashburn, Va., took advantage of an existing set of specially mutated flies. In each animal, small groups of neurons, usually between two and 15 cells, were engineered to respond to blue light. By activating handfuls of neurons with light and analyzing videos of the resulting behaviors, the researchers systematically explored most of the 10,000 neurons in the larval brain of *Drosophila melanogaster*.

"It's like saturation bombing," says neuroscientist George Augustine of the Center for Functional Connectomics in Seoul, South Korea. "They're marching through pretty much all the neurons in the nervous system of this simple little creature and finding out what all of them do. That's dramatic. That's profound."

Because much of the experimental work was automated, it took only several months to test 37,780 larvae. "The real challenge," Zlatic says, "was dealing with the data."

The team developed a mathematical approach to look for patterns of behavior elicited by activating small groups of neurons. This computational method revealed behaviors that would have been impossible to identify otherwise, says neuroscientist Aravinthan Samuel of Harvard University. "If you just look at tons of videos, it's very hard to see structure," he says. "The human eye just isn't able to handle that much data."



A larval fruit fly will turn and then turn again when researchers activate two neurons in its brain (circled in red).

But mathematics certainly can. By tracing larval movement in thousands of videos, the algorithm neatly described 29 distinct sequences of behaviors, the team reports. These behaviors included wiggles that help a larva escape from a threat, left turns followed by right turns and backward crawling.

The blue light failed to trigger some characteristic larval behaviors, such as a particular sort of predator-escaping roll, Zlatic says. That action is usually observed in moist environments, unlike the dry surface the larvae were tested on.

With the 29 behaviors in hand, scientists then used mathematics to look for neuron groups that seemed to bias the fly toward each behavior. The relationship between neuron group and behavior is not one to one, the team found. For example, activating a particular pair of neurons in the bottom part of the larval brain caused animals to turn three times. But the same behavior also resulted from activating a different pair of neurons. On average, each behavior could be elicited by 30 to 40 groups of neurons, Zlatic says.

And some neuron groups could elicit multiple behaviors across animals or sometimes even in a single animal.

Stimulating a single group of neurons in different animals occasionally resulted in different behaviors. That difference may be due to a number of things, Zlatic says: "It could be previous experience; it could be developmental differences; it could be somehow the personality of animals; different states that the animals find themselves in at the time of neuron activation."

The team also found that stimulating the same neurons in one animal would occasionally result in different behaviors. The results mean that the neuron-behavior link isn't black-and-white but rather probabilistic: Overall, certain neurons bias an animal toward a particular behavior.

The findings have implications for more complex brains. "I don't think anybody cares about how fly maggots move," Augustine says. But the same principles in this simple organism are probably at work in more sophisticated brains, he says.

Zlatic and her colleagues are currently studying a handful of these neurons and behaviors in more detail to understand how the neurons communicate with one another. The team ultimately plans on overlaying its results on a forthcoming map of all the physical connections between neurons in the *Drosophila* larval brain, offering scientists a more powerful way to study how neurons work together to control behaviors. ■

COURTESY OF JW. TRUMAN AND H. LI

Watch a video of fruit fly larval behaviors at **bit.ly/SNflyneurons**

First chromosome made synthetically from yeast

Feat is major advance toward creating engineered eukaryotic life-forms in the lab

BY TINA HESMAN SAEY

Designer organisms have crept closer to reality. Scientists have stitched together a version of a yeast chromosome. It is the first synthetic chromosome ever assembled based on a eukaryotic organism, which stores DNA in nuclei.

Other groups have previously synthesized chromosomes from bacteria, but this is the first step in designing synthetic eukaryotes.

Researchers from Johns Hopkins University, including a small army of undergraduate students, and colleagues report the achievement March 27 in *Science*. The synthetic chromosome is based on chromosome III from the yeast *Saccharomyces cerevisiae*, but it is not an exact replica.

In creating the synthetic version, researchers jettisoned some of the chromosome's extra baggage. These parts include gene-interrupting pieces of DNA called introns, genes that produce protein-building molecules called transfer RNAs, repetitive regions near the chromosome ends and remnants of genetic parasites called jumping genes, or transposons, that can replicate and move to other parts of the genome. The team also endowed the synthetic chromosome with a scrambling system that can reshuffle the chromosome's genetic deck to produce organisms with new properties.

The finished chromosome measures 272,871 base pairs long — much shorter than the original yeast chromosome's 316,617 base pairs. Base pairs are the information-carrying chemical units of DNA.

"What we're doing is essentially genetic engineering on steroids," says Jef Boeke, a yeast geneticist now at New York University who helped spearhead the project while at Johns Hopkins University.

> "What we're doing is essentially genetic engineering on steroids."
>
> **JEF BOEKE**

Boeke and colleagues created an undergraduate class called Build-A-Genome that aided in the assembly. Students started with single DNA strands that copied yeast chromosome III, 60 to 79 bases at a time, and melded them into 750-base-pair building blocks. Other team members snapped those DNA building blocks together to form "minichunks" 2,000 to 4,000 base pairs long.

Then the researchers let the yeast take over. Yeast cells are masters of a process called homologous recombination in which bits of matching DNA can swap with one another. In a series of 11 experiments, the researchers inserted an average of 12 synthetic minichunks into the yeast. The yeast then swapped the synthetic chunks for the matching portion of the native chromosome, eventually creating a fully engineered chromosome. Similar yeast assembly lines might stitch together chromosomes from other eukaryotic organisms such as humans, fruit flies, mice or plants.

Losing nearly 14 percent of the bases in chromosome III could have crippled the yeast's ability to compete, but the team found that yeast cells carrying the synthetic chromosome grew as well as ones with the natural version under 20 different conditions. Only one situation, growing with high concentrations of a sugar alcohol called sorbitol, put fungi with the synthetic chromosome at a slight disadvantage.

"The fact that it grows as well as it does is really encouraging," says Philip Weyman, a synthetic biologist at the J. Craig Venter Institute in La Jolla, Calif. Now the researchers can scramble the chromosome and learn which parts are really important for function and which bits cells can do without. That portion of the research will be the most interesting, but it will also be the most difficult, says Weyman. "Building it, as much of a hurdle as that was, was really the easy part."

Chromosome III is just one of yeast's 16 chromosomes, all of which Boeke and his colleagues plan to synthesize. The group hopes to have a completely synthetic yeast genome in three to five years, Boeke says.

He doesn't expect all of the altered chromosomes to work as well as this one did, but creating one that cripples yeast would be great for learning the rules about how to build a stripped-down genome.

"We aim to fail here," Boeke says. "We think we know a lot about the biology, but surely we don't know everything." Synthetic chromosomes that damage yeast's evolutionary fitness or fail to produce viable yeast may teach researchers how chromosomes evolved and give clues about the minimal requirements for eukaryotic life, he says.

The work may have other benefits too. The scrambling system built into the synthetic yeast chromosome could help create organisms that can more efficiently produce chemicals for drug and chemical companies, Boeke says. ■

Chromosome comparison

272,871 base pairs	316,617 base pairs
Synthetic chromosome's length	Original chromosome's length

Burmese pythons turn out to have fine navigation skills — not good news for people trying to control their spread in South Florida.

LIFE & EVOLUTION

Relocated pythons find way home

Invasive Burmese snakes show ability to return to territories

BY SUSAN MILIUS

Burmese pythons need no GPS to find their way home. The enormous snakes that have invaded South Florida turn out to be determined and able navigators, with unexpected homing abilities.

Since at least 1995, *Python molurus bivittatus* snakes have been breeding in Everglades National Park, and for just as long, people have worried about how to get rid of them (*SN: 2/25/12, p. 5*). The pythons eat many small mammals, including the endangered Key Largo wood rat.

In a study of how the pythons wriggle around the landscape, five of six adults that researchers captured and trucked 21 to 36 kilometers away managed to travel back to within five kilometers of their original locations, says Shannon Pittman of Davidson College in North Carolina.

The snakes seemed highly motivated, spending about three to 10 months moving in an oriented way. They may even have a compasslike sense of direction and a maplike sense of where they are, she and her colleagues propose in the March *Biology Letters*.

The snakes' ability to navigate doesn't bode well for efforts to contain them, Pittman says. Since the snakes seem to have a good sense of where they are, they may be able to take risks in exploring new habitat.

Few studies have looked in detail at navigation in any snake. Sea kraits demonstrate homing powers, showing up in the waters of their native island in Fiji after being moved to another island about five kilometers away. But studies translocating timber rattlesnakes and Aruba rattlesnakes found the snakes wandering broadly, with no sign they were heading homeward.

Pittman and her colleagues caught 12 adult pythons and drove them in covered containers to a wildlife center, where the researchers implanted radio transmitters under the snakes' skin. Researchers then returned six of the snakes to their original capture spots and drove the other six to new patches where pythons are known to flourish.

The translocated snakes moved faster and along straighter paths than the put-back-in-place snakes, the researchers found. Pittman now would like to know how younger snakes behave, because they're the ones that move out in search of new homes.

How the snakes steered their course isn't clear, Pittman says, though she notes that other reptiles can sense Earth's magnetic field, solar cues, odors or polarized light. Pythons might rely on these and landmarks to find their homes.

In the Everglades, the snakes don't have to find a den for overwintering, so snake ecologist Howard Reinert of the College of New Jersey in Ewing finds it "a bit surprising" that pythons there would be so motivated to get back home. He'd like to know more about whether the nontranslocated snakes seemed particularly attached to their homes. ■

BODY & BRAIN

Brain areas solve moral dilemmas

Regions balance competing interests in ethical judgments

BY LAURA SANDERS

Deciding whether to kill one person to save five is a deeply disturbing brain teaser. A study in the March 26 *Journal of Neuroscience* describes the neural tug-of-war that occurs in such a cruel, albeit hypothetical, situation.

Cognitive neuroscientists Amitai Shenhav of Princeton University and Joshua Greene of Harvard University asked 35 people to weigh in on 48 wrenching scenarios while undergoing functional MRI brain scans. The researchers used scenarios akin to the famous trolley choice: The hypothetical dilemma forces a person to decide whether to push an

MATTER & ENERGY

Filter lets light through from only one direction

Angle-sensitive device could improve photography and solar energy harvesting

BY ANDREW GRANT

Like a traffic cop directing cars, a layered stack of transparent materials permits light arriving from only a single direction to pass through. Such angle-sensitive filters could improve cameras and telescopes or allow solar cells to convert sunlight into energy more efficiently.

People have created light filters for thousands of years. Stained glass, for instance, filters light by color, allowing only a single shade to shine through while reflecting light at other wavelengths. But scientists have had trouble filtering light based on the angle from which it arrives.

In creating a directional light filter, MIT physicist Yichen Shen and his team

NATIONAL PARK SERVICE

View a slide show of moral dilemmas at **bit.ly/SNmorals**

innocent man to his death to stop a runaway trolley from killing five people.

This moral quandary evokes competing motivations: the urge to save the greatest number of people and the desire to avoid emotionally repellent behavior. In their experiment, the researchers separated these considerations by asking people to consider how emotionally wrenching a certain behavior would be or to consider only the greater good.

In one such dilemma, a live grenade sails into a cafe where 10 people sit. Participants were told that they could ignore the grenade, leaving the 10 people to die, or throw it onto the patio, saving the 10 people but killing a lone diner outside. When study participants considered only emotions ("Which do you feel worse about doing?"), activity increased in the left amygdala, one of a pair of almond-shaped structures deep in the brain. The more emotionally repellent an action was to participants, the more activity they had in the left amygdala, Shenhav and Greene found. The experiment couldn't pinpoint where utilitarian "greater good" considerations get made.

When people were asked to make a decision between actions considering all aspects of the moral dilemma, a different brain area seemed to step in. Activity in the ventromedial prefrontal cortex, a patch of tissue near the front of the brain, was greater when people were asked to make an overall choice compared with when people considered only emotions. After being lobbied by other brain regions, the vmPFC ultimately makes the call, the researchers suggest.

"It's some of the best support we've seen so far for the theory that the vmPFC is integrating emotional assessments from the amygdala," says cognitive neuroscientist Molly Crockett of University College London.

Earlier work implicated the vmPFC as a final arbiter in other types of decisions. This brain region seems to be involved in the choice to eat healthful or sinful food, for instance.

Crockett cautions that many of the moral dilemmas used in the experiment fall outside the realm of possibility for most people. "We're interested in moral decision making, but we're studying really unrealistic situations involving pushing people off bridges," she says. "It's not clear whether these neural mechanisms apply to real moral decisions."

Understanding how morality arises in the brain might have legal implications. Lawyers have argued that defendants' culpability depends on certain neural traits, for instance. And a 2011 study found that psychopaths have weak connections between the amygdala and the vmPFC (*SN Online: 11/30/11*). Knowing which brain areas guide moral decisions might lead to a better understanding of what behavior to expect when those regions are damaged, Shenhav says. ■

knew that at the interface between any pair of materials, light arriving from one specific angle, known as the Brewster angle, can simply pass through unimpeded — it won't get reflected or bent.

Knowing an interface's Brewster angle provided a way to allow light from a desired angle to pass through, but Shen's team had to figure out how to block the light from all other directions. The team solved the problem by creating a stack of 84 alternating layers of two transparent materials: glass and tantalum oxide. Whenever light struck the stack at any angle other than the Brewster angle, it underwent some combination of reflection and bending as it encountered each boundary between layers. By the time the light reached the 83rd and final boundary, it got totally reflected away. "It's a very clever approach," says Peter Bermel, an electrical engineer at Purdue University in West Lafayette, Ind.

To test the device, the team pointed a camera at a picture of a rainbow and placed the 2-by-4-centimeter rectangular stack in between. (The stack was placed in a tank of colorless liquid that has the same optical properties as glass so that no light would bend or reflect when entering the glass part of the stack.)

At first, the stack acted like a mirror, showing an image of the camera. It kept its reflective properties and remained a mirror when the researchers rotated the sample. Only at the magic angle of 55 degrees — the Brewster angle — did the glass–tantalum oxide stack become as transparent as a window and the rainbow came into view, the team reports in the March 28 *Science*. "It works over the entire range of visible wavelengths," Shen says, and should also work with nearly any pair of transparent materials. Bermel hopes to see future research combine angular control with filters of color and polarization.

Shen envisions someday fitting a camera with an angle-sensitive filter that would address the tricky proposition of photographing people outside on a bright day. As long as the sun isn't directly behind the person being photographed, the filter would eliminate background glare. Similarly, the technology could work in telescopes so that a backyard astronomer could spy on a faint star that would otherwise be overwhelmed by the brightness of a neighboring object.

A device made of glass and tantalum oxide filters light based on its incoming angle. The light ray coming in at 10 o'clock reflects off the filter. But the other beam streaks right through the device unimpeded.

Shen says a variation of the device could also improve solar thermal energy systems, which focus the sun's light and generate energy from its heat. A directional filter could allow the sun's rays, which come in at a predictable angle, to pass through but prevent the escape of heat in the form of infrared light. ■

WEISHUN XU AND YUHAO ZHANG

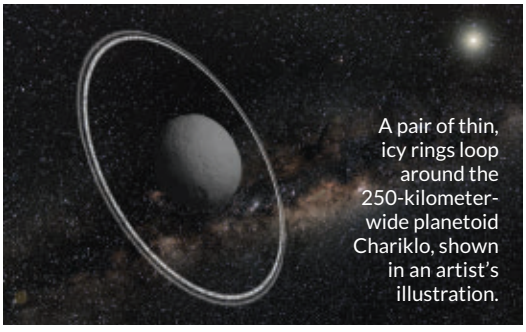
ATOM & COSMOS

Icy rings found around tiny space rock

Solar system may be home to many more small, haloed objects

BY CHRISTOPHER CROCKETT

All of the giant planets in our solar system are adorned with rings. Now, for the first time, a much smaller planetoid can take its place on the list of ringed objects.



A pair of thin, icy rings loop around the 250-kilometer-wide planetoid Chariklo, shown in an artist's illustration.

Astronomers have discovered a pair of thin rings encircling 10199 Chariklo, a rock-and-ice asteroid-comet hybrid known as a centaur. Chariklo is about 250 kilometers across and orbits the sun between Saturn and Uranus. The rings are probably the result of a collision.

The rings revealed themselves during an occultation, a chance alignment between Chariklo and a distant star when viewed from Earth. When seen from multiple vantage points, an occultation can tell researchers about the size and shape of the intervening body. Felipe Braga-Ribas, an astronomer at Observatório Nacional in Rio de Janeiro, coordinated occultation observations on June 3, 2013, with over a dozen telescopes scattered across South America.

The researchers expected to see a dip in the starlight as the centaur passed in front of the star. What they didn't expect was a pair of dips just before and another pair just after the main event. The additional flickers meant something else was traveling with Chariklo. "It was a big surprise," says Braga-Ribas. By combining all the observations, the team inferred the presence of a double ring composed mostly of water ice. The observations appear in the April 3 *Nature*.

"There's no doubt that there's a ring," says David Jewitt, a UCLA astronomer who was not involved in the study. "But nobody knows what it means." Even planetary rings are an enigma, he says. Chariklo's rings have a few possible origins. Another body may have smashed into Chariklo, excavating ice from the centaur's interior. Alternatively, Chariklo's gravity may have torn apart an icy moon orbiting the centaur. Or two moons could have collided.

Regardless of the cause, Braga-Ribas and Jewitt agree that unseen moons probably orbit Chariklo. Unless confined by the gravity from "shepherd moons," the rings would be quickly torn apart by Chariklo's gravity, collisions and radiation pressure from the sun.

Jewitt says that the rings mesh well with what astronomers already know about the outer solar system. Chariklo most likely came from the Kuiper belt, an icy debris field beyond Neptune, before settling into its current orbit. Many Kuiper belt objects are known to have at least one moon. A ring, he says, is the next logical step.

The rings also help explain Chariklo's recent odd behavior. From 1997 to 2008, the centaur grew fainter and its amount of ice apparently decreased. After 2008, it gradually returned almost to normal. The new observations suggest that in 2008, the rings appeared edge-on as seen from Earth. Chariklo appeared fainter because the rings blocked some of its light. The ice, which researchers now know is mostly in the rings and not on Chariklo's surface, appeared to vanish when only the rings' edges were visible.

Since astronomers haven't observed many centaur occultations and have already found a ring, Jewitt says, it may be that rings are relatively common. Braga-Ribas agrees. "We don't think it's the only one," he says. "We may have others." ■

BODY & BRAIN

Retired athletes have big bones

Health advantages of exercise persist for former ballplayers

BY MEGHAN ROSEN

Got baseball? Spending one's youth playing catch is good for the bones, with benefits that last a lifetime.

Years of hurling balls boosted bone size, mass and strength in the throwing arms of current and former professional ballplayers, researchers report March 24 in the *Proceedings of the National Academy of Sciences*. Though bone mass withered away when players retired, some of the size and strength stuck around — even 50 years after the athletes last played ball.

"If you exercise when you're young, it makes your bones bigger and stronger for life," says study coauthor Stuart Warden, a bone physiologist at Indiana University in Indianapolis.

Scientists have known for years that childhood physical activity makes bones stronger. But no one knew just how long the benefit lasted or whether it fended off fractures and bone diseases such as osteoporosis, Warden says.

He and colleagues recruited 103 current and former professional baseball players to look at differences between throwing and nonthrowing arms. Pro ballplayers have typically spent their youths heaving horsehides, Warden says. The game and its training methods haven't changed much in 100 years, so his team could compare today's athletes with those who played decades ago.

Bone scans revealed major league differences in the upper arm bone strength of men's throwing and nonthrowing arms.

"Just by exercising, they've made their bone twice as strong as the same bone on the other side of their body," Warden

LUCIE MAQUET

Watch a video about Chariklo's rings at **bit.ly/SNrings**

ATOM & COSMOS

Sea hides below Enceladus' ice

Gravity maps of Saturn moon reveal ocean of liquid water

BY CHRISTOPHER CROCKETT

Scuba divers take note: An underground ocean awaits on a moon of Saturn. Astronomers have, for the first time, measured the depth and extent of a subsurface sea on ice-covered Enceladus. The findings shore up the notion that an underground reservoir feeds the moon's ice geysers and raise questions about Enceladus' habitability.

Astronomers once thought of 500-kilometer-wide Enceladus as an unchanging, dead world. But the Cassini spacecraft, which arrived at Saturn in 2004, found geysers shooting particles of salty water ice through fissures that dot the southern hemisphere. The fissures expand and contract in rhythm with the tides of Saturn (*SN Online: 7/31/13*). Heat wells up from the moon's interior through the cracks. Putting this evidence together, many astronomers suspect that a sea of liquid water lurks beneath the ice.

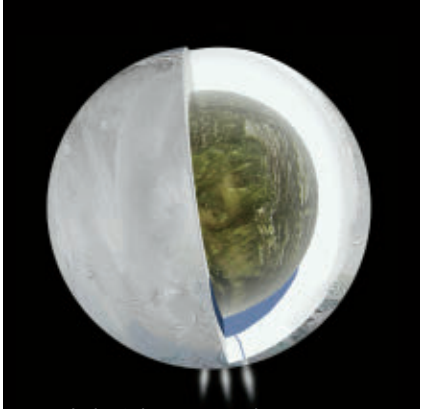
To peek inside Enceladus, Luciano Iess, an aerospace engineer at Sapienza University of Rome, and colleagues looked at Doppler shifts in Cassini's Earth-bound radio signal during three flybys of the moon. The Doppler shifts, tiny changes in the frequency of the radio waves, track the spacecraft's speed. Whenever Cassini passed over a part of the moon with slightly more mass, the increased gravity accelerated the probe. Iess and colleagues used the changes in speed to map Enceladus' interior.

The team concluded that a 10-kilometer-deep ocean must sit under 30 to 40 kilometers of ice and on top of the moon's rocky core. Extending from the south pole to mid-southern latitudes, the sea has a water volume similar to Lake Superior's.

Iess says he's not surprised that the gravity data suggest an underground sea. "I would have been more surprised if we didn't find anything," he says. The results appear in the April 4 *Science*.

"This is a major piece of the puzzle," says Candice Hansen, a planetary scientist at the Planetary Science Institute in Tucson. Until now, researchers had only a trickle of clues hinting at the possibility of a subsurface ocean. "It's one thing to think that's what's happening," says Hansen, "but it's another thing to have data that says, 'Yeah, we're right.'"

One thing that is not yet clear is how the sea feeds the ice geysers tens of kilometers above it. "It seems unlikely that you'd have a lake directly connected to the surface," says planetary scientist Joseph Spitale, also of the Planetary Science Institute. "There must be an intermediate plumbing system."



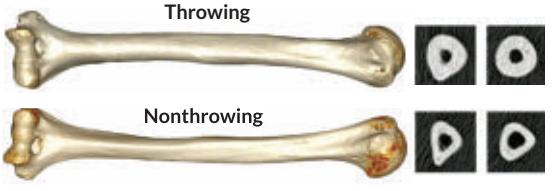
Enceladus is home to a subsurface ocean of liquid water near its south pole, as seen in this diagram of the moon's interior. Narrow tunnels may jut through ice to water vents visible on the surface.

Spitale adds that the new gravity data appear to rule out one of the leading explanations for how the moon's interior generates heat: a slight rocking motion induced by Saturn's gravity. The new data provide no evidence for any rocking.

Regardless of how the interior stays warm, Iess says some interesting chemistry may go on where the seawater meets rock. And discussions of heat rising from the moon's interior mixed with a salty ocean inevitably lead to musings on the possibility for life. Though no evidence suggests that aliens swim the seas of Enceladus, a warm, briny environment raises intriguing possibilities.

For now, those ideas will remain theoretical. Cassini has only three more flybys of the moon planned. "We're essentially done with Enceladus," says Iess. "Unfortunately, it will be a long time before we return to a very interesting moon." ■

FROM TOP: JPL-CALTECH/NASA; COURTESY OF S. WARDEN



The upper arm bone of a professional baseball player's throwing arm is bigger (see bone cross section, right), stronger and more massive than that of his nonthrowing arm. Some of the strength and size benefits last long after athletes stop throwing.

says. "There's no drug that you can take that will do that."

The upper arm bones of throwing arms also had larger cross-sectional areas and more mass.

These bone benefits began to dwindle once players permanently stepped away from the plate, and mass gains eventually faded away completely. But retirees in their 80s kept more than half of the bone size benefits and a third of the strength benefits built up in their youth.

Still, people who don't grow big bones in childhood haven't struck out entirely: They might be able to cover their bases by exercising throughout adulthood. Former ballplayers who continued to throw staved off bone loss and retained more bone strength than those who didn't.

The study shows that "you can expect some permanent benefits from physical activity," says René Rizzoli of Geneva University Hospitals in Switzerland. "This is an important public health issue."

Kids who spend their time in front of the TV instead of at Little League games may miss out on a critical bone-building window that closes after puberty. "If we can get more children out and active, we can improve their bone health," says kinesiologist Adam Baxter-Jones of the University of Saskatchewan in Canada. Otherwise, he says, kids "will increase their risk of developing bone disease." ■

LIFE & EVOLUTION

Zebra stripes may be antifly defense

Fur pattern's function not tied to camouflage or cooling

BY SUSAN MILIUS

Protection from flies may best explain the function of zebra stripes.

A head-to-head test of five explanations that researchers have proposed for zebras' distinctive patterning finds no support for other long-discussed ideas, says Tim Caro of the University of California, Davis. Looking at the ranges and ecology of various zebras and other members of the genus *Equus* undermines, for example, the notions that stripes camouflage animals in woods or dazzle big predators into misjudging prey movements and flubbing an attack.

The best explanation for the iconic stripes' function turns out to be discouraging bloodthirsty tabanid flies and tsetse flies, Caro and his colleagues report April 1 in *Nature Communications*. Other experiments have found that flies prefer landing on solid colors instead of contrasting stripes.

To test five broad categories of the many ideas that scientists have proposed, Caro and colleagues looked first at geography. The team analyzed the ranges of 20 subspecies or species in the *Equus* genus: the vividly striped zebras, three wild asses with striped legs plus eight asses and a wild horse with an unstriped coat. Researchers quantified stripiness, or lack thereof, of each subspecies using such measures as the number of bars on the neck or the intensity of color in leg stripes. Then the analysts looked for links between striping and ecological factors such as fraction of range covered by woodlands.

The researchers failed to find much of a link between woodsiness and stripe measures, negating the idea that dark and light stripes benefit zebras by letting them blend in with the dark-and-light patterns in thickets of branches.

An unusual head-to-head test of ideas about the function of zebra stripes finds little support for some old notions and only one winner: defense against biting flies.

Likewise environmental links didn't support the importance of stripes for setting up little convection currents that might cool zebra skin or for disrupting the attacks of big predators such as lions. This idea of disruption included the appealing concept called motion dazzle, in which bold, repetitive patterns twist perceptions of motion. Studies of diets among intensely monitored wild lions also debunk this idea, since lions catch an abundance of zebras, Caro notes.

And the size of social groups in various *Equus* subspecies showed no strong

HUMANS & SOCIETY

Youth programs lift adult health

Preschool intervention offers benefits that last decades

BY BRUCE BOWER

High-quality early-childhood programs may reduce adults' obesity and blood pressure, a new study finds.

Kids from poor families who were randomly assigned to a program of educational activities, basic medical care and healthy meals for the first five years of life displayed better health in their mid-30s than peers who didn't get the intervention, say psychologist Frances Campbell of the University of North Carolina at Chapel Hill and her colleagues.

It's not clear precisely how the childhood program might have boosted adult cardiovascular and metabolic health, the researchers report in the March 28 *Science*. But since a program with an average annual cost of about $18,000 per child in today's dollars appeared to yield sustained health benefits, the researchers conclude, early-childhood interventions may represent a way to bring down health care costs.

Campbell's team analyzed data from the Carolina Abecedarian, or ABC, Project. Its designers wanted to see if an intensive early-childhood program could prevent low IQs among kids born into poverty in North Carolina between 1972 and 1977. Researchers have found that IQ gains among children in the ABC Project and other early-childhood interventions, including Head Start, tend to fade by young adulthood.

Evidence indicates, however, that participation in these programs leads to greater self-control and motivation, higher standardized test scores and better-paying jobs.

In the new study, a doctor performed physical exams on 68 individuals in their mid-30s. Of that group, 37 had participated in the ABC program from about age 2 months to 5 years. The rest had received social services only as needed.

The ABC intervention consisted of day care in small groups with teachers who led educational games; regular medical exams; and breakfast, lunch and a snack.

Health benefits among grown-up ABC participants, especially men, included relatively low rates of hypertension, obesity and vitamin D deficiency. Mean systolic blood pressure among these men was 126 millimeters of mercury, compared with 143 millimeters of mercury among men in the comparison group.

No men from the ABC program, versus one of every four men from the comparison group, exhibited the combination of elevated weight, cholesterol and blood pressure that signals high risk of heart disease, stroke and diabetes.

Women from the ABC program had a lower rate of obesity than comparison women: 56 versus 76 percent.

connection with stripes, downgrading the hypothesis that stripes might facilitate social interactions.

But stripes did correlate with geographic regions that had months of weather favoring dense fly populations. Further backing the idea is that flies in zebra zones spread four diseases lethal to *Equus*. Studies of domestic livestock in North America have shown that biting flies cause substantial blood loss and lower milk production.

The analysis gets at what functions the stripes serve now, not what drove their original evolution in ancient climates and ecosystems, Caro says. Scientists may never know how zebras first got their stripes.

Altogether the case is "compelling," says Innes Cuthill of England's University of Bristol, whose favorite hypothesis had been dazzling predators. He's cautious about declaring the function of zebra stripes absolutely resolved, but he says he now ranks Caro's evidence for flies as "the leading contender in the queue of explanations." ■

Men, but not women, assigned to the early-childhood program were also more likely to have health insurance at age 30 and to get medical care when sick.

Reduced obesity rates during the first two years of life, as a result of better nutrition and medical care, may somehow have primed kids in the ABC program for relatively good health decades down the line, the researchers speculate. But the study contains too few participants to determine whether any one aspect of the intervention mattered more than the others.

"Early-childhood intervention is a viable strategy for health promotion over the life course," remarks child development professor Arthur Reynolds of the University of Minnesota, Twin Cities. The new report and several other long-term studies of preschool interventions suggest that kids who acquire self-control and other skills underlying school success also achieve better physical and mental health as adults, he says. ■

GENES & CELLS

Neandertal legacy written in fat metabolism

DNA from interbreeding may have helped humans adjust to new environments in Europe

BY MEGHAN ROSEN

Fat metabolism genes inherited from Neandertals may have helped Stone Age humans survive in Europe around the time when cave lions and saber-toothed cats ranged the continent.

Remnants of these genetic gifts pop up in the genomes of modern-day Europeans and appear to tweak fat levels in their brains, researchers report April 1 in *Nature Communications*.

The findings are the latest in a string of studies trying to suss out the role Neandertal ancestry played in human evolution (*SN: 3/8/14, p. 12*).

> "It's like you have a Neandertal in your family tree."
>
> MICHAEL LACHMANN

Each newly discovered Neandertal genetic detail — about their hair, skin, pigmentation and now fat metabolism — reshapes scientists' view of the ancient hominids, says paleoanthropologist John Hawks of the University of Wisconsin–Madison, who was not involved with the new work.

"The reality is that they contributed to our population and they gave us things that had value," he says.

From about 200,000 to 30,000 years ago, Neandertals lived in Europe, the Middle East and Asia. After humans migrated out of Africa some 70,000 years ago, they met up and mated with Neandertals. As a result of this interbreeding, about 2 percent of non-Africans' DNA today comes from Neandertals.

That's about as much DNA as people inherit from a great-great-great-great-grandparent, says evolutionary biologist Michael Lachmann of the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. "It's like you have a Neandertal in your family tree."

To find out whether interbreeding with Neandertals helped humans, Lachmann and colleagues compared Neandertal genomes that had been previously sequenced (*SN: 3/14/09, p. 5*) with genomes from 11 modern human populations. The researchers looked for collections of genes with unusually high or low levels of Neandertal DNA.

Neandertal DNA is typically sprinkled evenly throughout non-Africans' genomes, Lachmann says. But in Europeans, genes involved in fat synthesis held more than three times as many Neandertal DNA sequences as did other regions of the genome. Genetic signals hinted that this DNA might have spread rapidly through ancient human populations.

Next the team examined whether the genetic remnants of Neandertals tinkered with fat metabolism in today's humans. Compared with people from other parts of the world, Europeans have different concentrations of fats in the brain — the first time scientists have reported a difference in brain fat composition among human populations.

"There must be some purpose" for the genetic changes in fat metabolism, says study coauthor Philipp Khaitovich, an evolutionary biologist at the Chinese Academy of Sciences and Max Planck Society Partner Institute for Computational Biology in Shanghai.

"But we still have no idea, no idea at all," he says.

One possibility lies in how the brain works. Fat helps messages zip between neurons, but researchers don't know whether the change in Europeans boosts brainpower, or whether a Neandertal-like fat metabolism helped ancient humans stay warm in cool climates. Or the genetic changes could have been adaptive for an entirely different reason that researchers haven't dreamed up yet. ■

Bank voles carry a version of a normal protein that makes them particularly susceptible to prion diseases, such as mad cow disease.

PETER TRIMMING/FLICKR (CC BY 2.0)

GENES & CELLS

Bank voles provide clue to prion disease susceptibility

Mice engineered with rodent's brain protein develop other species' degenerative brain disorders

BY TINA HESMAN SAEY

Prion diseases, the frightening neuron-killing disorders that transmit via infectious proteins rather than by viruses or bacteria, rarely jump from one species to another. But a change in just a few amino acids can tear down the barriers that prevent an animal from contracting another species' prion disease.

Bank voles are particularly susceptible to the infectious brain-wasting diseases. That susceptibility stems from the structure of one of the vole's brain proteins, researchers from the University of California, San Francisco report April 3 in *PLOS Pathogens*.

Prion diseases include mad cow disease in cattle, scrapie in sheep, chronic wasting disease in deer and several degenerative brain diseases in people, including Creutzfeldt-Jakob disease. The disorders result when a normal brain protein called PrP twists into an abnormal shape known as a prion and remakes other PrP proteins in its disease-causing image. The misshapen proteins clump together and form plaques, killing brain cells in the process.

Normally, one animal's prion has a hard time corrupting the PrP proteins from different species. That species barrier is stronger for some species than for others. For instance, dogs and rabbits don't get prion diseases under normal circumstances, says study coauthor Joel Watts, a neuroscientist at the University of Toronto, who did the research while at UCSF.

But bank voles (*Myodes glareolus*), small mouselike rodents that live in woodlands in Europe and parts of Asia, "are susceptible to every prion disease known to man," Watts says. That's true when researchers inject prions directly into the rodents' brains in the lab; no wild bank vole has ever been diagnosed with a prion disease, he adds.

Watts and his colleagues wondered whether this vulnerability results from the vole's physiology — perhaps the animal produces some other protein that helps contort PrP into the brain-killing form — or because of something special about the vole PrP protein itself.

To find out, Watts and colleagues genetically engineered mice to have the vole PrP protein. Researchers then injected infectious prions from any of eight species into the mice's brains. The mice that produced high levels of vole PrP developed neurological diseases. Those results suggest that bank vole PrP is a "universal acceptor" that can adopt the infectious prion form from any species.

Exactly what gives the vole protein its mighty morphing abilities is a mystery. It differs from the normal mouse PrP by only eight amino acids, and, Watts says, "none of them grab you and make you say, 'Ah! That's it!' "

Mice making high levels of their own PrP are only marginally susceptible to other species' prion diseases and sometimes don't contract them at all, Watts says. But mice bearing the vole version of PrP developed prion diseases surprisingly quickly, says David Westaway, a neuroscientist a the University of Alberta in Edmonton. "That's a logistical advantage" for researchers studying prion diseases, he says, because it could speed up experiments by months or years and accelerate work toward potential therapies.

"The idea that bank vole PrP might be a universal acceptor could be significant for both scientific and practical reasons," says Surachai Supattapone, a biochemist at Dartmouth College's medical school. If prions from any species can really wrench the vole PrP into disease-causing states, researchers could test whether a new neurological disease is a prion disease by injecting brain matter from sick animals into mice carrying the vole proteins. If the mice then develop the disease, there's a good chance it's caused by a prion, he suggests.

Such a test would have been useful at the beginning of the mad cow or chronic wasting disease outbreaks, he says. "It would also be useful to detect and study potential cases of prion disease in unusual species, such as zoo animals." ■

HUMANS & SOCIETY

Herders spread crops across Asia

Pastoralists transported grain from two big farming centers

BY BRUCE BOWER

Nomadic herders who inhabited Central Asia more than 4,000 years ago spread crops across much of Asia and took up cultivation themselves surprisingly early, a new study suggests.

The findings fit with other emerging evidence that ancient Asians flexibly mixed herding and farming lifestyles. That type of adaptability enabled agriculture to initially spread via mainland herders, not migrating farmers or trading networks of urban civilizations as anthropologists had previously thought.

Seeds recovered at two herder campsites in Kazakhstan represent the earliest evidence of the combined use of bread wheat and broomcorn millet, say anthropologist Robert Spengler of Washington University in St. Louis and colleagues. Radiocarbon dates of charred wood and seeds at these sites range between roughly 4,800 and 4,300 years ago.

The findings push back the dates of the two crops' first appearance in Central Asia, suggesting that the herders transported bread wheat from west to east and broomcorn millet from east to west. Bread wheat originated about 6,000 years ago in Southwest Asia and arrived in East Asia by 4,500 years ago. Broomcorn millet was domesticated 8,000 years ago in East Asia and turned up in Southwest Asia 4,000 years ago.

No signs of farming appear in the earliest occupations at the two camps in Kazakhstan, the team reports April 2 in the *Proceedings of the Royal Society B*. The seeds mainly came from special chambers in which the dead were cremated.

> "Agriculture and settled life are shown to be separate and independent activities in these new discoveries."
>
> DAVID ANTHONY

"Early pastoralists in Central Asia may have obtained crops solely for burial rituals," says anthropologist and study coauthor Michael Frachetti of Washington University.

Seeds discovered by the researchers at two other camps in Turkmenistan indicate that mobile herders ate millet, wheat, barley and green peas over 3,400 years ago. An absence of broomcorn millet at nearby farming villages suggests that herders cultivated that crop, and perhaps others, about 600 years before previous evidence of farming in Central Asia.

Spengler's team regards these herders' camps as seasonal outposts, not permanent settlements. "Agriculture and settled life are shown to be separate and independent activities in these new discoveries," says David Anthony, an archaeologist at Hartwick College in Oneonta, N.Y.

Spengler and colleagues provide "much needed data" showing that herding groups transported domesticated crops out of two cultivation heartlands to the rest of Asia, adds Harvard University archaeologist Rowan Flad.

Ancient herding groups, each containing no more than about 20 individuals, built numerous camps in summer and winter pastures across Central Asia, says Frachetti. Previous finds suggest that groups periodically congregated at certain summer pastures. Exchanges there led to the gradual expansion of wheat from west to east and millet from east to west, he proposes. ■

MATH & TECHNOLOGY

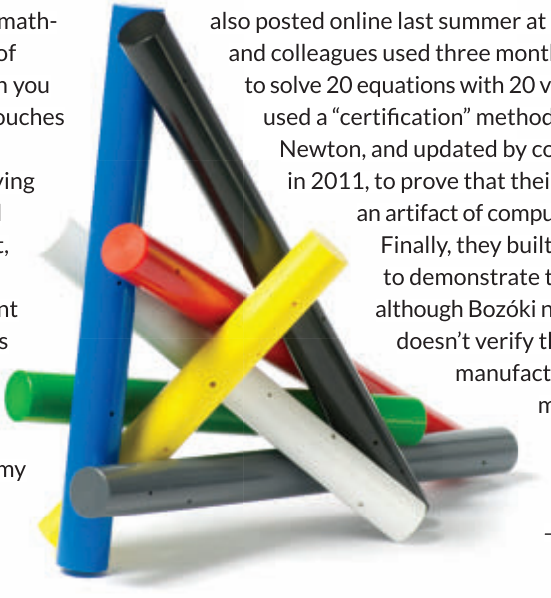
A tale of touching tubes

ATLANTA — Over 50 years ago, the popular mathematics writer Martin Gardner and readers of *Scientific American* pondered a challenge: Can you place seven cigarettes so that each cigarette touches every one of the others?

Gardner had a solution, but it was unsatisfying because some of the cigarettes' ends touched others' sides. If that end of a cigarette were lit, it would no longer touch its neighbor. Mathematicians wondered whether an arrangement could be found with only side-to-side contacts using infinitely long cylinders.

On March 20 at Gathering 4 Gardner, a conference to celebrate Gardner, mathematician Sándor Bozóki of the Hungarian Academy of Sciences in Budapest presented a solution of this more challenging problem, which was also posted online last summer at arXiv.org. Bozóki and colleagues used three months of computer time to solve 20 equations with 20 variables. Then they used a "certification" method developed by Isaac Newton, and updated by computer scientists in 2011, to prove that their solution was not an artifact of computer rounding errors. Finally, they built a wooden model to demonstrate their answer — although Bozóki notes that the model doesn't verify the result because manufacturing errors are much greater than any errors the computer could have made.

— Dana Mackenzie



PÉTER GÁL

LIFE & EVOLUTION

How hummingbirds manage to fly the unfriendly skies

Swirling air can make hummingbirds work harder to hover when the air's vortices open wider than a bird's wing. The first measurements that show a flying animal's metabolism revving up when coping with turbulent air come from five Anna's hummingbirds (*Calypte anna*) that Victor Ortega-Jimenez of the University of California, Berkeley and colleagues tested. In a wind tunnel, the hummingbirds hovered at a feeder downwind from a cylinder. Buffeted by vortices of air whipping off slim cylinders 2 or 4 centimeters in diameter, the birds held position without needing extra oxygen even with wind speeds of 9 meters a second, or about 20 miles per hour. But when researchers used a 9-centimeter-wide cylinder, vortices widened to 173 percent of wing length. This time hummingbird metabolisms increased some 25 percent on average — even at gentler wind speeds of 3 and 6 meters per second. The hummingbirds relied on asymmetric tail and wing motions to hover in place, the researchers report March 26 in the *Proceedings of the Royal Society B*. — *Susan Milius*

BODY & BRAIN

Electronic cigarettes don't help smokers quit

Electronic cigarettes may not shut off the urge to smoke cigarettes. A survey of 949 smokers found no difference in quit rates a year after some had taken up e-cigarettes (one shown, right), researchers at the University of California, San Francisco report March 24 in *JAMA Internal Medicine*. E-cigarettes deliver nicotine in vapor form without the cancer-causing combusted materials of a lit cigarette. Manufacturers suggest that using them is a first step toward quitting smoking. Of 949 smokers who answered online questionnaires, 88 reported having tried e-cigarettes at the study's outset. One year later, about 13.5 percent of all participants had quit smoking. Roughly equal fractions of e-cigarette users and smokers who didn't use them had successfully quit regular cigarettes; differences in quit rates between the two groups fell within the study's margin of error. The authors suggest that regulators prohibit ads claiming that e-cigarettes help people quit smoking unless scientific evidence emerges to prove it. — *Nathan Seppa*

Dietary supplement eases Huntington's symptoms in mice

Huntington's disease robs a person of a healthy mind and body. A simple dietary fix may guard against the theft, which currently can't be stopped or slowed with treatments. Mice carrying a mutation similar to that in people with Huntington's disease showed improvements after eating a diet rich in cysteine, a simple amino acid commercially available as a dietary supplement, scientists report March 26 in *Nature*. The mice with the mutation had reduced levels of an enzyme that makes cysteine in key parts of their brains, Solomon Snyder of Johns Hopkins University School of Medicine and colleagues found. These mice also had movement and brain problems, as do people with Huntington's disease. But a cysteine-rich diet improved these symptoms. With a cysteine boost, the mice were better able to balance, had a stronger grip, had heftier brains and even lived longer. But the mice still weren't as healthy as mice that didn't carry the Huntington's disease mutation. And it's unclear whether the results will hold up in people. — *Laura Sanders*

ATOM & COSMOS

Icy planetoid found lurking at edge of solar system

There's a new icy world in the outer suburbs of the solar system. Named 2012 VP113, the planetoid could be a member of the Oort cloud, the giant icy junkyard thought to envelop the sun (*SN: 10/19/13, p. 19*). The newly found object joins the dwarf planet Sedna as one of only two worlds known to orbit beyond the Kuiper belt, where Pluto resides along with hordes of icy boulders left over from the solar system's formation. Astronomers discovered 2012 VP113 as a spot drifting against the fixed backdrop of stars in pictures of a patch of sky taken a couple of hours apart. Continued observations at Chile's Cerro Tololo Inter-American Observatory and Las Campanas Observatory revealed a roughly 450-kilometer-wide planetoid that comes no closer to the sun than 12 billion kilometers — about 80 times the distance between Earth and the sun. Astronomers argue in the March 27 *Nature* that the presence of 2012 VP113 and Sedna hint at a vast unseen reservoir of icy worlds at the edge of the solar system. The two could also help researchers understand the solar system's early development and interactions with its neighbors in the galaxy. — *Christopher Crockett*

GENES & CELLS

New mechanism discovered for decades-old cancer drug

A popular cancer drug has previously undiscovered tricks up its sleeve. The chemotherapy drug paclitaxel (Taxol) treats breast, lung, ovarian and other cancers. Animal studies and experiments on human cells in lab dishes had suggested the drug worked by freezing cells in the act of dividing. But those studies used drug doses much higher than tumor cells in a patient's body would encounter. Beth Weaver of the University of Wisconsin–Madison and colleagues measured drug levels in breast tumors taken from women who had been treated with paclitaxel before surgery. Instead of stopping cell division, low, clinically relevant doses of the drug caused cells to pull their chromosomes in multiple directions. Cells caught in the multidirectional tug-of-war died after the split because their chromosomes had been scrambled, Weaver's team reports in the March 26 *Science Translational Medicine*. Knowing how paclitaxel really works may help doctors better predict who will benefit from the drug. The understanding may also aid researchers in designing more effective versions. — *Tina Hesman Saey*

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Unsolved DRUGS

A simmering controversy surrounds what's still unknown about how antibiotics kill

By Beth Mole

Antibiotics such as penicillin attack a bacterium's cell wall, bursting it (left, shown next to an intact *Staphylococcus aureus* cell in false color). Some studies suggest that antibiotics also trigger other kinds of damage that could be harnessed in the battle against antibiotic resistance.

CNRI/SCIENCE SOURCE

Penicillin attacks with a calculated strike, splitting open cell walls. Kanamycin sends a bacterium's protein assembly line into mayhem. Ciprofloxacin dices a microbe's DNA into a genetic hash. Like trained snipers, each of these common antibiotics seems to dispatch bacteria with a simple tactic: Target a high-profile molecule crucial to survival and, with a single, clean shot, defeat the whole cell.

For decades, this notion of how antibiotics kill has guided the design and deployment of drugs that have saved countless lives. Since scientists introduced penicillin in the 1940s, antibiotics have tamed some of the most deadly microbes into mild nuisances. Bacterial infections that cause tuberculosis, pneumonia and diarrhea, the three leading killers in the United States a century ago, have become curable or rare in the developed world.

But after myriad clinical victories, some scientists are questioning what we really know about the modus operandi of these bacterial assassins. In 2007, researchers proposed that, in addition to direct hits, antibiotics rev up cellular power plants and generate explosive waste. The resulting biochemical chaos might be as important to an antibiotic's deadly force as the better-studied precision tactics. The intriguing idea of antibiotics as sloppy killers created a buzz followed by a heated controversy in an otherwise sedate corner of science, says Gerry Wright, who studies antibiotic resistance at McMaster University in Hamilton, Canada.

A string of studies have come out for and against the theory, with more under way. Last year, two critical studies sparked widespread skepticism of the science behind the theory. But the clash hasn't deterred some scientists from digging further, in hopes that the fresh perspective could lead to new therapies.

Chief among them is systems biologist James Collins of Boston University, who first proposed the controversial theory. Most scientists, he says, thought that they understood how antibiotics kill. But scientists may know only a "tiny, narrow slice of what's happening," he says.

That might not be bad news: Understanding these unseen complexities of how drugs actually kill could inspire a new generation of desperately needed treatments as mounting numbers of bacteria become resistant to antibiotics.

In an evolutionary arms race with drug development, some microbes have evolved to guard or disguise prominent drug targets. Other bacteria have come up with ways of simply deporting drugs from their cellular borders. Health experts around the world have warned that modern medicine is losing a war that most thought was already won. In the United States, about 2 million people get sick with a drug-resistant bacterial infection each year and 23,000 or more die. As those numbers rise, bacteria keep developing new defenses. Some

tuberculosis infections, for instance, can withstand assaults by every drug in a physician's arsenal.

In response, researchers are busily drawing up blueprints for new antibiotics and picking out new molecular targets to attack. But the efforts have been slow, scientifically challenging and expensive. Between 1998 and 2012, the U.S. Food and Drug Administration approved 14 new antibiotics. Only four had novel mechanisms.

New thinking about how old drugs actually kill could speed the development of new therapies, says Collins. If collateral damage is important in killing microbes, then figuring out ways to increase that damage could help extend the usefulness of existing drugs. Collins envisions antibiotic sidekicks that would stir up molecular trouble in the cell, creating a combination therapy that would make existing antibiotics more lethal.

The idea is to restore the effectiveness of current antibiotics as opposed to discovering a new class of antibiotics, says Jeff Wager, president of EnBiotix, a Boston company developing drugs based on Collins' work. "This just seems like a smarter, faster, more broadly applicable way to approach the problem," he says.

But Collins' antibiotic theory has vocal foes. "You never say never," says microbial physiologist James Imlay of the University of Illinois at Urbana-Champaign. But to Imlay, the evidence against the idea is "as ironclad as you can get."

Whoever is correct, the disagreement is telling of the many unknowns about how classic antibiotics work. It points to uncharted territory potentially rife with new targets and attack strategies. "I'm not convinced we really understand how all these antibiotics work," says Wright. "The people who are trying to figure it out are doing us all a favor."

Antibiotic aim

Collins, a biomedical engineer, didn't set out to design new antibiotic therapies. In the early 2000s, he waded into the nascent field of systems biology. The field, which itself has created a stir among biologists, eschews traditional molecular techniques and instead applies an engineer's logic to understanding how networks of proteins and molecules orchestrate the workings of an organism. When a graduate student came up with data revealing unseen effects of quinolone antibiotics, a class that treats skin, lung and urinary tract infections, Collins grew intrigued.

Generally, scientists have thought that quinolones such as ciprofloxacin and nalidixic acid kill bacteria by leaving DNA in bits. The drugs latch onto bacterial enzymes called topoisomerases, including gyrase, which clamp onto DNA strands, untwisting and unhooking the A's, G's, C's and T's so other molecular machinery can move in and repair, decode or make copies of the genetic sequence. Quinolones lock on after the enzymes break strands of DNA for untwisting, leaving dangling fragments. Since dozens if not hundreds of gyrase and topoisomerase enzymes work along a bacterium's DNA at once, a large-scale invasion of quinolone molecules can swiftly reduce a whole chromosome to rubble. If the bacterium can't copy its genes, it can't function. The minced genetic mess, it seemed, spelled eventual death for the cell.

But Karl Drlica, a molecular biologist at Rutgers' New Jersey Medical School in Newark who has studied the drugs for years, points out that damaged DNA does not necessarily equal cell death. "You get these drug-DNA complexes, and that blocks growth," says Drlica. But bacteria have a repair system, aptly called the SOS response, to fix DNA breaks. Quinolones' main strike "is reversible, so it's not lethal, because death is not reversible," he says.

2 million

Number of people in the United States who get sick with a drug-resistant bacterial infection each year

Approaching the issue at a systems level, Collins and his team used tools such as DNA microarrays to take a cellular census of which genes were turned up or down during an antibiotic strike. "What our systems analysis shows is there's more to the story," says Collins. While scrambling to repair DNA, the microbes were also fending off cell-wide damage. Dying cells cranked up their metabolism, deployed fighters to mop up dangerous superoxide molecules and threw iron regulation into disarray. Unknown to scientists, quinolones triggered a bevy of defensive maneuvers.

Drugs of mass destruction

By investigating each defense separately, Collins and his team came up with a model of how it all fits together: The direct hit to DNA indirectly sends metabolism into overdrive as the cell desperately attempts to power up its defenses. The boost of activity at the bacterial power plants leads to a buildup of a cellular toxic waste product called superoxide. If there's too much, it can overload a bacterium's normal disposal systems. Spilling through a cell, the reactive molecule sets

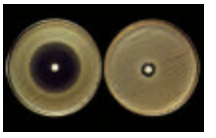
Battling bacteria

Antibiotics have proved powerful in the fight against disease. But from the beginning their use has fostered resistance.

1941
Penicillin first given to a human patient

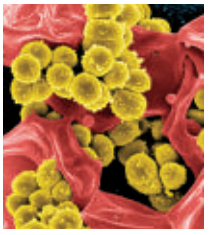
1944
Streptomycin, the first aminoglycoside, used to treat TB

1945
Reports confirm penicillin-resistant bacteria (right)



1950s
"Golden Age" of antibiotic development, with introduction of erythromycin, vancomycin and more

1961
Methicillin-resistant *Staphylococcus aureus* (shown) isolated



1962
Nalidixic acid, the first quinolone, used to treat urinary tract infections

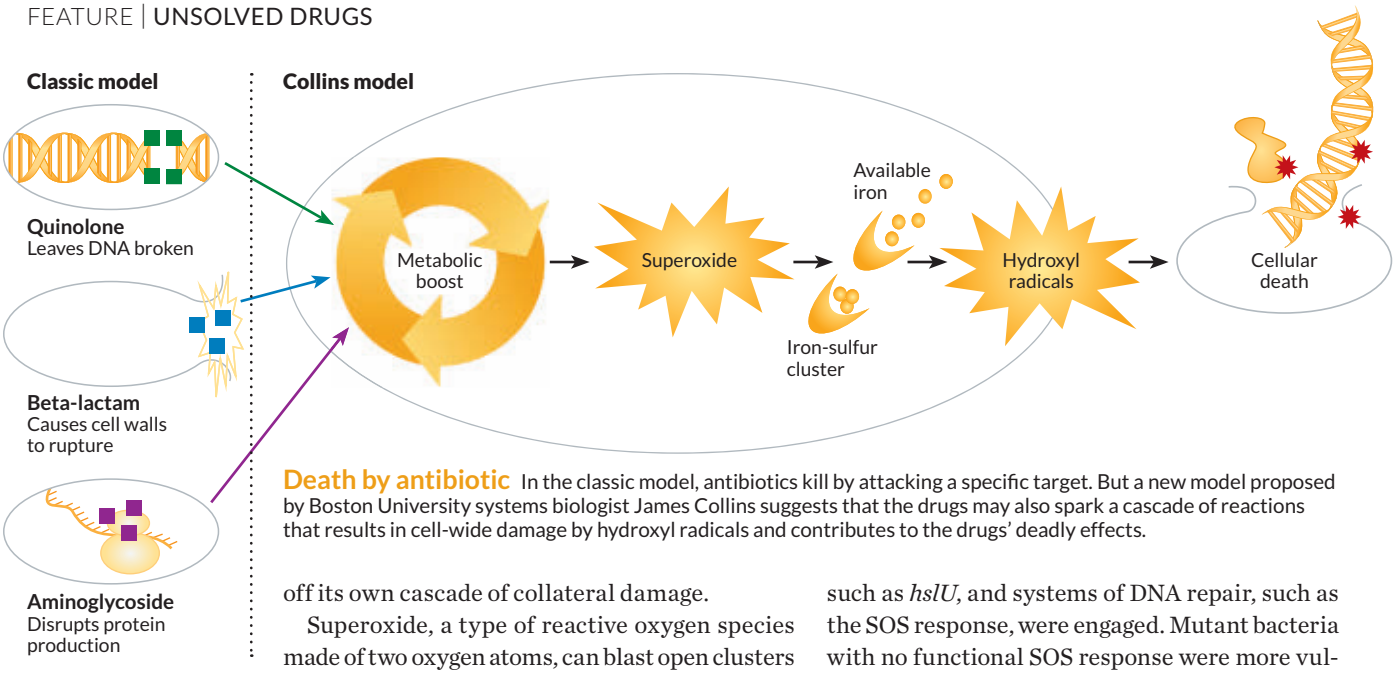
1968 to 1998
No new classes of antibiotics introduced

2006
World Health Organization identifies extensively drug-resistant tuberculosis

2009
Telavancin, approved by the FDA to treat MRSA, represents only the fourth new class of antibiotic approved since 1998

2014
President Obama asks for $30 million to fight antibiotic resistance

FROM TOP: JOHN DURHAM/SCIENCE SOURCE; NIAID/FLICKR (CC BY 2.0)



Death by antibiotic In the classic model, antibiotics kill by attacking a specific target. But a new model proposed by Boston University systems biologist James Collins suggests that the drugs may also spark a cascade of reactions that results in cell-wide damage by hydroxyl radicals and contributes to the drugs' deadly effects.

off its own cascade of collateral damage.

Superoxide, a type of reactive oxygen species made of two oxygen atoms, can blast open clusters of iron and sulfur found on certain proteins. Chemically available iron can mix with hydrogen peroxide, another noxious scrap of power production, to create hydroxyl radicals, a potent reactive oxygen species. These dreaded atomic bandits violently raid the cell, indiscriminately hacking electrons off molecules and wrecking DNA, cell membranes and protein-production machinery. The rampaging radicals can quickly destroy the whole cell.

To see if reactive oxygen species are generally involved in antibiotic killing sprees, Collins and his team turned to the two other classes of deadly drugs. One is the aminoglycosides, represented in the studies by kanamycin, which target the cellular equipment that translates RNA messages into new proteins. The team also looked at beta-lactam antibiotics, such as penicillin and ampicillin, which hack apart links in bacterial cell walls.

To monitor for antibiotic aftereffects, Collins and his colleagues set a flashy bait in bacterial cells — a molecule that glows when cut by hydroxyl radicals. In each case, the antibiotics triggered a flare of fluorescence, indicating a cascade of damage similar to that set off by the quinolones.

Next, the team sprinkled in another chemical, called thiourea, which can quell rebellious reactive oxygen species. With the chemical addition, researchers stifled the fluorescent flash, fueling the hypothesis of savage side effects. Plumbing deeper into dying bacteria, Collins and his team found more genetic and molecular evidence of off-target impacts. They monitored a coenzyme called nicotinamide adenine dinucleotide, which is crucial to cellular energy production, and found its activity boosted. Genes that control protein repair, such as *hslU*, and systems of DNA repair, such as the SOS response, were engaged. Mutant bacteria with no functional SOS response were more vulnerable to the three classes of antibiotics studied.

In 2007 in *Cell* and in 2010 in *Nature Reviews Microbiology*, Collins and his colleagues laid out a generalized model of how these classes of antibiotics set off a chain reaction leading to reactive oxygen species and death. Collins is careful to point out that the model isn't suggesting an overhaul to everything scientists know about antibiotics, nor is it saying that reactive oxygen species are solely killing the cells. "We're not overturning, but expanding upon," accepted thinking, he says.

Opposition forces

That cautionary stance comes after a spate of fiery criticism. Last year in *Science*, back-to-back papers blasted Collins' model, rejecting his experimental design and pointing out alternative interpretations of the data.

Imlay, lead author of one critical paper and an expert on reactive oxygen species, raised an eyebrow to Collins' unconventional systems approach to investigating antibiotic assassinations. "It's a great idea," he says. But the theory that hydroxyl radicals play a role doesn't hold up, he says. "If it were true, it would reveal something new about how reactive oxygen species would be made."

In Collins' model, the cell's power plants kick off the cascade with spewed waste. But Imlay argues that energy production, even when cranked up, is a wimpy starter for the full-scale hydroxyl radical assault needed to kill off a cell. It usually makes only a small amount, he explains.

With a phone call to Collins' lab, Imlay's group got the protocol for some of the experiments. The team could repeat the results, but found contrary

B. RAKOUSKAS; DNA ICON: DANMITCHELL/ISTOCKPHOTO

data as well. The chemical bait that glows when cleaved by reactive oxygen species is not that specific. It can be hacked by other molecules produced during antibiotic death, Imlay says. When his researchers looked for a boost in free iron, central to the creation of hydroxyl radicals, they couldn't detect it. "That's the key reaction," Imlay says. "And we measured it and did not see a change."

Despite Collins' finding that certain emergency repair systems spring into action during an antibiotic siege, Imlay points out that OxyR, a hallmark system for repairing damage from reactive oxygen species, was missing. And, when Imlay and coauthor Yuanyuan Liu looked at antibiotic attacks in the absence of oxygen, which is essential for forming damaging oxygen species, they found that the drugs were still lethal. The finding suggests that oxygen species aren't required for — though they still could play a role in — death.

A second *Science* paper, coauthored by biologist Iris Keren, a former member of Collins' lab, reexamined each experiment in Collins' 2007 original paper in detail, pointing out flaws. Keren, who has since left academic research, did not respond to interview requests.

Though Keren's personal connection to Collins has led many in the field to question the motivation of her work, the data have taken a toll on the reactive oxygen theory. "There is, I think, a fairly widespread perception that the overall Collins model has been disproven," says molecular biologist Graham Walker of MIT, who has found supporting data for Collins' model. Many doubt that it's a universal explanation of how antibiotics kill.

But research continues. A battery of studies, including Walker's, has found evidence of oxygen radicals killing cells alongside antibiotics in a variety of types of bacteria. In a 2009 study in *Antimicrobial Agents and Chemotherapy* led by microbiologist Xilin Zhao of Rutgers' New Jersey Medical School, bacteria genetically engineered to lack metabolic enzymes that make hydrogen peroxide (a precursor to hydroxyl radicals) survived what should have been a lethal antibiotic attack. In 2012 in *Science*, Walker and colleagues published data suggesting that drug-derived reactive oxygen species damage the cell's warehoused guanine bases — the G in the genetic code — furthering the central idea of Collins' model.

The model has been particularly useful to tuberculosis researchers. In 2011, infectious disease researcher Harvey Rubin of the University of Pennsylvania and colleagues found evidence that clofazimine, a type of antibiotic that fights leprosy and drug-resistant tuberculosis, also sparks the formation of lethal reactive oxygen species in microbes. "There's no question in my mind," Rubin says. "In our hands, reactive oxygen species can be generated and can kill cells." Rubin's team published its results in the *Journal of Biological Chemistry*. In 2012, molecular biologist Deborah Hung of Harvard and her team built on the clofazimine data, reporting in the *Proceedings of the National Academy of Sciences* that the same damaging molecules could also annihilate lingering tuberculosis-causing microbes.

The conflicting data leave lingering questions about how antibiotics kill, says Wright. "There's not a one-size-fits-all model." But he's reluctant to say that reactive oxygen species play no role. "I think that's unlikely," he adds. "What the role of reactive oxygen species is is going to be an interesting thing to figure out."

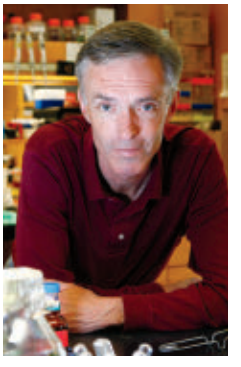
Therapeutic fallout

Some worry that the backlash over Collins' initial data has stalled efforts to make current antibiotics more effective. "The big deal is that we have squandered one of the most valuable medical advances of all time and we're losing it," Drlica says of antibiotic therapy. "If you don't believe that Collins is fundamentally right, then you won't pursue that line of investigation. That's why it's an important controversy."

A handful of researchers, including Rubin and Walker, continue to explore the idea. In 2012, Collins cofounded EnBiotix with Wager, a venture capitalist. "Is there more to learn about how this mechanism works? Of course," Wager says. "That doesn't shake our confidence one iota regarding the validity of the general hypothesis." EnBiotix aims to boost the amount of reactive oxygen species an antibiotic creates, thereby fortifying its killing ability, he says.

For Collins, continuing to study how current drugs kill is an obvious way forward. "We still have only scratched the surface of what's happening with these drugs," he says. Collins and his lab have written a paper, expected later this spring, that backs the reactive oxygen species model and addresses the weak points that Imlay's paper pointed out. The debate isn't over, he says. Yet more studies will be needed to completely understand how antibiotics kill and how to make them kill even better.

"Death," he adds, "is a complicated process." ■



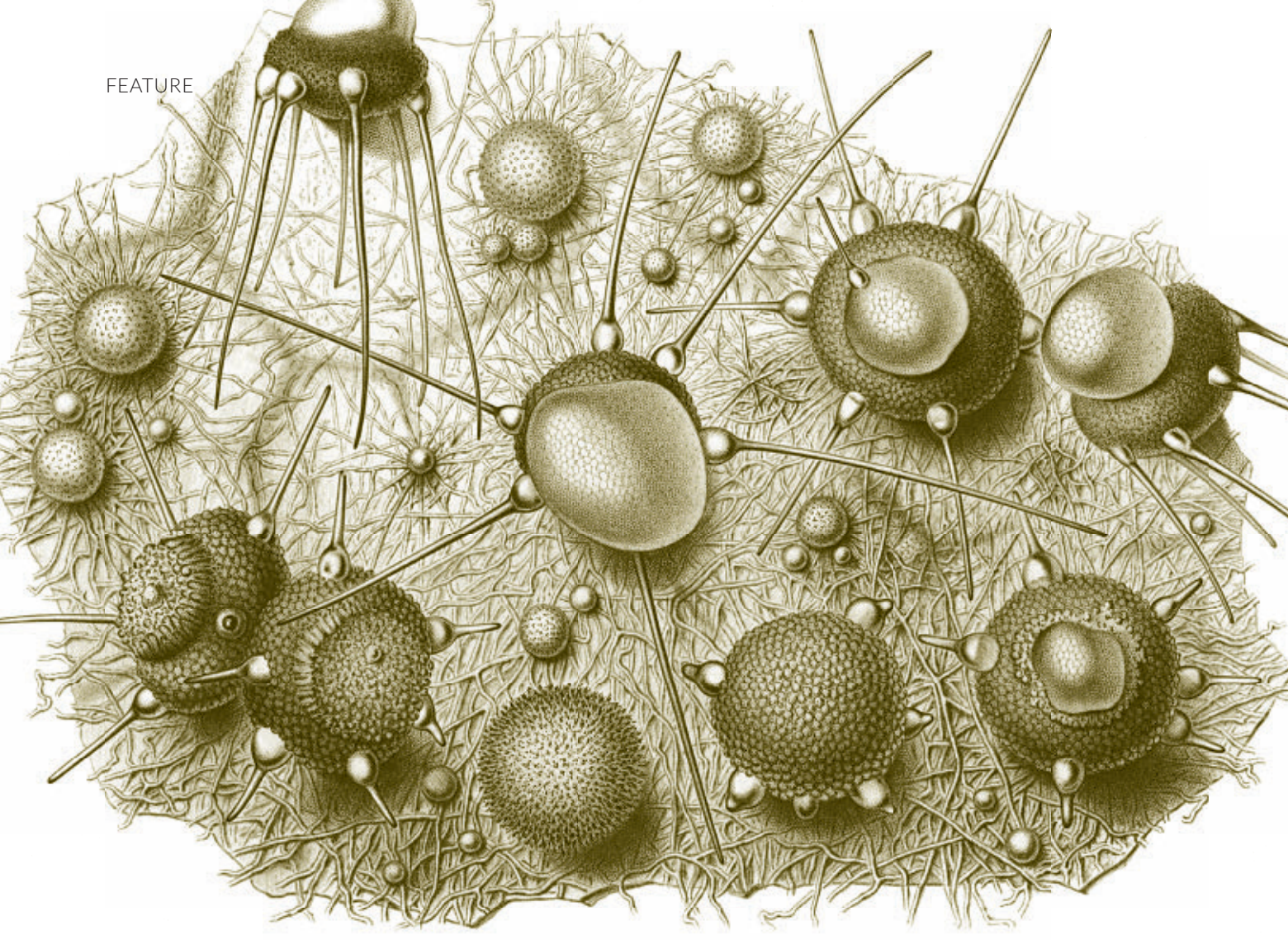
ROBERT E. KLEIN

"We still have only scratched the surface of what's happening with these drugs."

JAMES COLLINS

Explore more

- Collins lab website: bit.ly/SNCollins



L. R. AND C. TULASNE/*SELECTA FUNGORUM CARPOLOGIA* 1861

The name of the fungus

Genetic advances spur mycologists to put their kingdom in order

By Susan Milius

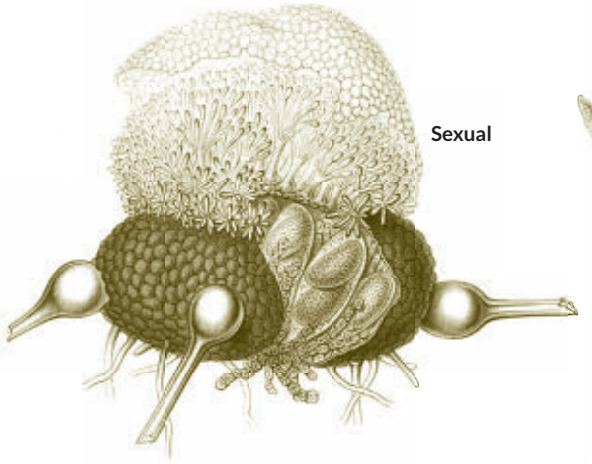
To a visitor walking down, down, down the white cinder block stairwell and through metal doors into the basement, Building 010A takes on the hushed, mile-long-beige-corridor feel of some secret government installation in a blockbuster movie.

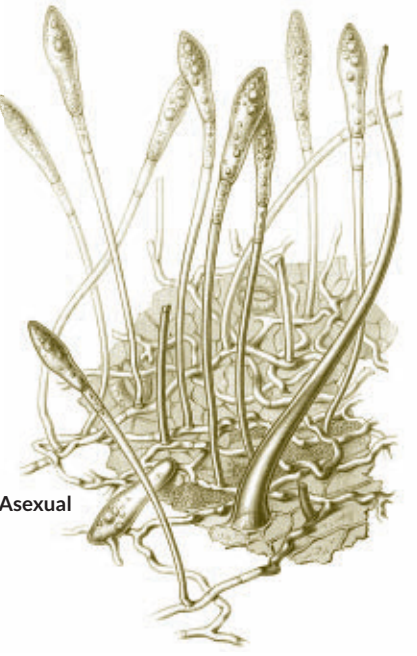
It's not open to sightseers, but it's far from secret. No jut-jawed military escort leads the way; biologist Shannon Dominick wears a striped sweater as she strolls through this Fort Knox of fungus, merrily discussing certain specimens in the vaults that are commonly called "dog vomit fungi."

This basement on the campus of the Agricultural Research Service in Beltsville, Md., holds the second largest fungus collection in the world, with at least 1,000,000 specimens. Snuggled into exquisitely customized boxes and folders stored in high banks of institutional-beige metal cabinets are organisms that can glow in the dark, turn living ants into leaf-biting zombies, fetch thousands of dollars per pound at gourmet food shops or snarl international commodities markets.

It may look like the ultimate triumph of human order over natural chaos. But with fungi, it turns out that looks can be deceiving.

Many fungi are shape-shifters seemingly designed to defy human efforts at categorization. The same species, sometimes the same individual, can reproduce two ways: sexually, by mixing genes with a partner of the same species, or asexually, by cloning to produce genetically identical offspring.





Early warning Long before DNA analysis, the sharp-eyed brothers Charles (left photo) and Louis René Tulasne (right photo) realized that the same fungal species can take different forms. In 1861 the brothers published drawings of a powdery mildew fungus that sometimes forms clusters of dark, spiked orbs (left drawing) or microforests of spore-bearing hairs (right drawing), now known to be sexual and asexual forms. At the time, the Tulasnes labeled both forms *Erysiphe guttata* (facing page).

The problem is that reproductive modes can take entirely different anatomical forms. A species that looks like a miniature corn dog when it is reproducing sexually might look like fuzzy white twigs when it is in cloning mode. A gray smudge on a sunflower seed head might just be the asexually reproducing counterpart of a tiny satellite dish–shaped thing. Just by looking at them, you'd never know.

When many of these pairs were discovered, sometimes decades apart, sometimes growing right next to each other, it was difficult or impossible to demonstrate that they were the same thing. So one species would get two names. Careful observation later suggested that officially different species are actually one, but the pairs of names remained. In fact, it soon became standard mycological practice to name many species twice — once for the sexual form, once for the asexual one.

"Zoologists," says mycologist David Hawksworth of Universidad Complutense de Madrid, "throw up their hands in horror." For animals, one name covers the caterpillar and the butterfly it becomes. Botanists don't just go naming a big tuft of fern fronds one thing and its separate, little green gametophyte another.

Yet until recently, mycologists had no choice. They knew full well that *Aspergillus flavus*, the powdery fuzz that taints peanuts with carcinogenic aflatoxin, is the exact same species as *Petromyces flavus*. They knew that *Cordyceps takaomontana* is also *Isaria tenuipes*. But faced with such a dizzying array of shape-shifters, they had to allow different names for things that might or might not be the same species.

Now, mycologists have a chance to set the record straight. A group of upstart scientists has rebelled against the dual-naming system, arguing that DNA analysis can endow fungi with a one-species, one-name system. Having won a major victory at a recent international scientific congress, they are poised to bring their field into a new era of genetic nomenclature. But however justified genetically, their project is not without perils.

6,995

Number of fungal genus names proposed for protection

What's in a name

Naming a species is not just a Latin version of deciding that your new kitten seems more like a Snowball than a Bobo. It means deciding what an unknown entity, in the most basic sense, is. It hangs a living thing on a new twig of the tree of life, showing how shared characteristics reveal its relatedness to known forms, but also arguing that unique characteristics distinguish it from every species described so far.

People have been guessing wrong for millennia about where fungi fit and what they are. Aristotle, and then about 2,000 years later, Carl Linnaeus, who fathered the system of two-word Latin names, divided the living world into the plantlike and the animal-like. Both put fungi among the plantlike.

Fungi finally got their own kingdom in the 1960s, and since the early 1990s genetic evidence has been building to place the fungal kingdom closer to animals than to plants. Analyses have also shown that some things that look and act like fungi actually belong in other taxonomic groups. Slime molds, including the dog-vomit–like specimens in the national collection, are not fungi but protists. And the *Phytophthora*, including the *infestans* species famous for causing the Irish potato blight, are closer to some kinds of algae than to fungi.

Linnaeus' observational approach has been no match for a group of organisms that is often not what it seems.

"He was hopeless," Hawksworth says. "He really lost the plot when it came to fungi."

To be fair, evolution's plot is hard to follow when all you've got to go on are five senses and the occasional chemical probe of the blobs, stalks, specks and toadstools that populate the fungal kingdom. But long before high-throughput DNA sequencing gave researchers the ability to essentially reassemble evolutionary history, there were hints of something funny going on.

By the mid-19th century, the French brothers Charles and Louis René Tulasne were fretting that what appeared under a microscope to be the same fungus sprouted into different kinds

PHOTOS FROM LEFT: CONVALLARIA MAJALIS/WIKIMEDIA COMMONS; MU/WIKIMEDIA COMMONS; DRAWINGS: L. R. AND C. TULASNE/*SELECTA FUNGORUM CARPOLOGIA* 1861

The U.S. National Fungus Collections house more than 1 million specimens, including slime molds (left), now considered part of a different biological kingdom, and the type specimen that defines the leaf-attacking species *Phyllachora concentrica* (right).

of reproductive forms. The brothers were "incredible observers," Hawksworth says. By studying details in structures, the Tulasnes realized that at least some fungi sprouted more than one kind of reproductive "seed," as they called the sexual and asexual products. The Linnaean system, assigning names to a species based on its supposedly uniform reproductive anatomy, was in trouble with fungi.

"But since the illustrious author," the brothers wrote in 1861 of Linnaeus, "always completely abjured the use of magnifying glasses, and therefore scarcely ever tried to describe accurately either conidia or spores, we fear (may he pardon the statement) that he really knew very few seeds of either kind."

> "The mycological establishment didn't want to accept what they were finding."
>
> DAVID HAWKSWORTH

At Beltsville's Systemic Mycology and Microbiology Lab, mycologist Amy Rossman pulls out three tall volumes by the Tulasnes, with full-page plates of delicate line drawings of nubbled globes or fatly bending projections, the brothers' minute records of fungal details. Rossman has donned white gloves to protect the old volumes, giving the book viewing a touch of past-century formality.

The lovely drawings failed to inspire much discussion about what to do with names. The brothers "became rather reclusive," Hawksworth says. By and large, "the mycological establishment didn't want to accept what they were finding."

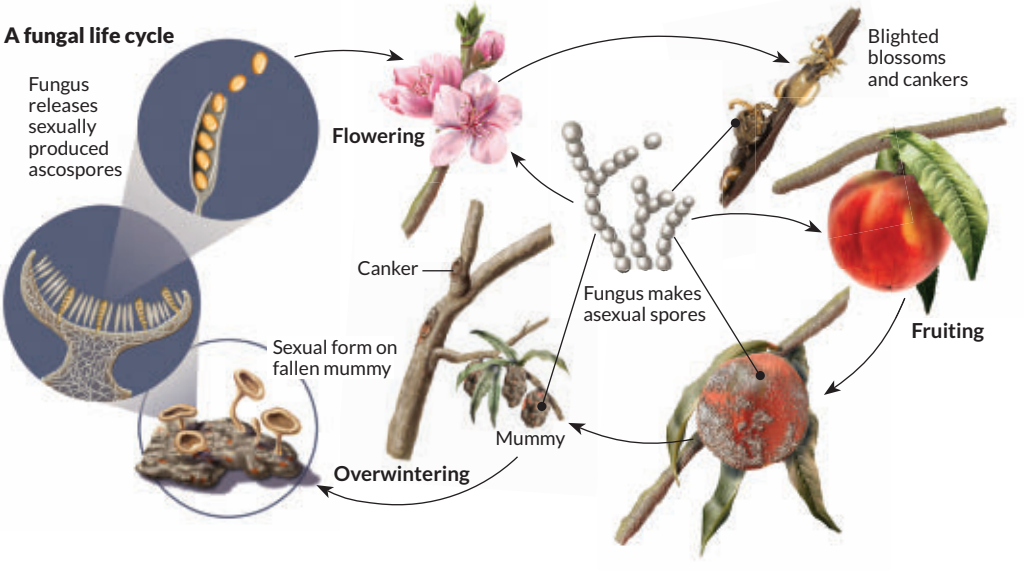
Before DNA tools, it was risky to claim that two fungal forms should be classified as identical species just because they were found in intimate contact. A parasitic species growing on its host could create the illusion of an alternative form. "People didn't want to assume all those [claims] were correct after some unfortunate incidents were discovered," Hawksworth says.

By the early 20th century it had become clear that many fungi could shift between one or more asexual forms, called anamorphs, and a sexual form called a teleomorph. But matching up the forms for the same species could be so difficult that mycologists devised what Lorelei Norvell of Portland, Ore., longtime editor in chief of the journal *Mycotaxon*, calls "a gentleman's agreement" to use two names.

The same plump, color-coded volumes of legalese that govern how to name plants and algae also cover fungi, a carryover from the centuries of treating fungi as plants. "If you have ever read the Internal Revenue Code," Norvell says of these rules, "it's the same sort of thrill."

By 1910, the code's authors had agreed on an early version of a rule that has become so widely discussed that it's just known as Article 59, as if it were a landmark ballot proposition. In two large fungal groups (Basidiomycetes and Ascomycetes that don't make lichens), the rule allowed fungi to have both a sexual and an asexual name. For speaking of the whole organism, the sexual name dominated. In practice, researchers often focused on just one form or the other, and names proliferated.

Shape-shifters Many fungi can reproduce either sexually or asexually, depending on circumstances. In *Monilinia fructicola*, which causes brown rot in fruit, spores wafting onto blossoms in spring can grow and produce asexual spores that spread to new blossoms, twigs or fruits and create more asexual spores. On rare occasions, when an infected fruit plops to the ground, one fungus finds a mate and forms a sexually reproducing stalked form. The stalked cups look nothing like the asexual fur on fruits and flowers.



CLOCKWISE FROM TOP LEFT: S. MILIUS; PEGGY GREB/USDA; NICOLLE RAGER FULLER

Until recently, fungal taxonomy has given the sexually and asexually reproducing forms of a single species different Latin names. In the foreground is the fungus long known as *Cordyceps takaomontana*; in the background is *Isaria tenuipes*, the name given to the asexual stage of the same fungus.

Name changer

The two-name system for fungi inspired grumbling and various upsets, and Article 59 went through revisions and re-revisions. But the current turmoil started with the advent of molecular tools that at last allowed people to look fungi right in the DNA. Now it doesn't matter if two specimens look entirely different and are never found together. If their DNA matches, they're the same species. DNA also lets mycologists position all those species on the fungal evolutionary tree, even if there's nothing more to go on than asexually reproducing fuzzes.

The movement eventually sparked outright rebellions. In 2006, Pedro Crous of the CBS-KNAW Fungal Biodiversity Centre in Utrecht, the Netherlands, penned a treatise on a big family of fungi, the Botryosphaeriaceae, and threw down a taxonomic gauntlet. He declared that he would not be citing both sexual and asexual names "even where both morphs are known."

Jos Houbraken, also at the Fungal Biodiversity Center, maintains a noninflammatory tone no matter what a reporter asks about the tumult in nomenclature. When he made his own early switch to a one-name system, he says that reviewers of his submitted papers flagged the unconventional names as against the nomenclature code. In one case a reviewer objected that "pragmatism is not enough" to justify violating the rules. But other reviewers noted the off-code names supportively, and the journal published the paper.

An international committee chaired by Rossman attempted to devise ways to move from double names to single names. But by 2010, after four years, the committee was so deadlocked that its secretary used the term "dysfunctional" in his report in the journal *Taxon*.

Changing Latin names does have its risks. Not everyone who deals with fungi is a trained taxonomist: Gardeners staring at spots on their boxwood stems, food mycologists checking spoilage in baked goods or regulators writing quarantine rules might easily overlook important information if it's filed under a different name from the one they've always used. Debates over Latin names can have real-world consequences.

A fungus by any other name

Lobster mushrooms

These mushrooms beloved of foragers are composites of a less appealing mushroom and the parasite often called *Hypomyces lactifluorum*. That genus name, from 1860, could get chosen over the name *Cladobotryum*, given to asexual forms in 1816.

Boxwood blight

Cylindrocladium fungi, with an asexual genus name from 1892, will probably be switched to the even more venerable *Calonectria*, the name given to the sexual state in 1867. By any name, an invasive *Cylindrocladium* has hit boxwood growers hard in recent years.

Asterophora

Asterophora fungi, such as *A. lycoperdoides*, sprout tiny mushrooms on not-so-tiny mushrooms. The genus name dates from 1809, so mycologists may save the relatively widely used name from being switched to an older asexual genus name, *Ugola*, from 1763.

CLOCKWISE FROM TOP: ALISHA OWENSBY/OREGON STATE UNIVERSITY (CC BY-NC-SA 3.0 US); MALCOLM STOREY/BIOIMAGES; MARGERY DAUGHTREY/CORNELL UNIV.; J. MICHAEL RABY/FLICKR (CC BY-NC-SA 2.0)

Monilinia vaccinii-corymbosi *Monilia vaccinii-corymbosi*

Proposed for both: *Monilinia vaccinii-corymbosi*

Mummy's curse Brown cups (left) sprouting from fallen blueberries represent the sexual phase of the fungus that causes mummy berry disease. Its asexual phase looks like white fuzz (right). Mycologists, choosing one name for the species, may preserve the more common sexual name.

The Melbourne incident

Actually changing fungal nomenclature rules requires discussion and voting at an international congress that is attended mostly by botanists. Thanks to the long tradition of treating fungi as plantlike, botanists get a vote on mycological matters. There has been mention of secession from the botanical code, but so far it's just been talk.

In July 2011, about a dozen mycologists found themselves in Melbourne, Australia, at a special session held a week before the International Botanical Congress dedicated to nomenclature changes. Mycologists were outnumbered almost 20-to-1 by botanists, and after years of deadlock were "anticipating a bloody fight," Norvell says. To top it off, many of the scientists were ill. "There was a crud going around," she recalls. "Everybody had it, and there was coughing like you wouldn't believe."

The battle-weary secretary of the deadlocked committee, Scott Redhead of Agriculture and Agri-Food Canada in Ottawa, had prepared three options for changing the two-name rules. He started with the most radical and least likely to succeed: Change the old Article 59 to remove the option for double names. "Here we are, braced for discussion, braced for a fight, and they take a vote on the first option — delete 59 — and it passes." Norvell says. "We just sat there looking like fish with our mouths open."

A tiny band of rebels, with help from a foreign power, had suddenly overthrown a century-old regime. Not all mycologists were pleased.

The single-name motion may have passed among congress attendees, but support for it among fungal taxonomists overall ran only about 50-50, says mycologist Keith Seifert at Agriculture and Agri-Food Canada. "Our side happened to win the vote," he says. "Many fungal taxonomists feel disenfranchised, cheated and angry."

Hawksworth is more optimistic. "There was a lot of backlash initially," he says, but some who objected strenuously are now settling down to the immense task ahead. And it is immense: Taxonomists have to sort out which former names will be the ones to use for perhaps more than 10,000 fungi.

Sometimes it's simple. Seifert takes the example of *Aspergillus flavus*, the asexual name for the powdery fuzz that is notorious for releasing carcinogenic aflatoxins into stored peanuts, corn and other commodities. Farmers, regulation writers, doctors and industrial microbiologists have long used the name *A. flavus*, and they may not have even realized that a sexual form was discovered and named *Petromyces flavus* in 2009. The new version of Article 59 gives priority to older names, so *Aspergillus*, from 1729, trumps *Petromyces*, from 1973. And the second words in the names are the same. Big sigh of relief: The familiar name prevails.

In cases where a familiar name could get bumped by an obscure old one, the new code intentionally leaves a huge loophole: Taxonomists may petition to give priority to a widely used name regardless of its age. This is the part that worries Seifert.

"We need to get our buns in gear," he says. Appeals to save names need to be ready for discussion in August at the 10th International Mycological Congress in Bangkok (where the botanists won't be in attendance). Otherwise those names can't be approved at the next botanical nomenclature congress, in 2017. Missing the August deadline would mean that cherished names couldn't go before a nomenclature congress until 2023, creating a long period of uncertainty. Yet efforts to select names to preserve are lagging. "Despite what some people might hope, there is no going back," Seifert says.

Shifting to single names gets especially tricky as DNA analyses keep redrawing species boundaries and relationships between taxonomic groups. Houbraken and his colleagues have concluded that the storied fungus long known as *Penicillium chrysogenum*, which gave Alexander Fleming his antibiotics, is actually *P. rubens*. And the fungus causing the disease penicilliosis in people with compromised immune systems doesn't really belong in the genus *Penicillium* at all.

It's a lot to deal with. But as dramatic as all the rethinking and renaming have been, some mycologists propose taking the implications of DNA analysis even further into the taxonomic frontier. Systems for processing massive quantities of genetic material allow mycologists to explore communities of fungi they can't identify in lab dishes. Soils, swamp glop, animal rumens and even human feet yield genetic signatures of distinctive fungi that no microscope has ever focused on. If DNA can sort out fungal species, then why not use it for naming in cases where no specimen can be found?

Vexed by how to settle on one name for a confusing multitude of physical forms, mycologists may soon have to figure out how to name a thing when there is nothing to see at all. ■

Explore more

■ A. Rossman and M. E. Palm-Hernández. "Systematics of plant pathogenic fungi: Why it matters." *Plant Disease*. October 2008.

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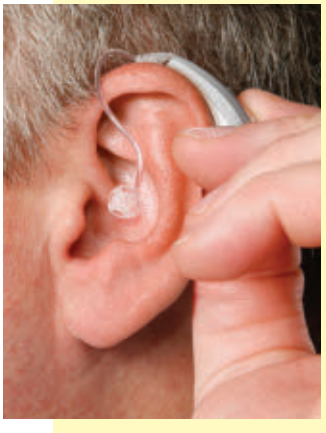

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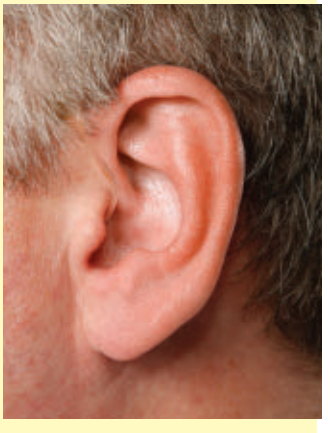

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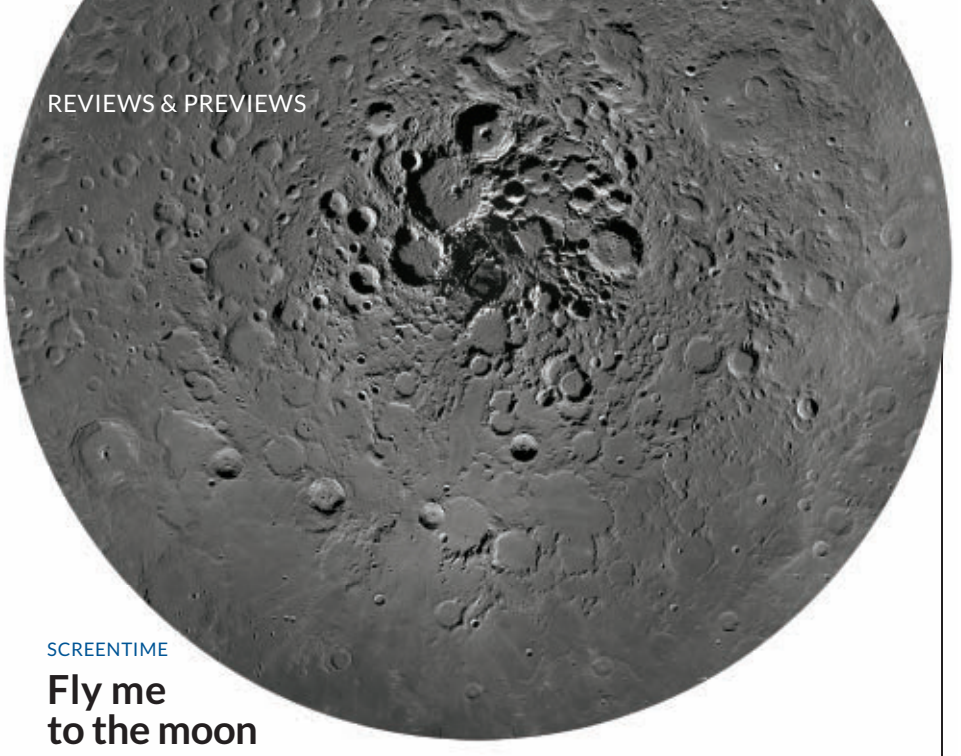

SCREENTIME

Fly me to the moon

If a private spaceflight doesn't fit your travel budget, NASA's new interactive lunar atlas may be the next best thing. The Lunar Reconnaissance Orbiter team has stitched 10,581 images into a gorgeous, detailed online map of the moon's north pole. The mosaic covers 2.54 million square kilometers — slightly larger than the combined areas of Alaska and Texas. The detail is stunning: At the highest zoom, each pixel shows a piece of the moon just 2 meters across. Anyone who has used online maps will be familiar with the interface. Simple pan and zoom buttons allow users to soar and swoop over the lunar landscape. Dynamically updated labels mark interesting sites and keep you oriented. The subtleties visible in the terrain are remarkable. You can see melted rock at impact sites, tracks from rolling boulders and fissures crisscrossing crater floors. Each vista highlights the cold beauty of our celestial neighbor and shows that the moon is an active, changing world. — *Christopher Crockett*

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BOOKSHELF

You Are Here

From the Compass to GPS, the History and Future of How We Find Ourselves

Hiawatha Bray

These days, it can be almost impossible to get lost. The creation of affordable smartphones has put personal homing beacons into over a billion pockets and pocketbooks, enabling even the most directionally challenged to locate the nearest Starbucks or find their way around a traffic accident. Yet the technology that enables easy navigation was centuries — even millennia — in the making. *Boston Globe* technology reporter Hiawatha Bray chronicles the evolution of navigational and mapmaking tools and how they have shaped modern society.

First invented to aid in exploration, then to guide instruments of war and to spy on Cold War enemies, location technology is now being used to track regular people. Governments and corporations can follow the movements of citizens and consumers, for peacekeeping or for profit. Over half of the most popular smartphone apps continue to transmit your whereabouts to outside parties such as advertisers or law enforcement, long after you've tired of *Candy Crush* or *Angry Birds*. Bray's account, at times exhausting but always informative, clearly conveys the scientific thinking and extraordinary effort that made location tracking possible. It took more than twice as long to complete the network of GPS satellites necessary to pinpoint your place on Earth, for instance, as it did to put a man on the moon.

The book raises concerns about the privacy that so many of us casually trade in during our pursuit of a latte or a faster route to work. Bray advocates for anonymizing features that will allow people to go off the grid from time to time while still enjoying the fruits of technology. That way, you and you alone will know that "you are here." — *Marla Vacek Broadfoot*
Basic Books, $27.99

FROM TOP LEFT: NASA/GSFC/ARIZONA STATE UNIV.; PURCHASED FOR THE NY HISTORICAL SOCIETY BY PUBLIC SUBSCRIPTION FROM MRS. JOHN J. AUDUBON

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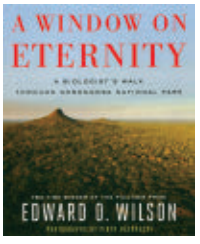
BOOKSHELF

A Window on Eternity

A Biologist's Walk through Gorongosa National Park

Edward O. Wilson



Starting in the late 1970s, Mozambique spent more than a decade embroiled in a brutal civil war that left millions dead or displaced. The effects of the human conflict echoed through the natural world. Soldiers encamped in Mozambique's Gorongosa National Park, an area rich in flora and fauna, hunted elephants, zebras, Cape buffalo and other animals for food. Populations of the big mammals dwindled to almost nothing.

In his latest book, entomologist E.O. Wilson chronicles both the shifting ecology of Gorongosa after the war and how researchers are trying to repair the damage. Without key species, the park began to change. Trees that previously had been bulldozed by elephants flourished. With no grazers to prune them back, low-growing plants provided abundant timber for wildfires. Dung- and carrion-eating insects died off.

Wilson, who is involved in the recovery efforts, offers an intimate view of some of the less-than-obvious effects of the conflict. The park's elephant population had dropped by as much as 90 percent by 2001, for example, and though numbers are now growing, biologists report that older individuals seem to be suffering from a kind of pachyderm PTSD. They have been left wary and more likely to charge vehicles.

Though Wilson's enthusiasm for the park is infectious, it's the photographs by entomologist Piotr Naskrecki that steal the show. Naskrecki's images are a delight, capturing the spirit of the recovering landscape and its animals, great and small.

Gorongosa has come a long way and still has a ways to go, says Wilson. Ultimately, the book is a cautionary tale about how human affairs are fundamentally entangled with the natural world. It's an age-old message, but one that's worth a reminder. —*Allison Bohac*
Simon & Schuster, $30

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MARCH 22, 2014

Estimated annual bird deaths in the United States by cause

CATS
1.3–4.0
billion

WINDOW COLLISIONS
365–988
million

WIND TURBINES
573,000

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Options for treating addiction

*Addiction is often seen as a chronic disease, but some long-term studies suggest it can be viewed as a temporary coping problem instead. **Bruce Bower** presented this alternative view in "The addiction paradox" (*SN: 3/22/14, p. 16*).*

"A nice job by Bruce Bower, as usual. I don't think it helps when researchers dichotomize conditions as behavioral or biological. Heart disease and diabetes are biological conditions, but they can be ameliorated by behavioral changes involving diet and exercise," noted **William Check** in an e-mail. "Life circumstances make a difference. It seems to me that a menu of treatment options — or nontreatment — makes the most sense."

The clear killers

*As many as 988 million birds die from slamming into windows each year in the United States, according to a new estimate reported by **Susan Milius** in "Windows are major bird killers" (*SN: 3/22/14, p. 8*).*

"So can they stop blaming windmill power generators [for killing birds] now?" asked online reader **bobfairlane**. "Not really, no," replied reader **RobertPPruitt**. "The problem with windmills isn't the total number of birds killed. It's the number killed compared to the number of windmills. There just aren't all that many windmills compared to windows. The biggest problem is the simple fact that windmills seem to work best in areas with a higher and more consistent wind speed. But those also happen to be the same areas that migratory birds prefer to use to travel long distances."

The online title for the story, "Windows may kill up to 988 million birds a year in the United States," prompted some readers to imagine a very different kind of threat. "I always thought that Windows was a big problem. OS X is much better," joked **Casey Burns**. "Microsoft should be ashamed!"

Cloud clarifications

*Clouds pose a conundrum for atmospheric scientists, since they have the potential to either accelerate or dampen global warming. In "Cloudy forecast" (*SN: 3/22/14, p. 22*), **Gabriel Popkin** discussed some of the challenges facing researchers as they try to sort out clouds' influence on climate.*

"In Gabriel Popkin's very interesting and educational article, cloud physicist Ulrike Lohmann is quoted as saying, 'The amount of ice in the atmosphere seems to exceed the amount of liquid almost everywhere,'" **George Sitts** wrote in an e-mail. "Our planet is home to a great amount of liquid water, from the seas, lakes, underground aquifers and cloud droplets. It would be easy to infer from her statement that ice in the atmosphere exceeds almost all of our planet's liquid water. That sounds incredible and unlikely."

Popkin says that Lohmann was indeed referring to only the liquid in clouds. "The reader is correct that when taking into account all of Earth's water, liquid is far more abundant than atmospheric ice."

Douglas B. Quine notes that among the many factors affecting cloud cover that were discussed in the article, one was left out: time of day. "Clouds cool through reflection during the day, and they reflect a lot of energy back to Earth at night. The global balance between these two depends in part upon the time of day that clouds are present. Cloudy days and clear nights will enhance cooling, whereas clear days and cloudy nights will enhance heating. To the extent that cloud cover has circadian patterns, how much of a difference might these patterns make?"

Popkin says that while clouds do cool Earth during the day and warm it at night, whether shifts in clouds' daily patterns will alter climate is unclear. NASA atmospheric scientist **Anthony Del Genio** adds, "so far I haven't seen any evidence that shifts in the time of day when clouds occur are a big factor in the cloud feedback we predict, probably because the big cloud feedbacks are mostly over the ocean, where day-night differences are much smaller. But in principle, it could happen."

Distant swirling galaxy overshadowed by violent star killer

Sixty million light-years from Earth in the Fornax constellation, two neighboring galaxies have very different histories. The smaller galaxy, NGC 1317 (right), is an unremarkable spiral, not much different from the Milky Way. Its neighbor galaxy NGC 1316 has a more sordid past: It's a cannibal.

In this mosaic of images from the 2.2-meter telescope at La Silla Observatory in Chile, dark dust lanes and twisting tails betray a history of galactic collisions that have built NGC 1316 into the monster it is today. Streams of dust in the core are all that remain of a spiral galaxy torn apart by NGC 1316's gravity roughly 3 billion years ago. Around the edges of the galaxy, faint wisps of stars hurled into intergalactic space preserve the record of a lifetime of collisions.

With each collision, gas and dust feed a supermassive black hole in the core of NGC 1316 that weighs about as much as 150 million suns. Blasts of energy from the tempest swirling around the black hole make NGC 1316 one of the brightest sources of radio waves in the sky. — *Christopher Crockett*

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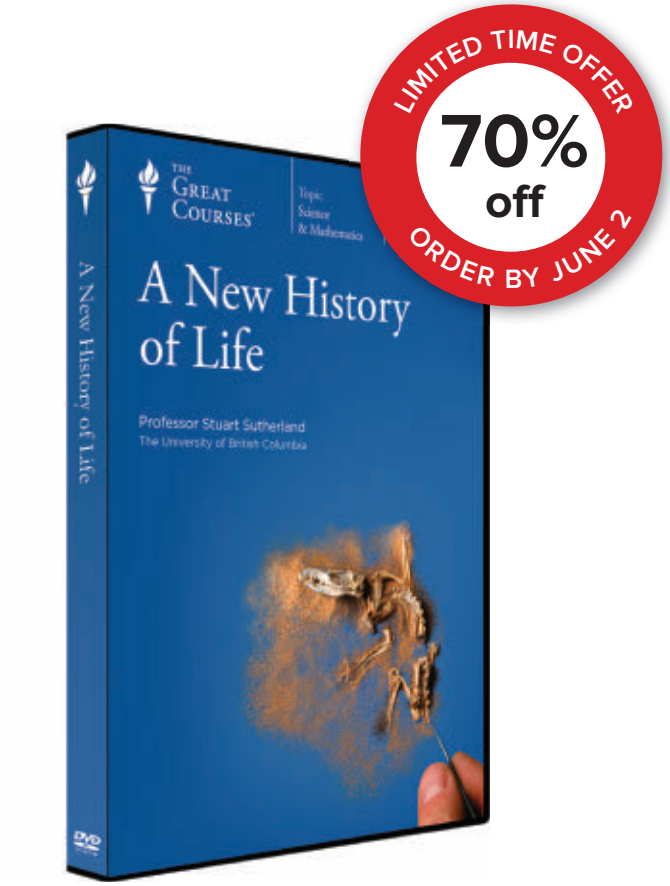

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