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ScienceNews

MAGAZINE OF THE SOCIETY FOR SCIENCE ■ JANUARY 13, 2024

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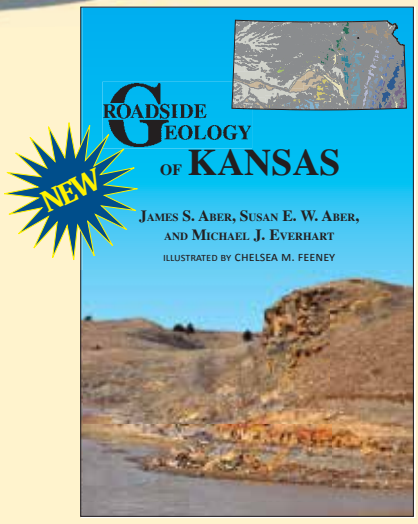

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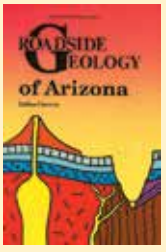
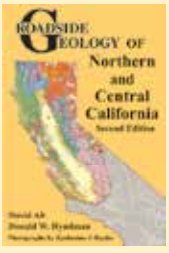
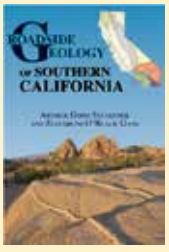
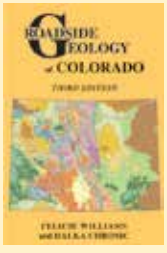
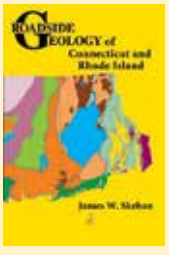
JAMES S. ABER, SUSAN E. W. ABER, AND MICHAEL J. EVERHART
318 pages, 6 x 9, paper $26.00

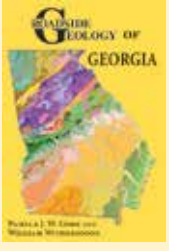


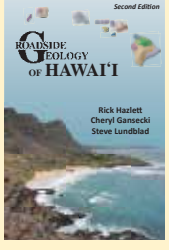


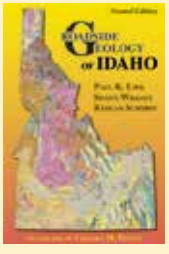


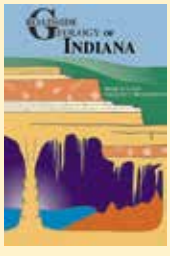


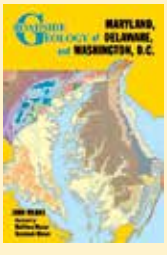


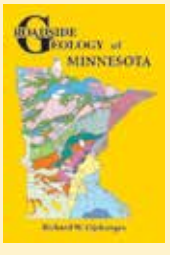


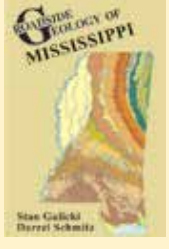


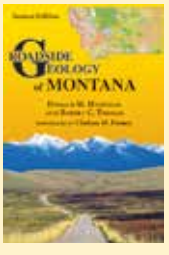


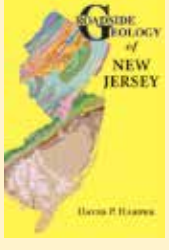


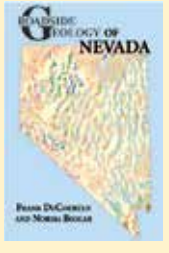


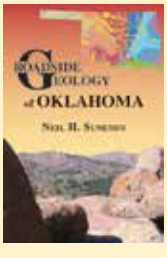


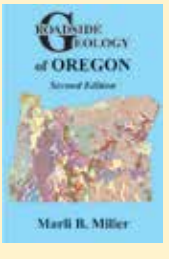


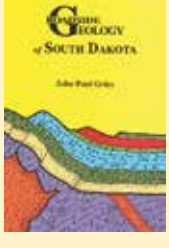


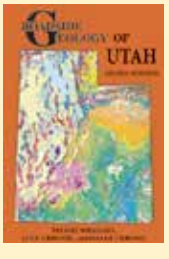


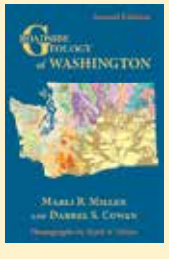


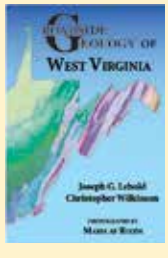


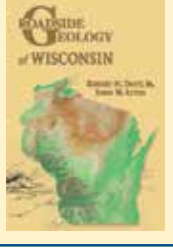


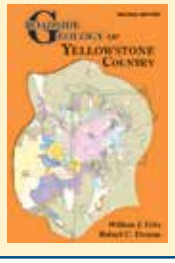












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VOL. 205 | NO. 1

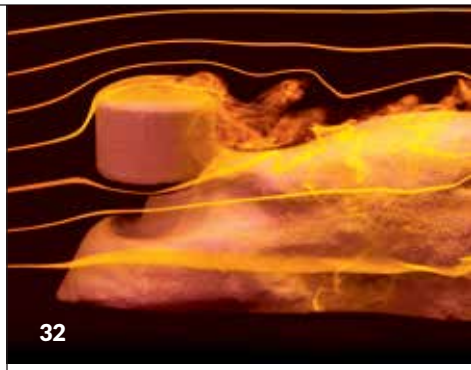
ScienceNews


18

Features

News


10


32

Departments

COVER Instead of prolonging life, the goal of geroscientists is to stave off some of the ailments that come with old age.
Sam Chivers

FROM TOP: THE UNSON FAMILY; APPLIED MATH LAB/NYU; W.Y. LEE

Bringing scientists' stories out of the shadows

When asked to name a scientist, most people's first pick is Albert Einstein. Marie Curie, Louis Pasteur and Thomas Edison also come up. But many people can't name a scientist at all.

When asked to name a living scientist, 72 percent of Americans came up blank in a 2021 survey. Among those who could name one, the top pick was Anthony Fauci, then director of the U.S. National Institute of Allergy and Infectious Diseases, followed by astronomer Neil deGrasse Tyson and Bill Nye, a mechanical engineer and host of the 1990s TV show *Bill Nye the Science Guy*.

So many more scientists deserve to be known, even if they never become household names. As journalists, we here at *Science News* are fortunate to be able to connect directly with scientists to get the scoop on their latest work, gain expert perspective and spot trends.

We increasingly try to spotlight scientists who should be better known, including early- and mid-career scientists who we feature in an annual series of profiles called the SN 10: Scientists to Watch (*SN: 12/2/23, p. 17*). We also connect with researchers, public health officials and citizen scientists around the world, as well as those in demographics underrepresented in science, including women, people of color and members of the LGBTQ+ community.

Bringing to light scientists who haven't been spotlighted previously requires detective work, especially with scientists from the past. In this issue, we profile Emma Rotor, a math teacher from the Philippines who played a key role in groundbreaking weapons research that advanced the Allied cause in World War II (Page 18). Our detective on the case was Erwin R. Tiongson, an economist at Georgetown University in Washington, D.C., and an amateur historian with a keen interest in Philippine–American history.

Emma had moved to the United States in 1941 with her husband, Arturo Rotor. Arturo was a renowned physician, author and musician. It was easy to find documentation of his activities. But the only mentions of Emma were as a supportive presence in the background, Tiongson told me.

"Growing up, I already heard so much about Dr. Rotor because my mother was a college literature professor," Tiongson said. "I always thought there must be more to Mrs. Rotor." He started digging and found a newspaper article saying that Emma was part of the Manhattan Project. He scoured lists of participants — no luck. Then he came across Emma's name in a citation of research on the proximity fuze, a new technology that radically improved the accuracy of Allied munitions. Emma had worked as a physicist for a group at what is now the National Institute of Standards and Technology.

Tiongson's research ultimately led him to relatives of Emma's in the Philippines with vivid memories of Tita Emma. One grandniece recalls her father asking, "Tita, did you work on the bomb?" No, Emma replied. "I worked on the fuze."

Emma Rotor's story is the most recent in our Unsung Characters series. We'll continue to connect our readers with scientists from the present and past who are worth knowing today. — *Nancy Shute, Editor in Chief*

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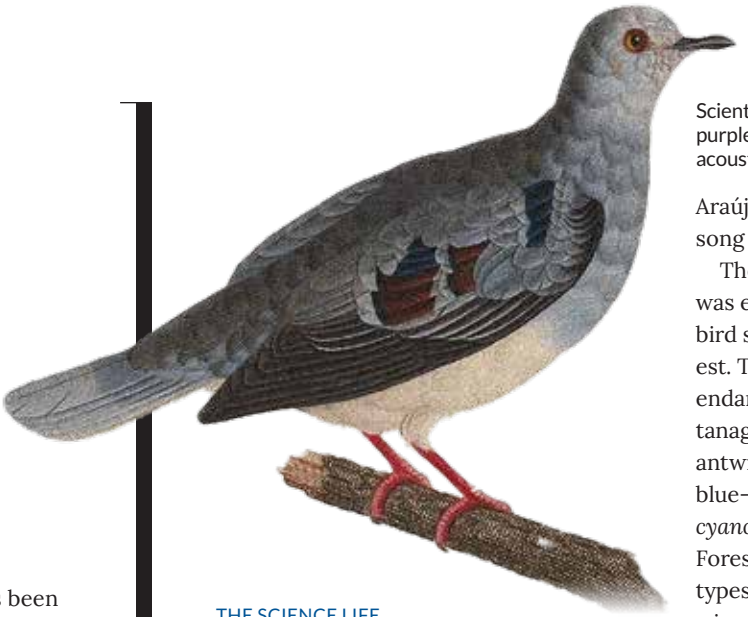
Excerpt from the January 12, 1974 issue of *Science News*

50 YEARS AGO

Happy leap second!

A "leap second" has been invented … to keep time signals used by navigators in step with the actual motion of the Earth. The latest leap second was celebrated New Year's Eve at the stroke of midnight Greenwich Mean Time, when around the world … radio stations added an extra "beep" to their hourly time signals.

UPDATE: Time is running out for the leap second. In 2022, metrologists voted to abandon the timekeeping quirk by 2035. Unlike the leap year, which occurs every four years, the leap second is deployed whenever clocks need adjusting due to variations in Earth's spin causing slight changes in the length of a day. Global officials have inserted a leap second 27 times since 1972. But satellites and other tech that rely on the precise time kept by atomic clocks can glitch when the clocks are adjusted (*SN: 4/22/06, p. 248*). Scientists have suggested using a leap minute instead, which would require atomic clocks be reset once every 50 years or so.



Scientists want to search for the long-missing purple-winged ground dove (illustrated) in acoustic data from forests.

THE SCIENCE LIFE

The song of a missing bird may help scientists find it

How do you look for an animal you don't even know exists anymore?

The last confirmed sighting of the purple-winged ground dove (*Paraclaravis geoffroyi*), a bamboo-loving bird native to the Atlantic Forest in Brazil, Argentina and Paraguay, was in 1985. But, researchers wondered, was it possible to capture the elusive bird's sound in the wild to find out if any individuals are left?

It's not an unheard-of idea. Scientists have used bioacoustics — a subfield of ecology that relies on sound to make environmental analyses — for studying bats from afar, dolphin communication patterns and more. With artificial intelligence, it is now possible to use large audio datasets to train algorithms to spot different animal sounds within the cacophony of a natural background.

The problem is that recordings of the dove singing are as rare as the bird itself. "I came across [the bird's song] watching a 1985 interview with Carlos Keller, a former bird breeder in São Paulo state, who had a few individuals," says ecologist Carlos Araújo of the Universidad Nacional de Misiones in Garupá, Argentina. "They sang while he spoke." With Keller's help, Araújo and colleagues isolated the bird's song from the decades-old recording.

The next challenge was to see if it was even possible to identify individual bird songs amid the sounds of the forest. The team focused on three critically endangered species: the cherry-throated tanager (*Nemosia rourei*), the Alagoas antwren (*Myrmotherula snowi*) and the blue-eyed ground dove (*Columbina cyanopis*). These birds live in the Atlantic Forest and Brazil's Cerrado, in the same types of environments as the purple-winged ground dove.

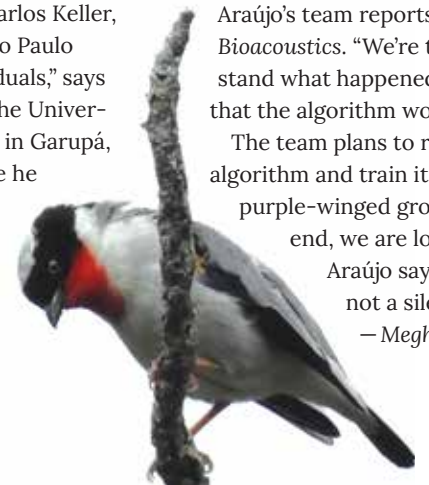
From July 2021 to April 2022, more than 100 recorders in bamboo-rich areas in the park captured a minute of ambient sound every five to 10 minutes. But readily available software wouldn't work for analyzing the recordings. "They need a lot of data input. With such rare species, we just don't have that much data to train the identification algorithm," Araújo says.

So the scientists started from scratch, working with the little data they had for the endangered birds. Araújo digitally cleaned up the few recordings of the birds' songs to create a signal template for each species. The algorithm compared those templates with the soundscape recordings, separating signal from noise. If it spotted a sound similar to a species's template, chances are that the sound came from that species.

The tool worked reasonably well to identify singing cherry-throated tanagers and blue-eyed ground doves, but not so much for Alagoas antwrens, Araújo's team reports October 23 in *Bioacoustics*. "We're trying to understand what happened, but we know that the algorithm works," he says.

The team plans to refine the algorithm and train it to look for the purple-winged ground dove. "In the end, we are looking for a ghost," Araújo says. But hopefully not a silent one.

— *Meghie Rodrigues*



An algorithm successfully picked out the song of the cherry-throated tanager in recordings of forest sounds.

FROM TOP: C.J. TEMMINCK/*LES PIGEONS* 1811; BEN PHALAN/PARQUE DAS AVES

Serotine bats (one shown) appear to use a mating strategy common in birds but never before seen in mammals.

FIRST

These bats mate like birds — without penetration

As the only flying mammals, bats are oddballs of the mammalian world. Serotine bats (*Eptesicus serotinus*) stand out for another reason. When erect, a male's penis swells to about 16 millimeters, a quarter of its body length and much too big to fit in a female's vagina. Footage of serotine bats in Ukraine and the Netherlands shows males using their penises to move aside a membrane covering females' vaginas, then pressing their penises against vulvas for about an hour or longer, biologist Nicolas Fasel of the University of Lausanne in Switzerland and colleagues report in the Nov. 20 *Current Biology*. Fur around vulvas looked wet afterward, perhaps due to semen. The findings suggest the bats mate without penetration, a common practice in birds but reported in mammals for the very first time. — *Darren Incorvaia*

INTRODUCING

A high-energy cosmic ray hails from the void

The Oh-My-God particle has a new companion. In 1991, physicists spotted a particle from space that crashed into Earth with so much energy that it warranted an "OMG!" At 320 quintillion electron volts, or exaelectron volts, it had the kinetic energy of a baseball zipping along at about 100 kilometers per hour. Now, a new particle of comparable energy has been found, researchers report in the Nov. 24 *Science*.

240
exaelectron volts
The energy of a newfound cosmic ray

Detected in 2021 by the Telescope Array experiment near Delta, Utah, the particle, known as a cosmic ray, had an energy of about 240 exaelectron volts. Cosmic rays consist of protons and atomic nuclei that zip through space at a wide range of energies. Rays with energies over 200 exaelectron volts are exceedingly rare — only a few have previously been detected. "Every time you have one of these very high-energy events, just because they are so rare, it's a big deal," says Vasiliki Pavlidou, an astrophysicist at the University of Crete in Heraklion, Greece, who was not involved in the research.

Cosmic rays hitting Earth collide with atoms in the atmosphere, creating cascades of other particles that can be detected on Earth's surface. Based on the times the cascade hit the Telescope Array's detectors, scientists determined the direction of the cosmic ray and used that information to trace it back to its origin. Oddly, the cosmic ray hails from a void, a region in space with very few galaxies. High-energy cosmic rays are thought to be churned out in violent environments, such as star-forming galaxies or jets from supermassive black holes. So one coming from somewhere with few violent processes going on makes it very interesting, Pavlidou says. — *Emily Conover*

MYSTERY SOLVED

Iron Age warrior grave belonged to a woman

A woman with a potentially violent streak has emerged from 2,000-year-old skeletal rubble found on Bryher Island off southwestern England's coast.

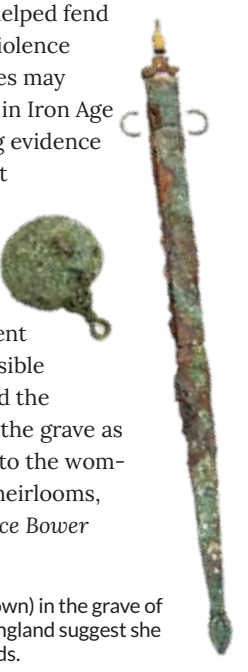
Tooth and bone fragments in a Late Iron Age grave belonged to a young woman who was interred with a sword, shield and bronze mirror, researchers report in the December *Journal of Archaeological Science: Reports*. The team used a sex-linked protein extracted from tooth enamel to classify the remains as female.

Radiocarbon analyses of the remains and artifacts date the grave to roughly 100 to 50 B.C. Tooth wear suggests the woman died in her 20s.

Since the burial's discovery in 1999, researchers have wondered whether the grave contained a man or woman. No other western European Iron Age grave includes both a sword, typically found in male burials from that region, and a mirror, often associated with female burials.

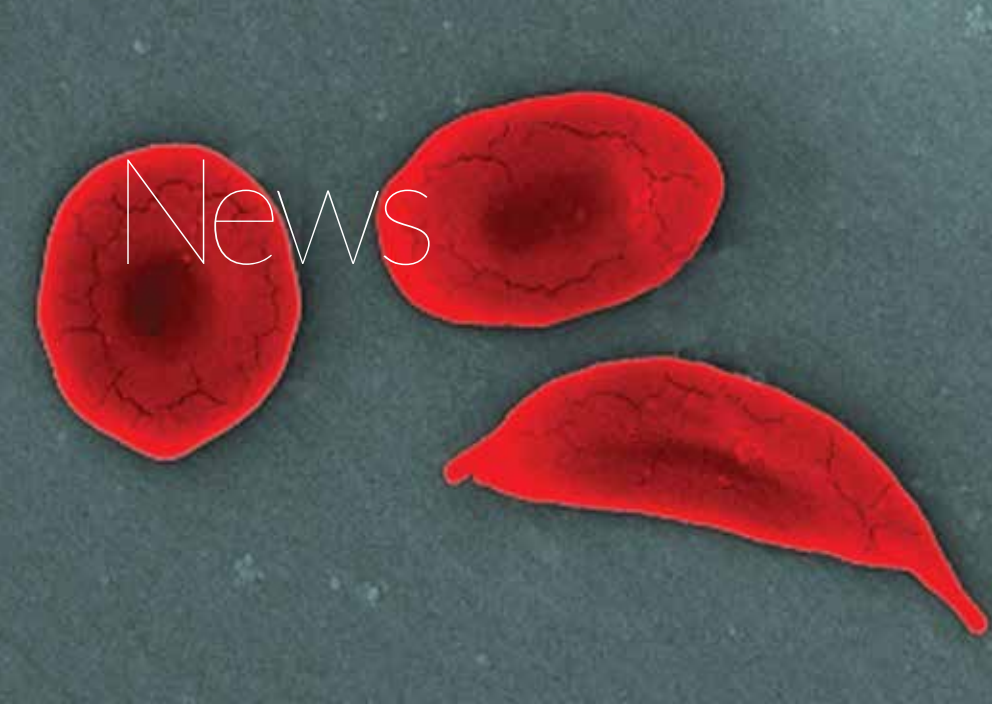
A team led by human skeletal biologist Simon Mays of Historic England in Portsmouth, a public organization that protects and studies historical places, speculates that the woman may have fought in raids and helped fend off enemy attacks. Violence between communities may have occurred often in Iron Age Europe. And growing evidence suggests that ancient women, not just men, were warriors (SN: 10/14/17, p. 6).

But the remains bear no signs of violent conflict. It's also possible that mourners placed the sword and mirror in the grave as tokens of allegiance to the woman's kin group or as heirlooms, the team says. — *Bruce Bower*



A mirror and sword (shown) in the grave of an Iron Age woman in England suggest she might have fought in raids.

FROM TOP: ALONA SHULENKO; © HISTORIC ENGLAND ARCHIVE



People with sickle cell disease have stiff, curved blood cells (right cell in this microscope image). Sickled cells get stuck in blood vessels and block blood flow to tissues, causing pain.

HEALTH & MEDICINE

Sickle cell can be treated with CRISPR

U.S. Food and Drug Administration approves gene-editing therapy

BY ERIN GARCIA DE JESÚS

People in the United States with sickle cell disease now have a novel treatment option: the first-ever CRISPR therapy.

On December 8, the U.S. Food and Drug Administration approved the gene-editing therapy for patients age 12 and older. The treatment, called Casgevy, is the world's first to genetically tweak cells using the molecular scissors CRISPR/Cas9 (*SN: 11/7/20, p. 13*).

Getting a green light for the first CRISPR-based medicine is exciting, says David Altshuler, chief scientific officer at Boston-based Vertex Pharmaceuticals, which developed Casgevy with CRISPR Therapeutics, a company that is also based in Boston. But the fact that the drug fills an unmet need for underserved patients is "more compelling to me, personally, than the fact that it's CRISPR."

Approximately 100,000 people in the United States, most of them Black or Latino, have sickle cell disease. It's caused by a genetic defect in hemoglobin, the oxygen-carrying protein in red blood cells. Unlike typical blood cells that are bendy enough to slip through blood vessels, sickled blood cells are inflexible and get stuck, restricting blood flow, which can cause debilitating pain. People with severe forms of the disease can be hospitalized multiple times a year.

The new treatment option can give patients a "new lease on life," says pediatric hematologist Kerry Morrone of Albert Einstein College of Medicine in New York City. People with the disease often miss school, work or special events due to pain.

At an FDA advisory committee meeting on October 31, several clinical trial participants given Casgevy recounted how it changed their lives. Victoria Gray, the first patient to enroll in the trial, had bouts of pain that felt like being struck by lightning and getting hit by a train at the same time. Now pain-free, she is able to enjoy time with her family, Gray said.

Jimi Olaghere, another participant, told a similar tale. Before the treatment, "sickle cell disease dominated every facet of my life," he said. "Hospital admissions were so regular that they even had a bed reserved for me." Now, Olaghere is free of pain. "Gene therapy has given me the ability to take full control of my life," he said.

The few existing therapies for sickle cell disease include taking hydroxyurea or undergoing bone marrow transplants. But hydroxyurea doesn't work for everyone, and transplants require a genetically matched donor, usually a sibling. Less than 20 percent of people with sickle cell disease have a matched sibling.

Casgevy works like a transplant, but instead uses a patient's own cells. CRISPR/Cas9 alters the genetic blueprint of bone marrow cells that give rise to blood cells. The edited cells make fetal hemoglobin, a type made by fetuses and young babies that doesn't make red blood cells sickle (*SN: 8/31/19, p. 6*). Patients receive chemotherapy to wipe out existing bone marrow cells so the new ones, which are edited in a lab and given to the patient through an IV, have a chance to thrive in the body.

In a clinical trial, 29 out of 30 patients given Casgevy and followed for 16 months didn't have pain crises for at least a year, Vertex vice president of clinical development William Hobbs said at the meeting.

The one-time treatment isn't without risks. Chemotherapy compromises the immune system, increasing the chances of dying from infections. And it can cause infertility and raise the risk of blood cancer. For some patients, the prospect of a year without immense pain may outweigh those risks, Morrone says. Others may want to wait and see what the outcomes are.

As for the potential for unintended edits, or "off-target effects," Vertex found no evidence of unwanted changes in treated patients—although researchers have identified a rare variation in the DNA of some people that could be an accidental target. It's unknown whether changing that unintended target, which is not part of the genetic code for hemoglobin, would have consequences for patients.

The advisory committee agreed that the treatment's benefits are clear and the off-target risk is small, but that additional research would be helpful. The FDA will decide by March 30 whether Casgevy can be used to treat beta-thalassemia, a blood disorder in which the body doesn't make enough hemoglobin. ■

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NEUROSCIENCE

Electricity may help treat brain injuries

Deep brain stimulation boosts cognitive skills, a small study finds

BY DARREN INCORVAIA

For people with traumatic brain injuries, cognitive functions like memory, attention and mood regulation can become exceedingly difficult. But electrically stimulating the thalamus, a brain structure that is an early stop for information coming in through the senses, could treat cognitive impairment caused by such injuries, a small study suggests.

That's good news. "There is no therapy for this kind of problem, even though it's so prevalent," says neurologist Nicholas Schiff of Weill Cornell Medical College in New York City.

Five people with moderate to severe brain injury scored higher on a test of attention and information processing a few months after having electrodes surgically implanted into the thalamus, Schiff and colleagues report December 4 in *Nature Medicine*. The study participants also reported improvements in their symptoms and daily lives.

In deep brain stimulation, implanted electrodes powered by a pacemaker stimulate targeted brain regions. The technique has long been used to treat symptoms of Parkinson's disease and epilepsy. More recently, scientists are studying its ability to treat obsessive-compulsive disorder, eating disorders and severe depression (SN: 9/23/23, p. 16).

More than 5 million people in the United States alone live with the effects of moderate to severe traumatic brain injuries, which are often caused by common events like falls and car crashes. To see if deep brain stimulation could restore cognitive function for such individuals, Schiff's team recruited six people to receive the implants. The time since the participants were injured ranged from two to 18 years.

The team targeted the thalamus' central lateral nucleus, which relays information to brain regions that handle executive function. After a severe brain injury, "you have the situation that lots of cells have been disconnected, and many cells have died," Schiff says. Electrically stimulating this relay center might restore some of those lost connections.

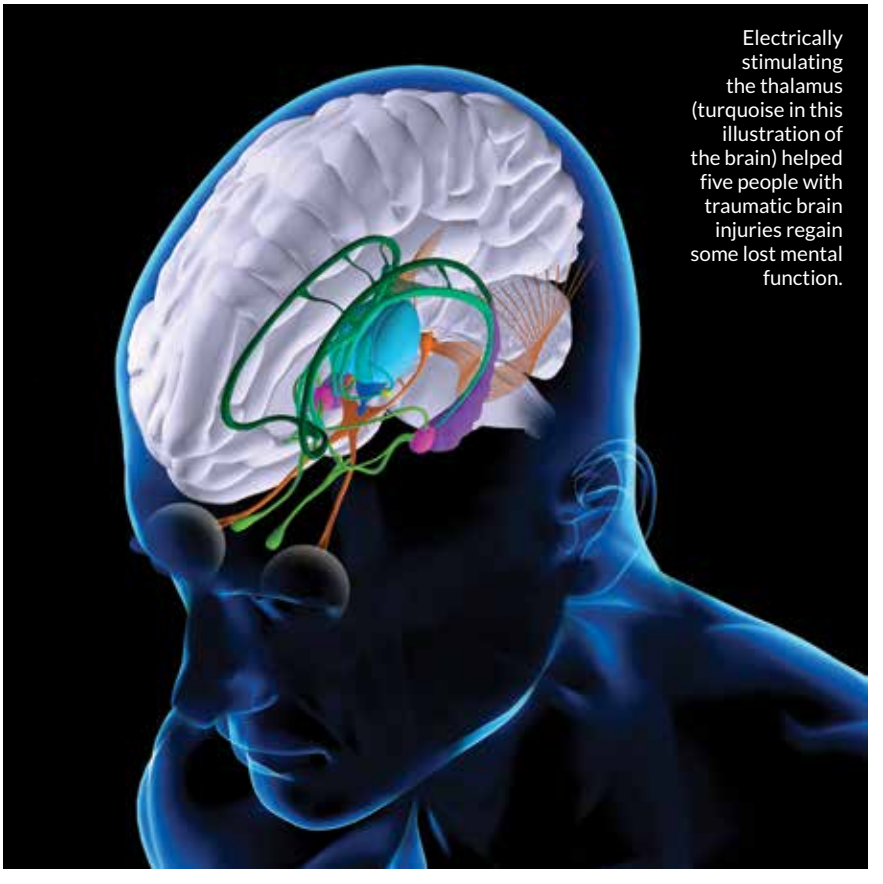
After identifying target areas in each person's brain and implanting electrodes, the researchers programmed the devices for a 12-hour on/off cycle and optimized them for each patient over a two-week period. One participant developed a scalp infection and had the device removed. The remaining five were put to the test after about three months of stimulation.

Participants were given a sheet of paper with 25 circles, each containing a number from 1 to 13 or a letter from A to L. The task, called the Trail Making Test, is to draw a line connecting the dots in ascending order while alternating between numbers and letters: 1-A-2-B-3-C and so on. Compared with before treatment, the average time it took for participants to connect the circles decreased by about a third. The person who improved the most took 171 seconds to complete the test before treatment and 83 seconds after treatment.

After receiving treatment, participants found that they were better able to do everyday things — such as reading, playing video games and watching TV — that their injuries had made difficult or impossible, Schiff and colleagues report October 18 in *Cambridge Quarterly of Healthcare Ethics*. One person said the treatment made him "more as I was before the accident." Families also noticed improvement. The mother of another participant told the researchers: "I got my daughter back. It's a miracle."

Work like this can help patients while also addressing "really fundamental questions about the basic science of human brain function," says Winston Chiong, a neurologist and ethicist at the University of California, San Francisco.

Schiff now plans to conduct larger studies of deep brain stimulation "to try to turn it into therapy," he says. ■



Electrically stimulating the thalamus (turquoise in this illustration of the brain) helped five people with traumatic brain injuries regain some lost mental function.

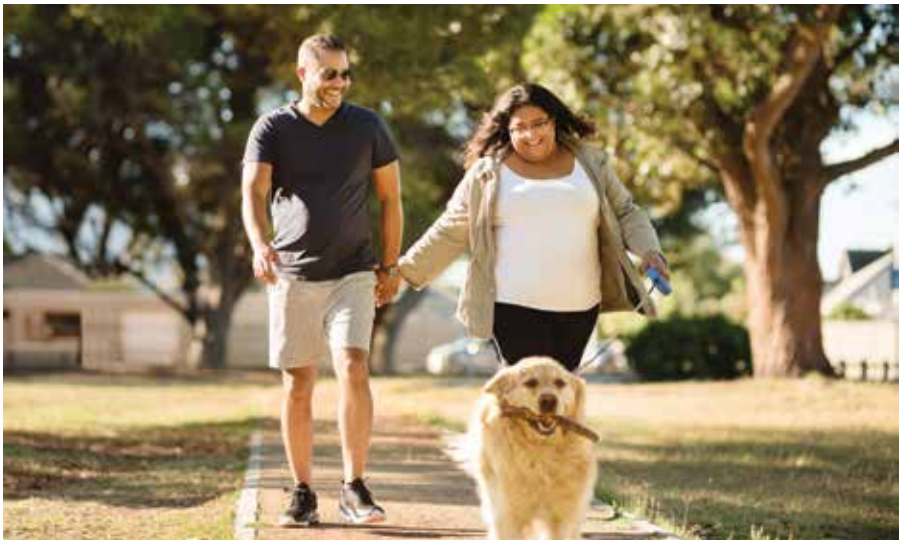
FERNANDO DA CUNHA/SCIENCE SOURCE

HEALTH & MEDICINE

Any movement is better than none

Daily physical activities, both big and small, can boost health

BY MEGHAN ROSEN



Daily physical activity can prolong life and improve health. But you don't have to run marathons or go to the gym. Walking, raking leaves, biking to the grocery store — it all counts.

We've stepped into a new year, which for many people means new resolutions. And this story was supposed to tackle a big one: the best exercise people can do to be healthy.

There's just one small problem. "There's simply no such thing as 'best exercise,'" says Emmanuel Stamatakis, a physical activity epidemiologist at the University of Sydney. If you see a headline like that, he says, it's probably clickbait.

What scientists do know — from piles of studies spanning 70 years — is that regular physical activity pays off in long-term health benefits. And recent work is starting to paint a clearer picture of all the activities that can help. You don't have to body build like Arnold Schwarzenegger or crush marathons like Tigst Assefa. And not everyone has the time, money or ability to join a gym or use special equipment. But you don't need to, Stamatakis says. Biking to the grocery store, raking leaves, playing soccer with your kids — it all counts.

Scientists still have much to figure out, like how physical activity's benefits cascade through the body and how to empower people to add movement to their daily lives. But at least one conclusion seems clear, says I-Min Lee, an epidemiologist at Harvard Medical School and Brigham and Women's Hospital in Boston. "Any physical activity is better than none."

For Lee, it comes down to finding something you enjoy. That can make the difference between getting off the couch or staying put. "The best physical activity," she says, "is something that you will do and stick with."

A slew of health benefits

The idea that physical activity is good for you might seem eye-rollingly obvious and peskily pervasive. Fitness influencers post workouts on social media, news reports tout exercise's benefits and governments worldwide try to get citizens moving.

Still, the United States' current physical activity guidelines, published in 2018, reported that some 80 percent of adults aren't doing enough. Adults need at least 150 minutes of moderate physical activity or 75 minutes of vigorous activity per week, plus muscle-strengthening activities at least two days per week, the guidelines suggest. (How long should these muscle-strengthening sessions last? "We don't really know," Lee says.)

But scientists are learning that even a little physical activity can be helpful, Lee says. Most of the studies underpinning the 2018 recommendations relied on self-reported data, Lee and colleagues wrote in a JAMA opinion piece in October. People tend to remember exercises like running or swimming laps, Lee says. But the myriad movements we take in a typical day — movements that scientists now know can improve health — were difficult to document via self-reports.

Today, wearable technology has yanked those missing movements out of obscurity and into the spotlight. Outfitting participants with tracking devices lets scientists collect mountains of in-depth data throughout a person's day, such as step count, acceleration and heart rate. That's helping reveal all the things that physical activity (or the lack of it) can do for people's health.

In the last year, scientists have shown people who did more physical activity were less likely to be hospitalized for common conditions like gallbladder disease, diabetes and urinary tract infections. These data add to recent survey-based studies and clinical trials linking exercise with lower risk of death due to flu and pneumonia, improvements in memory and attention, and better outcomes after a COVID-19 infection.

"Study after study has demonstrated the benefit of physical activity," says Bryant Webber, a preventive medicine physician at the U.S. Air Force Academy in Colorado Springs, Colo. His team has shown that it's never too late to start. In a study of more than 100,000 people age 65 and older, both aerobic training and muscle strengthening seemed to lower the risk of dying over the next eight years. Even people older than 85 saw benefits, says Webber, who did the work while at the U.S. Centers for Disease Control and Prevention. "We were impressed."

Some people may be getting the benefit of physical activity without realizing it. Stamatakis' team in Australia studies incidental activity: the typical, routine movements people perform in their daily lives. His team analyzed data from

NICK YLLOYD/MOMENT/GETTY IMAGES

people who don't exercise in their free time but had worn tracking devices for a week. Just a minute or two of intense activity—like taking the stairs or dashing to catch a train—a few times per day reduced the risk of dying in the following seven or so years by about 40 percent, Stamatakis and colleagues reported in 2022. (The more activity, the better, he says.) And last year, his team linked just three and a half minutes of daily vigorous activity with a roughly 18 percent reduction in cancer risk.

Walk uphill, carry a heavy grocery basket, "get a little bit out of breath," Stamatakis says. Any burst of exertion that briefly boosts your heart rate a few times per day could have long-term health benefits, he says. That's something many people don't understand. In interviews with middle-aged people, Stamatakis has heard a common misconception. "The majority of them still think that you need to go to the gym, otherwise there's no point."

Don't get him wrong, Stamatakis isn't saying people should skip the gym. "I want to make it absolutely clear that exercise is a fantastic option," he says. But for people who can't afford a gym membership or aren't able or willing to exercise in traditional ways, short bursts of vigorous activities several times per day could be good too.

Even a little bit helps

At Harvard, Lee has worked to dispel another popular misconception: that people need to take 10,000 steps every day to stay healthy.

She started looking into the idea in 2018, when her hospital launched a campaign to get employees to walk more. Because Lee studies physical activity, her administrators asked her to put employees into teams. It was mostly middle-aged and older folks, she says. "For some of these people, if you're asking them to do 10,000 steps—I mean, no way," she says. "Even doing 5,000 steps is a stretch."

In 2019, Lee and colleagues found that older women who took around 8,500 steps per day were 66 percent less likely to die during the study than those who took roughly 2,500 steps per day. In a 2022 analysis, the researchers reported something similar in men and women of different ages. Bottom line: For mortality benefits, people age 60 or older should shoot for 7,000 steps a day, and people younger than 60 should shoot for 9,000, Lee says.

But those targets shouldn't be discouraging, she says. Lee takes a tiered approach. "If you do nothing, just do a little bit," she says. "If you already do a little bit, do a little bit more."

She follows that advice herself. Lee grew up in the tropics in Malaysia, where it was hot, and she didn't see many people exercising, including herself. "I did diddly-squat," she laughs. That changed after she came to the United States for her doctorate. "It got to be so embarrassing to do physical activity research without being physically active," she says. So she started running. "I run slowly, and I don't run a lot, but I do it," she says.

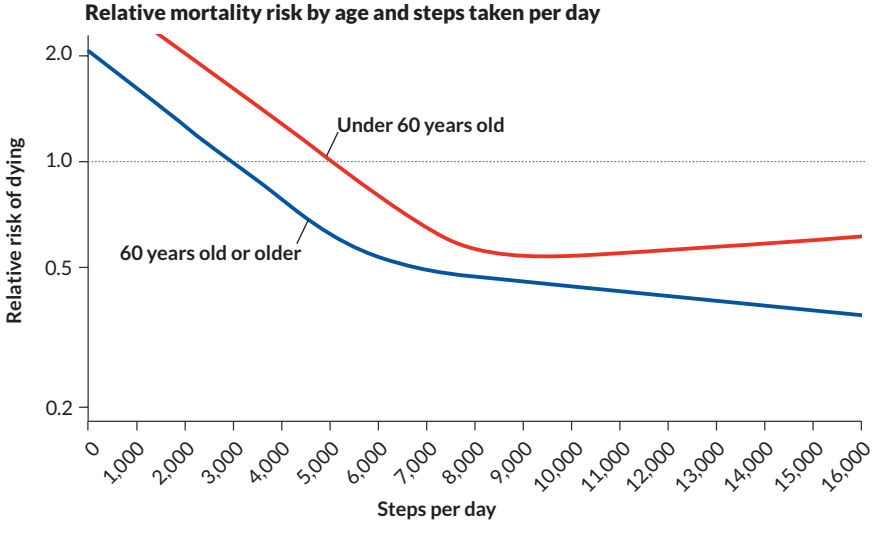
The message that people can reap health benefits from even a little bit of physical activity is seeping into the mainstream, says Lyndon Joseph, an exercise physiologist at the National Institute on Aging in Bethesda, Md. "You don't have to be able to sprint like Usain Bolt," he says. "You just need to be active."

Scientists know physical activity works, Joseph says, though they don't fully understand how. Why does walking, for example, which relies on leg muscles, also help the heart, lungs, kidneys and immune system? "The whole body responds to exercise," Joseph says. Scientists are trying to figure out the molecules at play, and how those released from one tissue can improve the health of another. "That is the big question."

How to get people to move—and do it consistently—is another big question. Changing people's behavior is no easy feat, Stamatakis says. And massive structural barriers often stand in the way. Even if scientists get people on board with upping their step counts, if their neighborhoods don't have sidewalks, hitting daily step goals becomes a lot more difficult.

More sidewalks, trails and bike paths would make it easier for people to rack up incidental physical activity. "The role of the environment is critical," Stamatakis says. Lee agrees. Physical activity is not simply a personal choice, she says. Government policies can influence how much or little we're able to do. Again, she emphasizes, it doesn't take much to get substantial health benefits. And people can start at any level.

In fact, Lee says, "the biggest bang for your buck actually comes among people who go from doing nothing to doing just a little bit." ■



Stepping up As people take more steps per day, the relative risk of dying over the next seven years drops sharply (below the horizontal line represents lower risk). This decrease slows around 7,000 steps for people age 60 or older (blue) and around 9,000 steps for those under 60 (red).

A. PALUCH *ET AL*/*THE LANCET PUBLIC HEALTH* 2022

ANIMALS

Penguin parents get over 10,000 winks

Sleeping in ultrashort bursts helps the birds protect their young

BY JAKE BUEHLER

Nesting chinstrap penguins take nodding off to the extreme. The birds briefly dip into a slumber many thousands of times per day, sleeping for only seconds at a time.

The penguins' breeding colonies are noisy, and threats from predators and aggressive neighbor penguins are unrelenting. The extremely disjointed sleep schedule may help the penguins protect their young, researchers report in the Dec. 1 *Science*.

The findings add to evidence "that avian sleep can be very different from the sleep of land mammals," says Jerome Siegel, a neuroscientist at UCLA who was not involved in the study.

Nearly a decade ago, behavioral ecologist Won Young Lee of the Korea Polar Research Institute in Incheon noticed something peculiar about the sleep of chinstrap penguins (*Pygoscelis antarcticus*) on King George Island in Antarctica. The birds would seemingly doze off for very short periods of time. Then Lee learned about frigate birds' ability to steal sleep while airborne on days-long flights.

In 2019, Lee and colleagues returned to the island to study the daily sleep patterns of 14 nesting chinstrap penguins. Surgically implanted electrodes measured brain activity while other instruments on the back recorded the birds' movements.

Nesting chinstrap penguins grab seconds of sleep at a time, perhaps so they can stay alert enough to defend chicks and eggs from predators and aggressive neighbors.

Nesting penguins took more than 600 microsleeps per hour, each nap averaging four seconds, the team found. At times, only half the brain slept while the other half stayed awake. All together, the oodles of snoozes provided over 11 total hours of sleep for each brain hemisphere across more than 10,000 winks each day.

Some other types of birds and marine mammals have strange or restricted sleep patterns too, often when staying alert is important. Dolphins sleep with half their brain at a time, and ducks can do the same to stay vigilant against predators (SN: 2/6/99, *p.* 86). And elephant seals dramatically reduce their sleeping hours while out at sea (SN: 12/16/23 & 12/30/23, *p.* 36). But the sheer number of microsleeps seen in chinstrap penguins is unprecedented, Lee says.

"The penguins do not have any time where they decrease their vigilance," says study coauthor Paul-Antoine Libourel, a sleep ecophysiologist at the Lyon Neuroscience Research Center in France.

The sleep pattern may help balance the brain's need for rest with the demands of nesting. Predatory birds like brown skuas (*Stercorarius antarcticus*) patrol penguin colonies looking to plunder undefended eggs and chicks. There's also constant commotion — and sometimes threats — from other penguins in the colony, which disrupts sleep. Still, the many micronaps appear to be restorative: The penguins functioned well enough to survive and successfully raise chicks. It's unclear if the sleep pattern changes after the breeding season ends.

"Sleep seems to be very diverse and flexible among species," Lee says. "By studying their sleep behavior, we can understand how animals have evolved to achieve brain restoration." ■

ANIMALS

Aussie mosquitoes dine on frog noses

The insects risk death for an easily accessible blood feast

BY JAKE BUEHLER

An Australian mosquito species knows the best spot to drink its bloody meals: a frog's nostril.

The bloodsuckers are surprisingly selective when dining on frogs, seemingly picking no other place on the body to feast, researchers report November 21 in *Ethology*. Frogs' sniffers may be an easy and productive place for the mosquitoes to pierce skin and drink up. The new finding could help scientists better understand the transmission of some frog diseases.

Behavioral biologist John Gould discovered the nostril-nibbling behavior while studying frogs in ponds on Kooragang Island in New South Wales. From 2020 through 2022, Gould occasionally noticed mosquitoes on the faces of the frogs he was surveying and would take photos.

"It was only once I laid out all the photos together that I realized something very particular and surprising was happening," says Gould, of the University of Newcastle in Callaghan, Australia. In the dozen photos that Gould took, every single bloodsucker was feeding on the skin of the frog's nostril.

Some mosquitoes feed only on frogs and toads, biting various parts of the body. The mosquito that caught Gould's eye, *Mimomyia elegans*, has a generalized diet of amphibians, mammals and birds. "Yet its feeding strategy when using frogs appears to be highly specialized," Gould says.

The nostril skin may be especially soft and thin, making it easy for the mosquito's biting mouthparts to pierce, Gould says. Alternatively, there could be a high density of blood vessels near the surface of the nostril skin, he suggests.

Dining at this venue on a renowned insect eater could be risky, considering the nostrils rest just above a powerful,

W.Y. LEE

sticky tongue (SN: 11/5/22, p. 10). But the mosquitoes might have a stealthy defense. Gould observed some of the mosquitoes landing on the frogs' backs and tiptoeing toward the head. This, he says, might keep the insects from being detected and eaten.

Gould and colleagues have shown in previous work that mosquitoes may be vectors for amphibian chytrid fungus, a grave threat to amphibians globally. "Determining where exactly mosquitoes land and subsequently feed on frogs may allow scientists to better understand the spread of the infection across the skin surfaces of frogs," he says.

Some of the photographed mosquitoes were feeding on green and golden bell frogs (*Litoria aurea*). Those frogs are considered vulnerable to extinction, in part due to chytrid fungus and habitat loss, by the International Union for Conservation of Nature.

In this series of photos, a mosquito walks along the back and toward the head of an eastern dwarf tree frog on Australia's Kooragang Island. The strategy might allow the insect to sneak up to its preferred feeding spot at the bright green frog's nostril without getting eaten.

A laboratory experiment to carefully watch frogs and mosquitoes to confirm the insects' nasal predilections would be helpful, says Manuela Carnaghi, an insect behavioral ecologist at the University of Greenwich in Chatham, England.

Determining precisely how mosquitoes target and bite multiple kinds of hosts is key for understanding disease transmission, Carnaghi says. It's particularly important when considering the ability of a mosquito-borne pathogen or parasite to jump between different species. ■

ANIMALS

Dolphins can sense faint electric fields

The ability has now been observed in two cetacean species

BY SAIMA S. IQBAL

To snap up fish, bottlenosed dolphins may rely on more than just sharp sight and echolocation. The marine mammals might also pick up on the weak electric pulses prey produce each time the heart beats or air filters through gills.

In an experiment, two bottlenosed dolphins in a zoo reliably sensed faint electric fields on the scale of microvolts per centimeter, says Tim Hüttner, a sensory biologist formerly of the University of Rostock in Germany. That puts the duo's skills on par with the Guiana dolphin (*Sotalia guianensis*) and some egg-laying mammals like platypuses.

The ability, called electroreception, has also been documented in fish, amphibians and sharks (SN: 7/9/16, p. 4). It was only in 2011 that the Guiana dolphin made the list, as researchers discovered telltale sensory receptors hidden in an organ on the snout (SN: 8/27/11, p. 12).

In 2021, Hüttner and colleagues reported the same structure in bottlenosed dolphins and confirmed that the creatures could detect electric fields on the scale of 0.5 millivolts per centimeter, similar to the electric fields that some large fish and crustaceans emit. The new finding suggests that common bottlenosed dolphins (*Tursiops truncatus*) can probably make out the much subtler signals emanating off the majority of fish, the team reports November 30 in the *Journal of Experimental Biology*.

Researchers trained the dolphins, named Dolly and Donna, to position their snouts in a metal apparatus and to swim away if they could sense an electric impulse delivered to their sensory organs. The dolphins proved sensitive to both direct current and alternating current, two forms of electricity that living things generate. The dolphins especially excelled at detecting direct current, which produces a steady signal. Dolly picked up on fields as low as 5.5 microvolts per centimeter and Donna on those of 2.4 microvolts per centimeter.

The research supports an intriguing theory, says Paul Nachtigall, a marine biologist at the University of Hawaii at Manoa. Perhaps pits on the snout that once held whiskers, long regarded as vestigial structures, evolved to fit another purpose: electroreception.

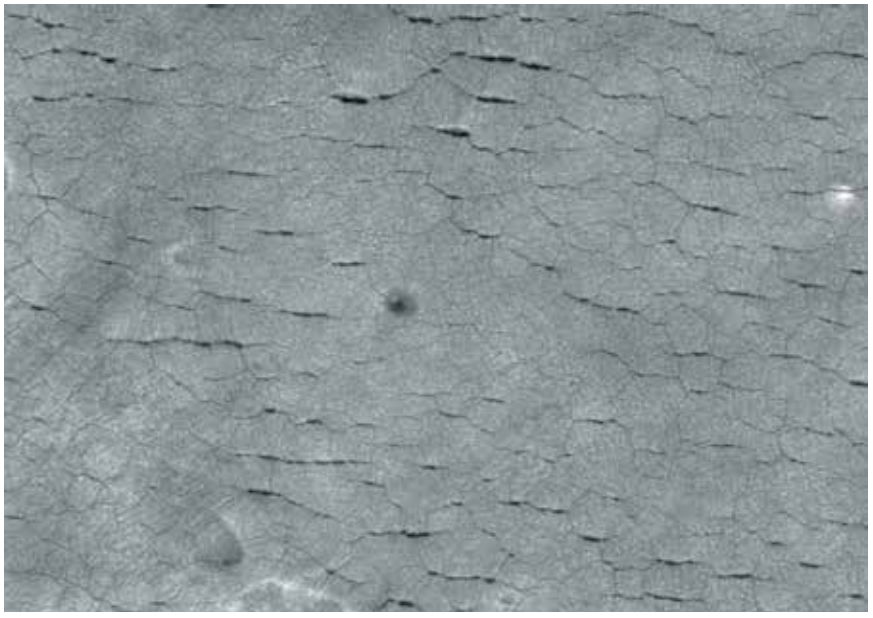
The ability may come in handy in situations where sight and echolocation are impaired. For instance, Guiana dolphins primarily hunt for food on the seafloor, where the sediment can muck up their senses.

Bottlenosed dolphins don't hunt the same way but do often reside in murky waters and occasionally stick their heads into sand to look for fish. Echolocation stops working close up, but electroreception could allow dolphins to detect prey a few centimeters away. It may be the last push they need to nail a target, Hüttner says. To test this idea, the team wants to study electroreception while dolphins are moving.

Other species of dolphins have pits on their snouts as well, which means electroreception may be more widespread, he notes. The ability might not be useful in some dolphins' hunting strategies. So Hüttner speculates that it could serve an additional function: helping dolphins to orient themselves along Earth's magnetic field lines as they navigate ocean waters.

"There's just so much to find out," Nachtigall says. "This study is just the first page of a book." ■

J. GOULD



Cycles of freezing and thawing could have formed the polygonal pattern shown in this satellite image of a 3-kilometer-wide patch of Mars' surface. Similar patterns recently found buried closer to the planet's equator hint that the region had an icier climate in the past.

PLANETARY SCIENCE

Buried polygons hint at Mars' tipsy past

Ancient terrain suggests the equator once had a polarlike climate

BY ELISE CUTTS

Enormous polygon patterns in rock lie dozens of meters below Mars' surface, ground-penetrating radar data suggest.

Similar patterns develop on the surface in Earth's polar regions when icy sediments cool and contract. A comparable process long ago may have created the shapes near the Red Planet's now-dry equator, scientists report November 23 in *Nature Astronomy*. If so, the finding hints that Mars' equator was icier when the polygons formed a few billion years ago.

"Buried possible polygons at that depth have yet to be reported" on Mars, says Richard Soare, a planetary scientist at Dawson College in Montreal who was not involved in the study. Searching for ancient polygonal terrain on Mars using ground-penetrating radar is a new idea that "could be powerful," Soare says, and could help scientists understand how Mars' climate changed in the past.

On Earth, polygonal terrain forms when sharp temperature drops cause icy ground to contract and crack open. These thermal fractures start small. But the cracks can fill with ice, sand, or both, forming wedges that prevent the cracks from healing and gradually pry open the earth. Because this wedging process requires multiple cycles of freezing and thawing, polygonal ground is a good hint that terrain was icy when the patterns formed.

China's Zhurong rover landed near Mars' equator in a region called Utopia Planitia in 2021. The rover's ground-penetrating radar sent radio waves into the crust and recorded the echoes that bounced back, providing a look at the subsurface directly below the spacecraft, says Lei Zhang, a geoscientist at the Chinese Academy of Sciences' Institute of Geology and Geophysics in Beijing.

Zhang and colleagues analyzed the radar data by considering both the frequency of the reflected radio waves and the waves' travel time. This allowed the researchers to detect what appear to be polygon patterns, which previous analyses of the same data missed.

The rocky shapes appear to be about 70 meters across and are bordered by wedges nearly 30 meters wide and tens of meters deep — about 10 times as large as typical polygons and wedges on Earth. So it's possible the structures here formed a bit differently than our ice-wedge polygons, Soare says.

The rover's landing site in a dry, sandy desert is not where scientists would expect to find polygons because the current climate isn't conducive to the freezing and thawing cycles that form the geometric terrain on Earth, says geoscientist Ross Mitchell, also of the Institute of Geology and Geophysics. Polygons spotted at higher latitudes on the Martian surface from orbit may have formed via such cycles in the relatively recent past (*SN*: 8/28/04, *p.* 142). The newfound buried polygons must have formed much earlier, in an icier environment similar to a polar region, Mitchell, Zhang and colleagues say.

Changes in the tilt of Mars' axis could explain such a shift in climate. Simulations of Mars' orbit have suggested that the planet's spin axis has at times been so extremely tilted that the planet essentially lay halfway on its side. In turn, the poles would have received more direct sunlight while equatorial regions would have frozen.

> Finding potential polygons buried near the Martian equator is smoking-gun evidence for the idea that the tilt of the planet's axis has varied so substantially.

Finding potential polygons buried near the Martian equator is smoking-gun evidence for the idea that the tilt of the planet's axis has varied so substantially, Mitchell says.

"We think of every planet other than Earth as dead," he says. But if the Red Planet's axis does swing around often, then our neighbor's climate would be far more dynamic than currently thought. ■

JPL-CALTECH/NASA, MALIN SPACE SCIENCE SYSTEMS

ASTRONOMY

Small stars could test ideas about E.T.

Ultracool dwarfs may reveal ways for life to arise on exoplanets

BY JAMES R. RIORDON

A survey of small, cool stars is helping to narrow in on the conditions that might set the stage for life beyond the solar system.

A look at about 200 ultracool dwarf stars shows that their ultraviolet light isn't intense enough to potentially jump-start life, scientists report December 1 in the *Monthly Notices of the Royal Astronomical Society*. Although the result may seem like bad news for finding signs of life on distant planets, the stars could serve as test-beds to determine what other conditions can create the chemical foundations of life.

The stars in the study are minuscule, weighing about a tenth as much as the sun. They're also among the most common types of stars.

Because dwarfs are cool and dim, it's often easy to spot planets orbiting them. For instance, astronomers studying the dwarf star TRAPPIST-1 found that it hosts seven Earth-sized planets, three of which may be within the star's habitable zone, where conditions are amenable to life (SN: 3/18/17, p. 6). But how life gets started on a habitable planet is unclear. One possibility is that UV starlight provides the energy needed for hydrogen, oxygen, carbon, nitrogen, sulfur and other atoms to link up into life-friendly compounds.

With that in mind, scientists used TESS, the Transiting Exoplanet Survey Satellite, to measure the UV radiation emitted by 208 ultracool dwarfs within 130 light-years of Earth. The stars emit steady streams of UV light, as the sun does, and many also emit bursts of UV light. But overall, the energy is too low to forge the chemicals needed to kick-start life, the team found.

The dearth of UV light doesn't quash hopes of finding life around such stars. "UV is an energy source for prebiotic chemistry that we can measure, and that is why we focused on it," says Antígona Segura, a space scientist at the National Autonomous University of Mexico in Mexico City. "But there are many other energy sources, like cosmic and stellar particles, and particles, radiation and heat produced by radioactive decay."

Therefore, planets around ultracool dwarfs might be useful to test whether something other than UV light can get life going. Researchers could search for life on planets with the least UV activity, "where we can know with confidence that UV-driven prebiotic chemistry cannot happen," says astrophysicist Paul Rimmer of the University of Cambridge. "If we find evidence of life on these planets, this will show that there are other paths to life."

But sources of energy that can start life can also make planets less habitable for complex life-forms, Segura notes. "We cannot currently say which [effects] would prevail. The best approach now is to study case by case and wait for more observational constraints."

Finding signs of life on planets orbiting ultracool dwarfs would confirm the potential for UV-free origins of life. Finding no signs of life on planets in the habitable zones of the most common stars would reduce the odds of discovering life outside of our solar system, Rimmer says. Either way, future surveys of such stars could give scientists a better idea of the possible prevalence of life in the universe. ■

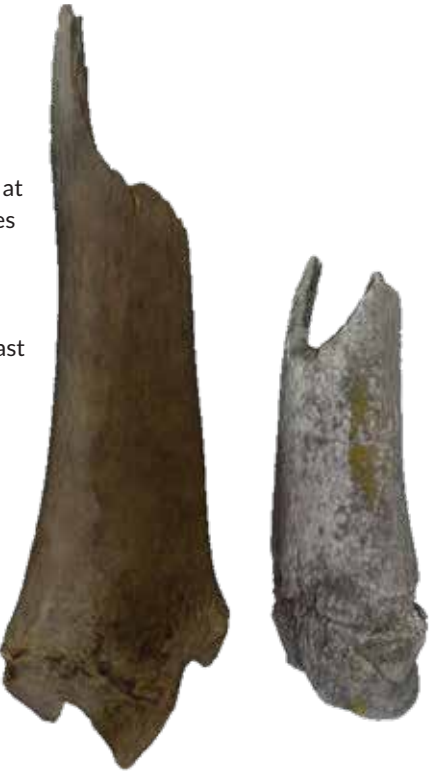
ANTHROPOLOGY

Indigenous Patagonians took in Spanish horses

Hunter-gatherers in Argentina's Patagonia region integrated horses with Spanish pedigrees into their societies around 400 years ago, long before Europeans occupied the area. Analyses of horse remains, including partial leg bones and teeth, uncovered at a site called Chorrillo Grande 1 suggest that locals raised and ate transatlantic equines by the early 1600s, researchers report December 8 in *Science Advances*.

Spaniards and their horses reached south-central South America around 1536, but those people moved north a few years later, leaving steeds behind. Patagonian hunter-gatherers incorporated growing numbers of horses into their way of life at least a century before Europeans settled in the region in the mid-1800s, archaeozoologist William Taylor of the University of Colorado Boulder and colleagues conclude.

DNA analyses of the remains identified three domestic horses, while radiocarbon dating of horse specimens, food crusts on pottery pieces and other finds places people at Chorrillo Grande 1 starting between 1599 and 1653. Fractures and burned patches on limb bones (two shown, right) suggest that two horses were butchered for food. Europeans in Patagonia during the 1800s wrote about local hunter-gatherers consuming horse meat and blood. Taylor suspects that horses quickly assumed many roles in the region's Indigenous cultures. Other historical documents describe groups riding horses, using the animals in ceremonies and making stringed instruments from horse products. — *Bruce Bower*



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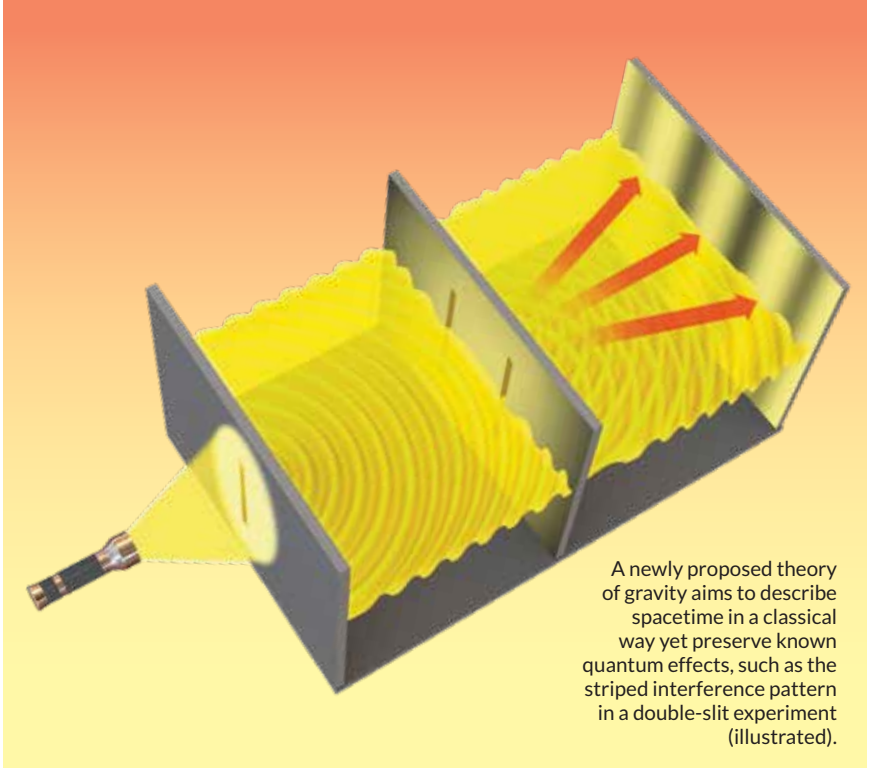
PHYSICS

What if gravity isn't quantum?

A physicist is building a case for scrapping an elusive idea

BY EMILY CONOVER



A newly proposed theory of gravity aims to describe spacetime in a classical way yet preserve known quantum effects, such as the striped interference pattern in a double-slit experiment (illustrated).

DORLING KINDERSLEY/GETTY IMAGES

A rift runs deep through the heart of physics. The general theory of relativity, which describes gravity, clashes with quantum physics. In an effort to seal that physics fissure, untold numbers of physicists have spent their careers working to build a theory of quantum gravity.

But since 2018, one physicist has championed a radically different path. Jonathan Oppenheim thinks that gravity might be fundamentally classical, meaning it isn't quantum at all. It's an unconventional idea, to say the least. "When we started, maybe 99 percent of our colleagues thought we were crackpots and that's now down to maybe 70 percent," says Oppenheim, of University College London.

All known forces except gravity are formulated in terms of quantum physics. The prevailing view is that gravity will need to assimilate with its quantum colleagues. But gravity is different, he argues. While other forces evolve within a landscape of spacetime, gravity is the warping of spacetime itself. So, Oppenheim says, "it is pretty unclear that it should have a quantum nature, in my view."

Physicists have devised several "no-go" theorems that seemingly forbid a classical theory of gravity. Such theorems highlight inconsistencies, apparently fatal to the idea, that arise when classical gravity is applied to quantum particles. But it's possible to get around those prohibitions by adding some randomness to the way that spacetime bends in response to quantum particles, Oppenheim reported December 4 in *Physical Review X*.

Consider the famous double-slit experiment of quantum physics (SN: 6/8/19, *p.* 14). Particles are sent toward a detector, through a barrier with two slits in it. When the particles arrive at the detector, they create stripes called an interference pattern. That pattern arises because, in quantum physics, each particle isn't constrained to pass through one slit or the other. Instead, each particle can exist in a superposition, taking a quantum combination of both possible routes. If a scientist makes a measurement to determine which slit each particle passed through, the pattern disappears.

If a standard classical theory of gravity were correct, it would be possible to measure the gravitational field of the particles so precisely that you could determine which slit each particle went through. This possibility would destroy the interference pattern, even without doing the measurement. Because scientists do observe interference patterns in the lab, that's a big blow for a standard classical theory.

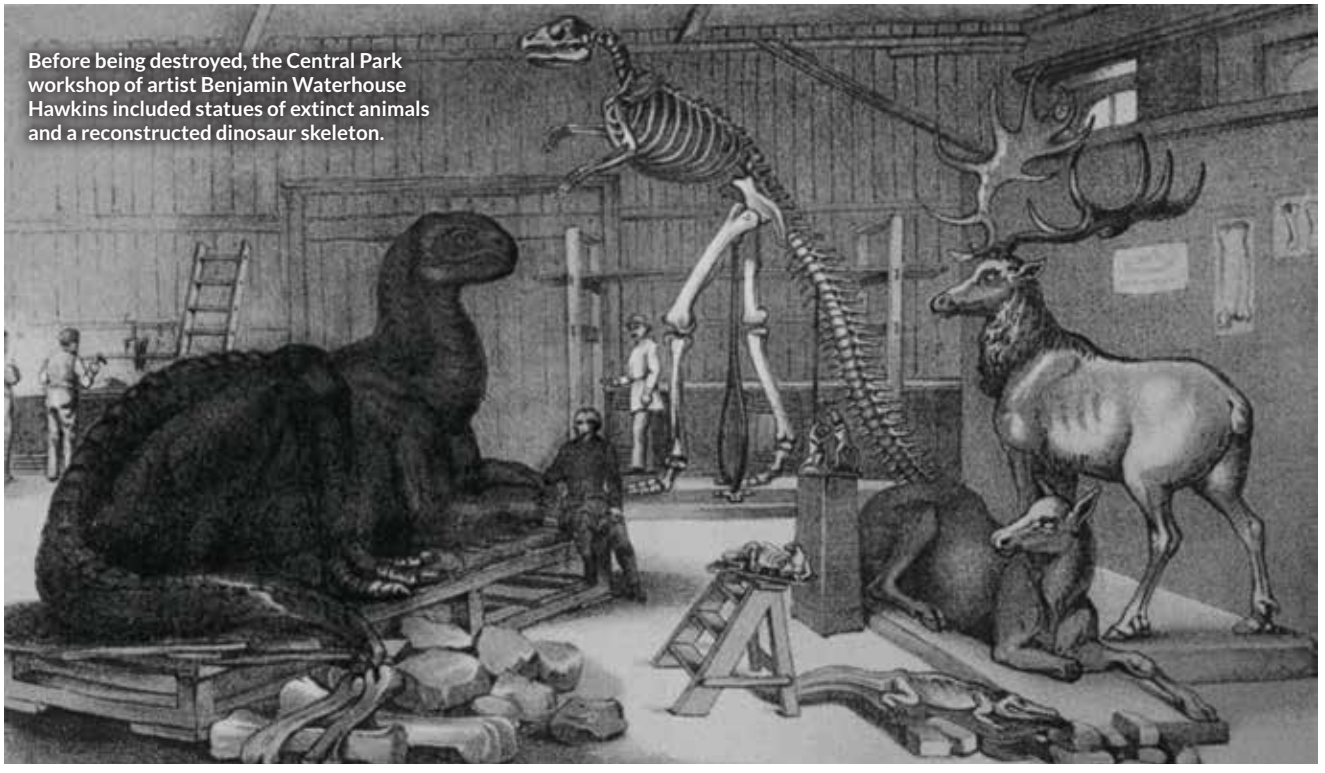
But due to the randomness baked into the new theory that Oppenheim proposes, instead of a particle having a determined gravitational field, the field fluctuates. That means, unlike for the standard version of classical gravity, it's not possible to determine which slit a particle went through by precisely measuring its gravitational field. Particles can pass through the slits in a superposition, and the interference pattern is saved, restoring the possibility gravity could be classical.

Experiments can test this theory by searching for those random gravitational fluctuations, Oppenheim and colleagues reported December 4 in *Nature Communications*. "Essentially, you very precisely measure the response of a mass to a gravitational field," says coauthor Zach Weller-Davies, who completed the study at the Perimeter Institute for Theoretical Physics in Waterloo, Canada.

Scientists have previously proposed ways to make classical gravity comport with quantum effects. But Oppenheim has been "leading a renaissance," says physicist Vivishek Sudhir of MIT. Sudhir hopes to test the theory with another type of experiment, measuring the correlations between the motions of two masses that interact gravitationally, he and a colleague reported September 16 at arXiv.org.

However, the theory has features some physicists might find unsatisfying. For example, the randomness involved means that the theory is not reversible: There's no way to start from the endpoint of an interaction and trace its steps backward.

Still, some quantum gravity proponents think that the work has merit. The idea is interesting regardless of whether gravity is found to be classical or quantum, says theoretical physicist Flaminia Giacomini of ETH Zurich. For an experiment to proclaim that gravity is quantum, scientists need to understand what experimental results classical gravity could produce. Only then "will we be able to prove in a strong way that gravity is not compatible with a classical description," she says. ■



Before being destroyed, the Central Park workshop of artist Benjamin Waterhouse Hawkins included statues of extinct animals and a reconstructed dinosaur skeleton.

SCIENCE HISTORY IMAGES/ALAMY STOCK PHOTO

PALEONTOLOGY

A new culprit ID'd in a paleo whodunit

Creationism isn't to blame for the loss of Central Park's dinosaurs

BY FREDA KREIER

A sledgehammer dealt the final blow to New York City's dream of a paleontology museum. On May 3, 1871, workers broke into the workshop of artist Benjamin Waterhouse Hawkins. Inside, they came upon a plaster skeleton of a duck-billed dinosaur—modeled after the first dinosaur fossil unearthed in New Jersey 13 years earlier—alongside a statue of the beast as it might have appeared in life.

These were the first 3-D renderings of any North American dinosaur, a testament to the continent's geologic past that scientists were only just beginning to understand. But the public would never see the skeleton or the statute.

The workers wrecked the workshop. Plans and drawings were torn to pieces. Sledgehammers shattered the dinosaurs.

In the more than 150 years since, this vandalism has remained one of the most infamous events in paleontology. The story passed down through the years is that New York political boss William Tweed ordered the workshop be destroyed in an act of political and religious vengeance.

Tweed viewed dinosaurs as "inconsistent with the doctrines of received religion," a paleontologist noted in 1940. The destruction is still cited as an early battle between a traditional Christian world view and a growing scientific understanding of Earth's deep past.

The loss has "always been a shock to the paleontological community," says Vicky Coules, an art historian at the University of Bristol in England. But the story might be due for a rewrite. Historical sleuthing by Coules and her Ph.D. adviser Michael Benton, a paleontologist at Bristol, suggests the demise of Hawkins' dinosaurs was not religiously motivated, or even ordered by Tweed.

Instead, the story that paleontologists tell about this affair may say more about the history of anti-evolution sentiment during the 20th century than in the 1800s.

Bringing prehistory to life

Dinosaurs' place today in the public imagination is in no small part due to Hawkins. His most famous artwork went on display in 1854 at London's Crystal Palace. Thousands flocked to this showcase of (sometimes looted) wonders from across the British Empire. One section featured life-size dinosaur statues made by Hawkins.

This was several years before Darwin published his theory of evolution and only about a decade after the term "dinosaur" had entered the lexicon. For many people, seeing these statues was the first time they had come face-to-face with the concept of deep time. Displaying dinosaurs in the flesh was innovative, Benton says. "No one had attempted anything like this before."

The exhibit made Hawkins the de facto expert on depicting prehistoric life. In 1868, the Board of Commissioners of Central Park—the group in charge of developing New York's new green space—asked Hawkins to build similar statues for the park's planned Paleozoic Museum, dedicated to American paleontology.

At the time, most major dinosaur discoveries were happening in Europe or its colonies. Scientists had yet to dig

into the ample bone grounds of western North America, and major finds — like *T. rex* — were at least a decade away.

But a small number of fossils were starting to come out of the East Coast. The Paleozoic Museum, the Central Park commission thought, would give Americans a chance to prove that they too had a prehistory worth remembering.

Hawkins accepted the job, dedicating the next few years to a museum that would never open its doors.

The standard story

In the 1860s, New York was a city on the rise. One man riding that high was William Tweed, a state senator who dominated the city's political scene. Tweed stripped power from all who opposed him. In May 1870, for instance, he dissolved Central Park's board of commissioners and created a new group filled with his cronies.

By year's end, the new commissioners canceled the Paleozoic Museum. Its demise had been simmering for months. Already, Hawkins' workshop had been relocated from a government building to a shed in the park. The move made room for the growing collection of the planned American Museum of Natural History, which had private financial backing from New York's wealthiest citizens.

The park commissioners decided that a museum dedicated solely to paleontology was just too big a burden to take on with public funding.

In March 1871, the *New York Times*, a frequent critic of Tweed, reported on the loss of the museum, which Hawkins had lamented publicly. Two months later, his dinosaurs lay in pieces. The destruction sent ripples through the scientific community, becoming one of the foundational stories in American paleontology. The story's villain: Tweed.

The *Times* article allegedly sent Tweed into a rage, and he ordered one of his cronies to descend "upon the Paleozoic Museum with vengeance in his soul," paleontologists later wrote. It wasn't just the bad press that supposedly angered Tweed. "There was always a rumor that there was some sort of creationist angle to it," says paleontologist Carl Mehling of the American Museum of Natural History.

This version of the story, which paleontologists have repeated since at least the 1940s, rests in part on Tweed referring to dinosaurs as "pre-Adamite." The argument fits into a common perception that emerged during the mid-20th century, that religion and prehistory were often at odds in the late 19th century.

Who's to blame?

But this story wasn't adding up for Coules when she began to read up on the saga. Why would Tweed wait two months after the *Times* article to retaliate?

He also had bigger things to worry about. He had been accused of everything from bribery to money laundering (and was eventually imprisoned). Coules thought it odd that Tweed, who was fighting for his political life, would take such offense to a news story buried on page 5 that didn't even mention him by name.

She suspected another culprit: lawyer Henry Hilton, who Tweed had appointed to the new Central Park board in 1870. He took to the role, regularly visiting the park to search for improvements.

His "improvements" were head-scratchers. Hilton had workers paint a bronze statue of the biblical Eve white, damaging the metal. He ordered a similar treatment for a whale skeleton destined for a museum.

> Natural history, including paleontology, was seen as a respectable, middle-class occupation in the 19th century.

One day while going through her notes, Coules came across park commission meeting minutes from the day before the models were destroyed. The committee had resolved to remove Hawkins' workshop "under the direction of the Treasurer" — Henry Hilton. "I was like, wow! Look at this!" Coules says.

She didn't find any hint that religion was the motivation. Rather, she argues, Hilton "had a strange relationship with artifacts," as demonstrated by his whitewashing habits. He would go on to harbor other destructive tendencies, including swindling a widow out of her fortune and running her late husband's business into the ground. Hilton had "quite strange ideas [that managed] basically to piss off everybody," says Coules, who published her findings with Benton last June in *Proceedings of the Geologists' Association*.

The fact that religion played no role makes sense to science historian Lukas Rieppel of Brown University in Providence, R.I. "I never understood that speculation."

For one thing, Benton points out, "pre-Adamite" was just a way to refer to deep time. So even if anyone had referred to Hawkins' models in this way, it would have been more descriptive than derisive. Natural history, including paleontology, was seen as a respectable, middle-class occupation in the 19th century, Rieppel says. "Natural history was seen as an expression of piety. So a way that one could express one's devotion to God was by learning about God's works in the natural world."

A more inflexible view of creationism, in which evolution is false and the world is only a few thousand years old, gained steam only in the 20th century, Benton says. Religion's supposed role in the Hawkins' saga may have been introduced by paleontologists writing about this incident in the mid-20th century. They may have been projecting their experiences with creationist movements into the past, Rieppel says. From there, the story stuck.

A lasting influence

The loss of the Paleozoic Museum might have been for the best. It would have been "obsolete almost immediately, and I fear almost comical," Mehling says, as bigger discoveries came from the American West.

But Hawkins' paleoart remains influential. While preparing for the Paleozoic Museum, he strung together the bones of the New Jersey duck-billed dinosaur into a standing skeleton and displayed it in Philadelphia. Previously, fossils had been shown flat on a table. It was so inspiring that in 1905, when the American Museum of Natural History unveiled its *Brontosaurus* specimen, it showed the skeleton upright.

Today, museums still display fossils using Hawkins' method. ■

A woman behind THE FUZE

THE UNSON FAMILY; TOLGA TEZCAN/E+/GETTY IMAGES

Filipino math teacher Emma Rotor worked on a crucial World War II weapons technology

By Erwin R. Tiongson

As an amateur historian and author of a book about Philippine-American history in Washington, D.C., I've long been familiar with the story of Arturo Rotor and Emma Unson Rotor.

In 1941, the couple moved from the Philippines to Baltimore for graduate studies. Then came World War II. Shortly after the attack on Pearl Harbor, Japan invaded the Philippines, occupying it for three years. The Philippine Commonwealth Government (the Philippines had not yet gained independence from the United States) escaped to Washington in May 1942; Arturo joined the government in exile soon after.

Arturo, who became secretary to the Philippine president and secretary to the Commonwealth cabinet, was something of a Renaissance man. He was an accomplished medical doctor and professor, a prize-winning writer, a classically trained pianist and an orchid enthusiast. Both Rotor syndrome, an inherited disease that causes jaundice, and an orchid variety, *Vanda merrillii var. rotorii*, are named for him. He was a man "surrounded by myth," cultural historians Edilberto Alegre and Doreen Fernandez wrote in 1984.

In contrast, his wife, Emma, was seen as a supportive background presence. "Emma Unson Rotor sat quietly by [making] an occasional comment, illustrating the supportive, companionable intelligence with which she had seen master short story craftsman Dr. Arturo Rotor through his long and shining career," Fernandez wrote in an article describing an oral history project focused on Filipino writers. In the postwar period, Arturo and Emma lived back in the Philippines. Arturo remained a public figure, active in medical and literary circles, while Emma taught math at Assumption College, a private school in Makati.

But Emma Rotor was not only a devoted wife and math teacher. While living in the United States, she conducted groundbreaking weapons research.

How the fuze changed warfare

In late 2021, I read an article by Maria Isabel Ongpin published in the *Manila Times* claiming that Emma Rotor was involved in weapons research during the war. I was astonished. "Mrs. Rotor was a teacher of higher mathematics, a subject I stayed away from, knowing my non-quantitative mind," Ongpin wrote. "But I had her in geography, and she was great. Mrs. Rotor as a mathematician had worked in the Manhattan Project, the secret project that produced the atomic bomb." I reached out immediately to Ongpin to ask for any reference material she might have that documented Emma Rotor's time with the Manhattan Project. She replied that it was simply known by everyone who studied at Assumption College, where Emma taught until the 1980s.

Emma Rotor was often portrayed as a devoted and supportive wife to her more famous husband, the writer Arturo Rotor.

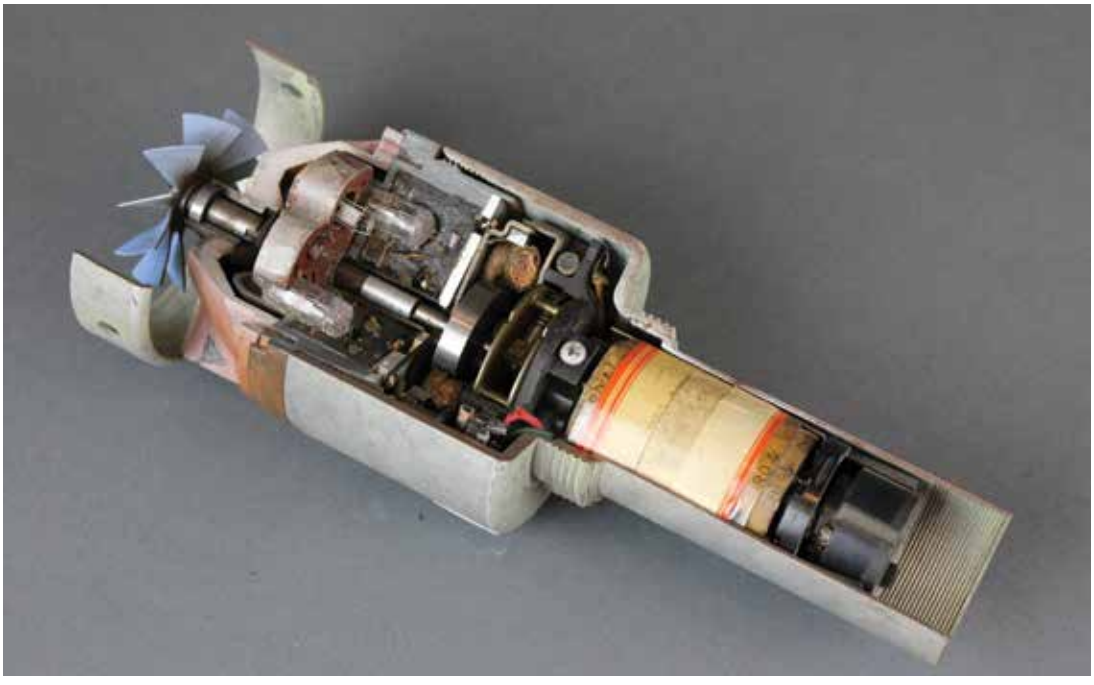
D. UNSON AND R. UNSON

After searching through archival material for several months, I found no evidence that Emma was involved in the Manhattan Project. But Emma was indeed part of another weapons research program, arguably just as consequential. She became a key member of a U.S. government division that developed an early version of the proximity fuze, the mechanism that makes a shell or bomb detonate when it gets close to its target rather than on impact.

Before the proximity fuze, bombs were known to be inefficient, detonating prematurely and away from their intended targets. An often mentioned and telling statistic is that it took 20,000 shells to take down one aircraft during the London Blitz. First used in June 1943 to bring down a Japanese aircraft, the proximity fuze, which relied on radar technology, was used in major battles in Europe and Asia. In June 1944, the fuze enabled the British to take out nearly 80 percent of German V-1 "buzz bombs." The fuze was, according to the War Department,

UNSUNG CHARACTERS

This article is part of a *Science News* series highlighting people of science — past and present — who we believe should be better known. Watch for more of these stories, and send your ideas to editors@sciencenews.org



The proximity fuze caused a shell or bomb to explode when it got close to its target, rather than requiring an impact.

NATIONAL INSTITUTE OF STANDARDS AND TECHNOLOGY DIGITAL COLLECTIONS, GAITHERSBURG, MD 20899

"one of the outstanding scientific developments of World War II" and "second only to the atomic bomb."

First headed by the British in the 1930s, proximity fuze research was later led largely by the U.S. government. The National Defense Research Committee assigned the task of developing fuzes "for non-rotating (e.g., fin-stabilized) munitions such as bombs, rockets and mortar shells" to the Ordnance Development Division at the National Bureau of Standards, or NBS, an agency of the U.S. Department of Commerce that is now known as the National Institute of Standards and Technology. The work on fuzes for rotating projectiles was assigned to the Department of Terrestrial Magnetism at the Carnegie Institution and to the Johns Hopkins Applied Physics Laboratory.

Work on the fuze

Emma Rotor first came to the United States in October 1941, at the age of 28, a few months after Arturo moved to Baltimore to begin his studies of allergies and medical history at John Hopkins. She had planned to study physics there while on leave from the University of Santo Tomas in Manila, where she was a mathematics instructor. The war interrupted those plans. She was undeterred, however, and worked briefly as a stenographer at the Enoch Pratt Free Library in Baltimore.

The fuze was "one of the outstanding scientific developments of World War II" and "second only to the atomic bomb."

"Oh I was not discouraged," she said in 1942 in an interview with the *Baltimore Evening Sun*. "Did I not see in the papers long columns of advertisements — girls wanted, no experience, good salaries? Well, I can work, can't I?" Though cut off from family, "we had to learn not to worry, not to think about the things we could not change," she said. As Emma talked, the article reported, "[h]er face is rich with smiles that soften the features and round out the contours." She is telling a story that is not happy, it continued, "except in that she makes it so."

In January 1944, Emma joined the Ordnance Development Division, led by radio engineering pioneer Harry Diamond. Hired as a physicist in a "war time appointment," she was tasked with supporting "experimental investigations connected with the development of new ordnance devices," including the "design, construction and testing of mechanical, electrical and radio devices," according to her appointment letter, which I tracked down with the help of independent researcher Emma Prince, based in St. Louis.

Keith Martin, a librarian at NIST, with whom I exchanged email messages, estimates that there were a total of 155 staff members in the division at the time, including those not involved in proximity fuze research, and about 31 in Emma's section.

She was promoted in less than a year, taking on her own analytical work. And in March 1945, her supervisor, physicist William B. McLean, gave her an

"excellent" performance rating. "Regardless of grade, Mrs. Rotor is one of the most valuable individuals in the present project," McLean wrote.

During her time with NBS, which ended in 1947, Emma wrote several scientific papers or coauthored them with colleagues, including Albert G. Hoyem, a physics professor at the University of Iowa in Iowa City who later became a senior official at the Naval Ordnance Test Station at China Lake in California. Her papers on "Air travel required for release of arming cover" and on "Measurement of dynamic propeller unbalance" are cited in the 1946 summary report of the Ordnance Development Division's work on the proximity fuze, titled "Radio Proximity Fuzes for Fin-Stabilized Missiles."

"Evaluation of the toss technique" (which she authored with Hoyem) reports the results of tests to map out trajectories of bombs. The paper appears in a collected volume of scientific articles on "Bomb, Rocket, and Torpedo Tossing" published in 1946 that also credits Emma "not only for the contributions mentioned above but also for her diligent supervision of the review and assembly of the final manuscript." She appears to be the only woman among the authors.

NIST's museum features a proximity fuze and a fin-stabilized missile that, as Martin wrote via email, "she, and others, had a hand in developing."

Emma Rotor's legacy

Arturo died in 1988 and Emma in 1998. The couple never had children of their own, but a niece, Delia Unson, and a grandniece, Ria Unson, remember Emma fondly. "Tita Emma was an amazing teacher," Delia, a retired clinical psychologist, recalls. "She could teach anyone and get them to love or at least like math. I met some of her students and found that they loved her!" Emma thought math was easy, Delia says, "because you didn't have to remember a lot. As long as one remembered the basic formulas and laws."

Ria remembers spending many afternoons when she was young at Emma's house. "She was an accepting, nonjudgmental person," Ria says. A visual artist, Ria has been keeping alive her memories of Emma through her art.

I asked Ria if she thought Emma would have been comfortable with an article highlighting her role in weapons research. She paused. "I don't think she looked for the spotlight," Ria said. "She just did what was asked."

It is clear, however, that Emma didn't talk openly with her family about this aspect of her life. A couple of family members did think briefly that she might have been part of the Manhattan Project. "Tita, did you work on the bomb?" Ria recalls her father asking Emma, probably at a family gathering decades ago. No, Emma replied with a faint smile. "I worked on the fuze."

The visual artist Ria Unson keeps her great-aunt Emma Rotor alive through art. Unson altered a book with a portrayal of Rotor to "write her back into history."

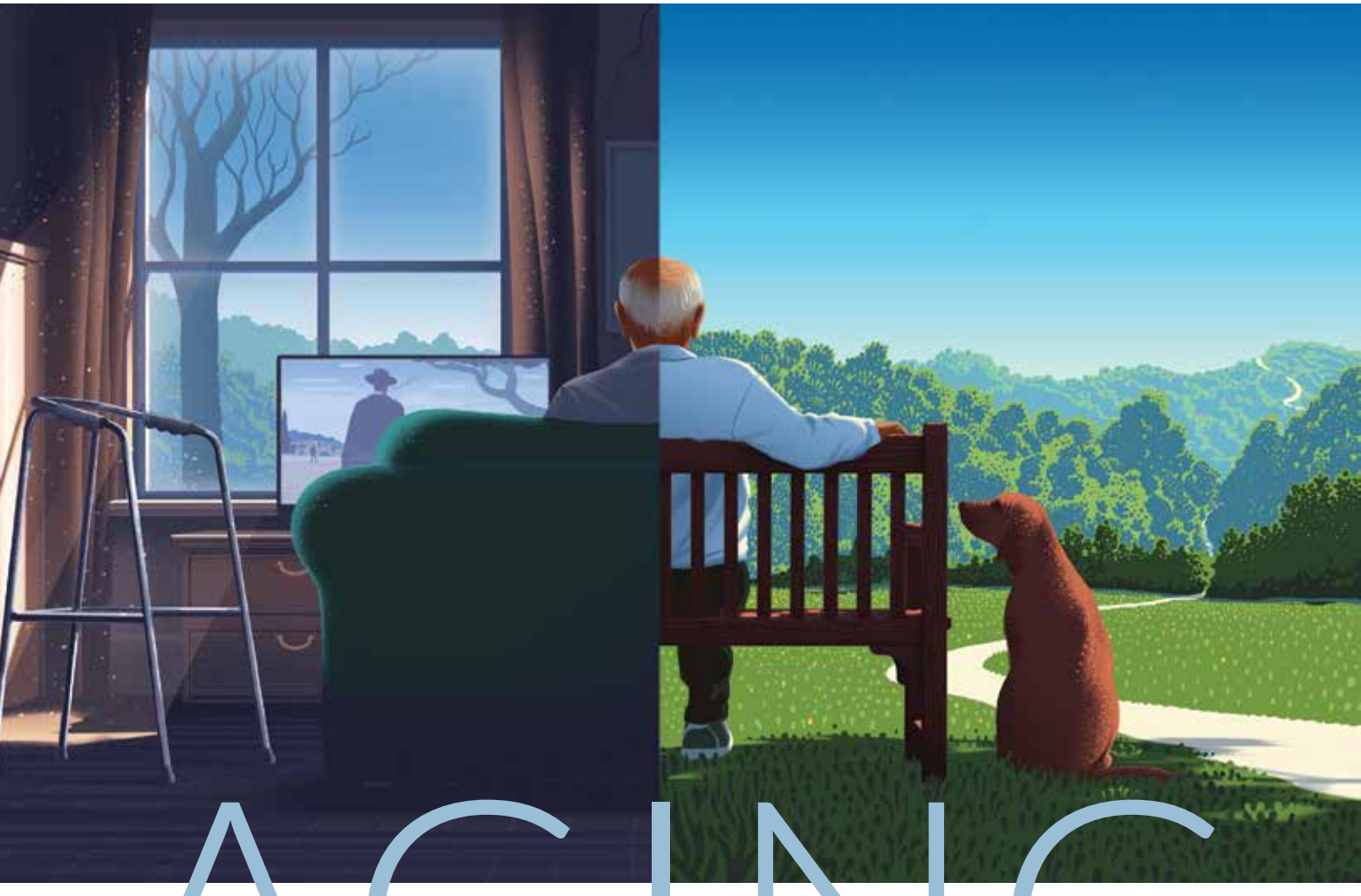
No one understood what she meant then, Delia and Ria said, until I showed them a few months ago her work on the proximity fuze. "That was so like her," Ria says, thinking of that moment at the family gathering. "She wanted people to know but didn't want people to know." ■

Explore more

- Listen to the author, Erwin R. Tiongson, discuss Emma Rotor's life and legacy on a recent episode of the *Lost Women of Science* podcast at bit.ly/emmarotorpodcast

Erwin R. Tiongson is a professor at Georgetown University in Washington, D.C., an author and a community historian.

BOTH: "EMINENCE GRISE" BY R. UNSON



AGING WITHOUT ILLNESS

Basic biology may point to drugs that can foster health in old age

By Cassandra Willyard

Each morning after breakfast, Scott Broadbent takes a plastic bottle from the refrigerator in his home in Alameda, Calif., pops the top, and drinks the contents, 2.5 ounces of milky liquid. "It has sort of a pineapple creamy flavor," he says. "It's really not bad."

The bottle might contain ketone ester, a supplement meant to help the body burn fat instead of carbohydrates. Researchers are now testing whether it might also slow the aging process. Or Broadbent might instead be getting a placebo. He is part of a clinical trial at the nearby Buck Institute for Research on Aging to assess the supplement's safety and side effects in older adults.

A retired chemist who used to work for pharmaceutical

SAM CHIVERS

companies, Broadbent is 70 and in excellent health today, but he worries about the future. He's not necessarily afraid of dying, but he doesn't want to be sick and in pain as he grows older. His dad had Parkinson's disease. Broadbent survived prostate cancer and recently developed tinnitus, which spooked him and sparked anxiety attacks. "I thought if I had to live like this the rest of my life, I don't know if I'd want to do it," he says.

Some scientists think there's a better way. These researchers—part of a burgeoning field called "geroscience"—aren't seeking immortality. The focus is much more pragmatic: By addressing the root causes of aging, they hope to stave off the disability and diseases that can make old age so miserable. They want to help people feel healthy for longer, compressing the years of illness that often accompany old age into a much shorter time frame. "Let's build a medicine that would be safe enough for someone in midlife to take almost like a supplement, like a daily vitamin, but with much more profound biological effects," says James Peyer, CEO of Cambrian Bio in New York City.

Just don't call these potential medicines antiaging therapies. "That term is associated with an industry that is trying to sell products to the public to separate people from their money," says S. Jay Olshansky, a demographer and geroscientist at the University of Illinois Chicago. The antiaging market includes everything from face creams meant to zap wrinkles to pills that promise to turn back the clock. "It's bogus," he says. Geroscientists instead are doing legitimate research at respected research institutions to find medicines that can slow the aging process. Many of the compounds under study show promise in mice and even humans, and some are in clinical trials.

Better health in old age is not just about individual benefits. By 2030, 73 million baby boomers in the United States will be 65 or older. By the same year, experts project, there will be a billion people 65 and older globally. And though people are living longer, they are not necessarily living healthier than previous generations. "There's this fear of what is this going to do to our health care system," says Laura Niedernhofer, a geneticist and researcher studying aging at the University of Minnesota in Minneapolis. "And it goes well beyond just health care. We don't have the nursing homes. We don't have the personal care staff to deal with this at all." Drugs to help keep older adults healthy, active and independent would be a societal boon.

But whether developing such drugs is even possible remains to be seen. Getting the medicines to market means securing more funding, overcoming stumbling blocks related to study design and combating near constant hype.

The origins of geroscience

The advent of modern medicine and public health has more than doubled the average human life span—from about 30 in the early 1800s to more than 70 today. "This is perhaps one of the biggest things that has happened to humankind, period," says Jamie Justice, a geroscientist who heads the health domain at the XPRIZE Foundation, which holds competitions to spur technological developments and announced a new prize related to aging in November. "We are a lot less dead than we used to be because of where public health and modern medicine has gotten us."

There's a downside, of course. We're living long enough to see the frailty and illness that comes with old age. Cells stop dividing, DNA degrades, the immune system falters. We become increasingly vulnerable to disease. Many of us spend our last decades beset by medical maladies—broken bones, weakness, dementia, cancer, heart disease and more. Doctors can do little more than play whack-a-mole, beating back illnesses one at a time.

For decades, scientists thought the gradual decline that comes with old age was unavoidable. But experiments in the 1980s and '90s suggested that the process might not be so fixed.

In one notable experiment, Cynthia Kenyon, a molecular biologist at the University of California, San Francisco, and colleagues found that mutations in a single gene in the roundworm *C. elegans* could double its life span (SN: 12/4/93, *p.* 375). Typical 13-day-old worms barely moved. "The animal is clearly in the nursing home," Kenyon said in a 2011 TED Talk. The mutant worms moved as if they were much younger, and they lived longer too.

For researchers interested in human health, this and similar findings from other teams led to a profound realization: Perhaps the aging process is malleable. If so, scientists might be able to develop therapies to attack the root of aging rather than simply combating the pileup of diseases.

By the late 2000s, "the whole perspective of the scientific community changed," says Felipe Sierra, who was then a program officer at the National Institute on Aging in Bethesda, Md. Aging biology moved from a phase of description into a phase of molecular investigation. Sierra wanted a name to bring the field together. He landed on *geroscience*, a word he had first seen in a grant proposal by another researcher studying aging, Gordon Lithgow. "Gero-" comes from the Greek word for old man. It wasn't difficult to convince other researchers to get on board. "Everybody listened to me because I was in charge of the money," says Sierra, now chief scientific officer at Hevolution Foundation, a nonprofit that funds geroscience research.

Promising compounds

Though there are no proven therapies for people yet, geroscientists are eyeing several compounds that can slow the aging process, at least in worms, fruit flies and mice (SN: 7/23/16, *p.* 16). Some have already been tested in humans, and many more clinical trials are under way. Which will work? "Let me see. Let me look at the crystal ball," Sierra jokes. "Who knows?"

Perhaps the best studied is rapamycin, a compound first discovered in a soil sample collected in 1964 from Rapa Nui, or Easter Island. Today, people who receive organ transplants take the drug to help keep their immune systems from rejecting the foreign tissue. But rapamycin also prolongs life in yeast, flies and mice. And it's being tested in people in clinical trials. How it counters aging isn't entirely clear. The drug inhibits a protein complex called mechanistic target of rapamycin, mTOR for short, which plays a role in cell growth and protein synthesis.

This inhibition appears to have wide-ranging effects, including reducing inflammation, clearing old and damaged cells, and altering cellular metabolism—some of the key processes that researchers think are to blame for the aging process.

Rapamycin isn't the only drug to impact mTOR. Researchers at the biotech company resTORbio tested other mTOR inhibitors in elderly adults to try and improve immune function. About 250 people participated in the clinical trial, which tested two mTOR inhibitors alone and in combination compared with a placebo. In 2018, the team reported that those who received the drugs had fewer infections and mounted a better response to the flu vaccine. The company tried one of those compounds in a subsequent study, though, and it failed to show an effect on self-reported respiratory illnesses. resTORbio no longer exists, but the company's chief medical officer, Joan Mannick, hasn't given up on mTOR inhibitors. She cofounded a new company called Tornado Therapeutics, based in New York City, that is working to develop new rapamycin analogs, or "rapalogs."

Another promising class of drugs targets cells that have stopped dividing but don't die. These senescent cells release chemical signals that can trigger inflammation, disrupt tissue repair and harm neighboring cells. In some cases, these signals even prod neighbors to become senescent too.

The drugs, called senolytics, aim to eliminate senescent cells by prompting them to commit suicide (*SN: 3/5/16, p. 8*). After showing promising results in mice, senolytics are now being tested in humans. More than 25 clinical trials have either been completed or are under way.

One of the most commonly tested senolytic regimens is a combination of two compounds: the anticancer drug dasatinib and quercetin, an antioxidant that occurs naturally in grapes, berries and other fruits and vegetables. Other research efforts plan to compare fisetin, a compound found in strawberries and apples, with a placebo to see if it has an impact on frailty and markers of inflammation in the blood.

Unity Biotechnology, based in San Francisco, is focused on senolytic therapies exclusively. The company's most advanced compound, called UBX1325, targets a protein abundant in the blood vessels and retina that regulates cell death. Preliminary results from a trial in patients with diabetic macular edema, a thickening of the retina related to diabetes, suggest that the compound can improve eyesight.

Diet is also known to profoundly affect the aging process (*SN: 6/4/11, p. 22*). Studies have found that the low-carb ketogenic diet, for example, can help mice live longer. But restrictive diets can be hard to follow and have side effects. Broadbent followed the ketogenic diet for a month or so, but his cholesterol levels went dramatically up. Ketone ester, the compound Broadbent might be downing each morning for the Buck Institute's clinical trial, may mimic the longevity benefits of such diets.

When the body runs out of glucose to use for energy, the liver creates another source by converting fat into molecules called ketone bodies. "If we don't eat for a day or so, we'll start to make ketone bodies," says John Newman, a geriatrician at the Buck Institute who is leading the trial. "And we'll make more and more the longer that we starve in order to fuel our bodies." These compounds are more than just fuel. They help regulate inflammation and control other cellular processes, many of them involved in the aging process. Drinking ketone esters, which quickly break down, is a way to deliver the ketone bodies without the diet.

Among the dozens of clinical trials testing potential gerotherapies, very few are yet assessing their ability to prevent the onslaught of diseases that come with aging. Instead, the goal is establishing safety or seeing whether a compound can nudge some biomarker in the right direction. And many of the potential treatments under study are natural compounds or existing drugs that are already off patent, which might leave drug companies hesitant to invest in future trials or to seek approval from the U.S. Food and Drug Administration or other regulatory agencies.

What's more, as James Kirkland, a geriatrician at the Mayo Clinic in Rochester, Minn., points out, many of the clinical trials happening now will fail. "In fact, most will," he says. That's just a part of drug development.

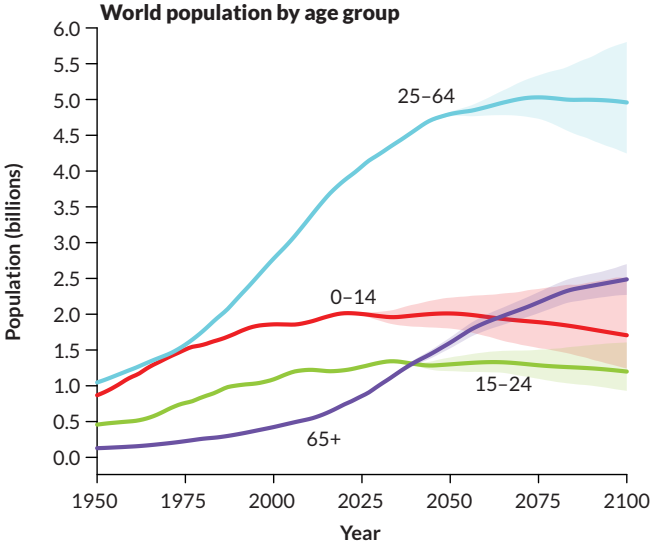
For now, some companies are bolstering their chances of success by pursuing many options. Cambrian Bio, the parent company of Tornado Therapeutics, for example, is "taking a number of different shots on goal," Peyer says. "We don't know which drug is going to really be the first multidisease preventative."

Barriers to progress

One of the big challenges for geroscience is to figure out how to show that a compound prevents age-related diseases in people. Scientists would have to give the drug to healthy people and then track their health as they age, an expensive and time-consuming endeavor. "In mice, it takes us four years. In humans, it would take decades and tens of thousands of people," Niedernhofer says.

Watching global growth Estimates suggest there will be a billion people age 65 and older globally by the year 2030, and the global figure could hit 2.5 billion by the end of the century.

SOURCE: WORLD POPULATION PROSPECTS 2022, UNITED NATIONS



C. CHANG

Hallmarks of aging

Researchers have identified a dozen hallmarks of aging that appear capable of nudging life span — and likely health span — one way or another across a range of animals. A network of interactions among genes and their products underlies these interconnected hallmarks, and it's that network that drugs such as rapamycin and senolytics are targeting.

Genomic instability includes changes to DNA, chromosomal rearrangements and defects in nuclear architecture.

Telomere attrition is the cumulative damage or loss of DNA at the ends of chromosomes, called telomeres.

Epigenetic alterations can change how a gene is expressed without changing the DNA itself, such as methylation.

Loss of proteostasis refers to imbalances in proteins in the body, which can lead to the formation of protein aggregates, as in Alzheimer's.

Disabled macroautophagy leaves cells unable to sequester and digest material in the cytoplasm, thus affecting the replacement of organelles.

Dysbiosis is the disruption of the body's microbial communities, which play a role in nutrient digestion, disease protection and more.

Chronic inflammation increases with aging, both across the body and locally, and typically comes with a decline in immune function.

Altered intercellular communication brings more noise into the system, disrupting neural, neuroendocrine and hormonal signaling.

Stem cell exhaustion leads to a reduction in tissue renewal rates across the body and can limit tissue repair after injury.

Cellular senescence, when cells stop dividing but don't die, has been linked to kidney disease, diabetes, Alzheimer's, Parkinson's and more.

Mitochondrial dysfunction can prevent a cell from producing the energy it needs, as well as contribute to inflammation and cell death.

Deregulated nutrient-sensing affects a whole series of pathways that shape cell activity, which makes diet a practical aging intervention.

Linked up In one example of interconnections, several hallmarks can affect cellular senescence, which then drives inflammation and dysbiosis.

ICONS: C. LÓPEZ-OTÍN ET AL/CELL 2023

An easier path to the clinic would be to develop these therapies as a treatment for a single disease rather than a multidisease preventive. "That's something that the FDA is very comfortable with," says Nathan LeBrasseur, a researcher studying aging at the Mayo Clinic. Once the drug is approved for one indication, it would be much easier to seek approval for others — and potentially widen to a preventive. "The absolute irony of all of this is, to try and advance geroscience, we're going right back to the old way of doing things, which is one disease at a time," Niedernhofer says. For example, Unity is developing its lead antiaging candidates as therapies for a variety of eye diseases.

One group of researchers, however, has developed a clever work-around. Rather than treating healthy people and waiting for them to age, the team has devised a study that will recruit people who have one age-related disease and assess whether a drug reduces the time it takes to develop another. In this case, the researchers have chosen the diabetes drug metformin (SN: *11/30/13, p. 18*). Metformin has a long safety record, and studies suggest it can impact heart disease, cancer and cognitive decline. Metformin may even reduce the risk of long COVID. The precise mechanisms that underpin these effects aren't entirely clear. The study, called Targeting Aging with Metformin, or TAME, will look at things such as cardiovascular events, cancer, cognitive decline, dementia and death.

But nearly eight years after investigators first announced the 3,000-person trial, they're still trying to get together funding. Metformin is cheap and readily available, no longer protected by a patent, so drug companies have no incentive to develop it for aging. Nir Barzilai, director of the Institute for Aging Research at the Albert Einstein College of Medicine in New York City, who is leading the study, has started telling people the trial will start in January. "And as long as I don't say which year, it will be true," he jokes.

Part of what holds the field back is society's approach to medicine, says Justice, of the XPRIZE Foundation. "We have a model of medicine that prioritizes treatment of diseases," she says. "You get one disease. You treat one disease. You restore homeostasis." But that approach isn't relevant for aging. Geroscientists also have

Kate Creevy (with dogs Poet and Sophie) is chief veterinary officer of the Dog Aging Project, an effort that seeks to identify factors contributing to dog longevity and may offer insights for humans too.

to fight society's views of aging. The mind-set is "things get old. It just happens. There's nothing you can do, just let it go," Justice says. "I think that is actually a fundamentally ageist view, as if people who are older don't have a right to health."

Geroscientist Matt Kaeberlein of the University of Washington in Seattle agrees. No one would argue that we shouldn't develop therapies for Alzheimer's or cancer because these diseases are a natural part of getting older. So, he asks, why would this argument hold for aging? "I don't really see what the field is trying to do as any different than trying to cure disease," he says. "It's just a much more efficient and effective approach, or it's likely to be."

Kaeberlein thinks dogs, because they age much faster than people, could bridge the gap between lab studies in mice and therapy approval. "You can do longevity clinical trials in dogs that you can't do in people," he says. And companion animal medications are regulated much like human medications. The Dog Aging Project, which Kaeberlein codirects, is testing rapamycin in 500 middle-aged dogs to assess its impact on the heart, the immune system, cancer incidence and cognition.

But even if geroscientists find compounds that treat the decline that comes with aging, it's not clear whether they will compress the period of age-related illness—that's the goal—or simply delay it. It's also uncertain whether such therapies might add to the average life span and, if so, how many years. John Davis, an ethicist at California State University, Fullerton, worries about how such increases might affect demographics. If the average life span jumps to 120 years, the effects aren't too pronounced, he says. If it goes far longer, there is the potential for "really spectacular increases in population on a planet that I personally feel is already overloaded." And then there are concerns about how longer life might impact existing inequalities. "Billionaires who live longer have more time to accumulate even more wealth," Davis adds.

Beware the hype

There's another potential obstacle: hype.

No one can say with any certainty that there will be a pill to prevent aging. Yet that hasn't stopped some companies and less scrupulous researchers from cashing in. The public's interest in escaping aging makes overselling results enticing. In 2019, for example, the FDA warned consumers against receiving infusions of plasma from young donors, which some companies offered for $8,000 a liter. The treatment was based on promising mouse studies that gave older mice blood from younger mice, either via transfusions or by connecting the two animals' circulatory systems. The young blood appeared to rejuvenate the older animals' muscles, livers and brains (*SN: 5/31/14, p. 8*), but the benefits haven't been proved in human patients.

The internet is likewise rife with supplements and even prescription medications with purported antiaging benefits. Many of the pills being touted as age-reversing miracle drugs are the same compounds geroscientists are currently testing in trials. "[Companies] start trying to sell it to the public before it's been tested for safety and efficacy," Olshansky says. That can make it especially difficult for consumers trying to sort fiction from fact.

LeBrasseur is concerned that if scientists oversell their progress, they run the risk of losing public trust. And a rush to clinical trials might lead to safety problems. "If something bad happens, that's going to set the entire field back," he says. "I just think we have to be patient and humble."

A student of Newman's, at the Buck Institute, asked him recently how geroscience has changed clinical practice. "The honest answer is it hasn't at all," he says. "We're still building the data. We're still running the clinical trials waiting for it to come in. And this all takes time."

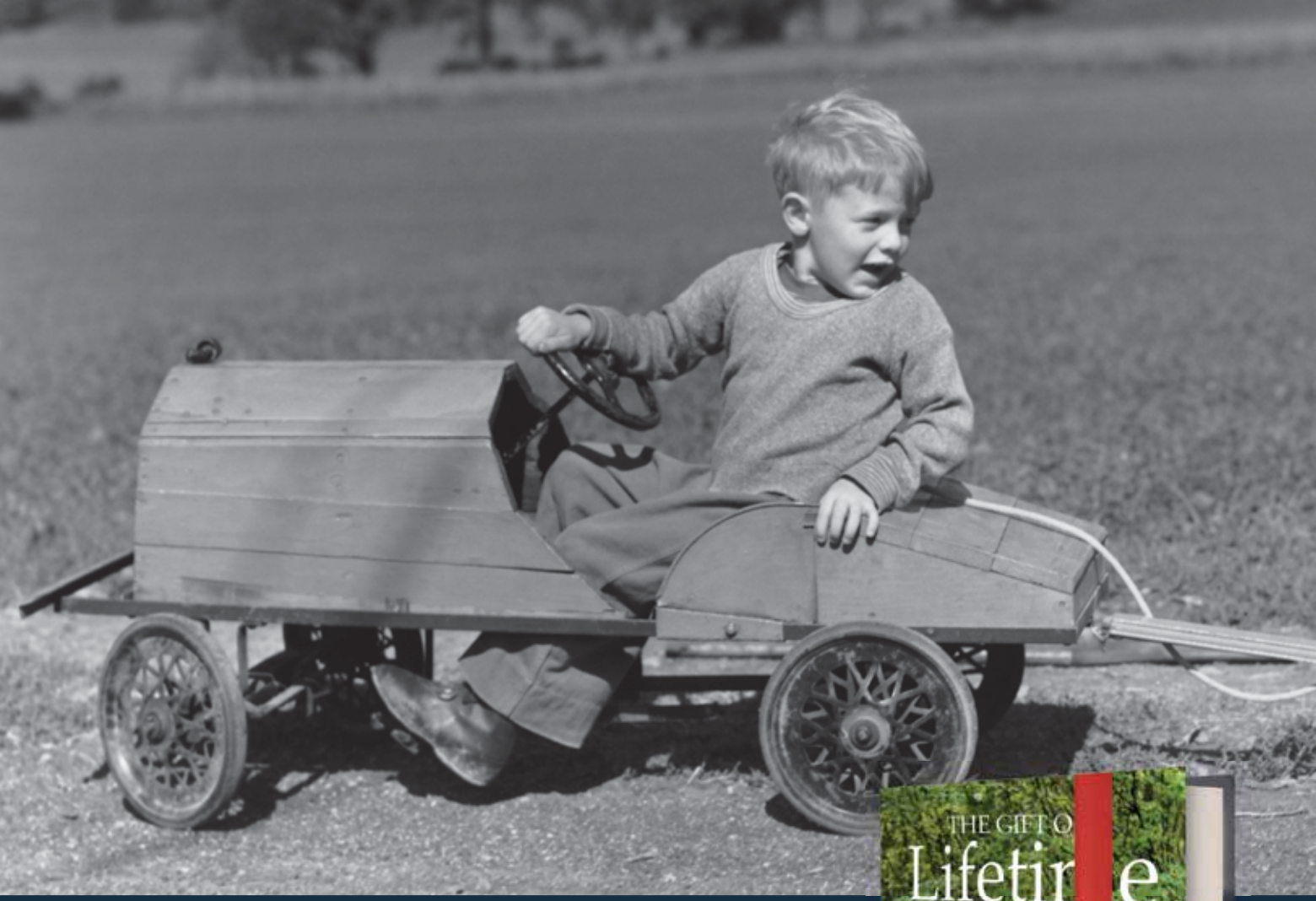
But Broadbent, and many others, aren't satisfied waiting for settled science. He likes to do his own research, and ketone ester seems like a good bet. The particular product he may be drinking as part of the clinical trial is now being sold as a supplement, and he plans to continue taking it after the trial ends. "I'll sign up right away," he says. "I'll be on an annual subscription."

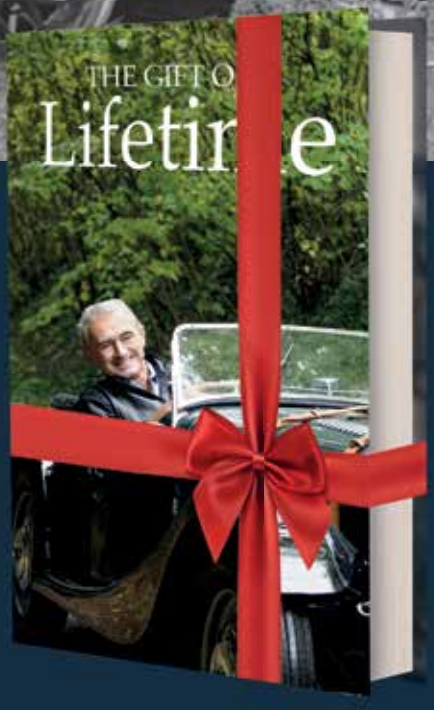
Many scientists are convinced that the geroscience revolution is coming. Until then, there's a tried-and-true method for improving your health span. It's the advice any doctor will give you: Eat a balanced diet, exercise, get vaccinated and avoid tobacco and alcohol. ■

Explore more

- For more on the Dog Aging Project, visit dogagingproject.org

Cassandra Willyard is a freelance science journalist based in Madison, Wis.





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CONVERSATIONS WITH MAYA

DR. PETER HOTEZ
Internationally recognized physician-scientist in neglected tropical diseases and vaccine development

Maya Ajmera, President & CEO of the Society for Science and Executive Publisher of Science News, chatted with Peter Jay Hotez, Dean of the National School of Tropical Medicine and Professor of Pediatrics and Molecular Virology and Microbiology at Baylor College of Medicine. Hotez is the Codirector of the Texas Children's Hospital Center for Vaccine Development and the Endowed Chair of Tropical Pediatrics at Texas Children's Hospital.

What sparked your interest in science?

I grew up in West Hartford, Conn., and had an early passion for studying microorganisms. My parents bought me a pretty serviceable microscope, and I used to collect water from a local brook and look for protozoa. I also had a passion for maps. I guess if you combine microorganisms with maps, you get parasitology and tropical medicine. I knew early on in my life that that's what I wanted to do.

Do you remember your STS project?

I do. My physics teacher at the time was a fantastic guy named Daniel Hoyt. He was one of those teachers who purchased a lot of used scientific equipment for his classroom. I worked with earthworms, including taking electrocardiogram recordings of earthworms. This led to my STS project titled "Effect of Acetylcholine Chloride and Atropine on the Electrocardiology of *Lumbrucus* and its Relation to Mammalian Smooth Muscle."

Throughout your career, you have worked to address neglected tropical diseases (NTDs). What major opportunities for progress stand out to you today?

I began investigating NTDs as a research scientist, and that thread still continues. Today, I devote my life to vaccines for neglected diseases. Our team of scientists at the Texas Children's Hospital Center for Vaccine Development at Baylor College of Medicine has accelerated two parasitic disease vaccines for hookworm and schistosomiasis, which are in Phase 2 clinical trials. We also have a new Chagas disease vaccine entering a Phase 1 trial, and we used that same approach to make two low-cost COVID-19 vaccines. The health economists we collaborate with said all our vaccines should be a couple of dollars a dose if we want them to be used. We therefore always use low-cost approaches to produce vaccines, like microbial fermentation and yeast.

Twelve years ago, we adopted a coronavirus program and started making vaccines for SARS and MERS. Then when the COVID-19 sequence came online in early 2020, we made two low-cost COVID vaccine technologies that were adopted by India and Indonesia. We transferred the tech to the pharmaceutical companies Biological E and BioFarma, which scaled up and produced Corbevax and IndoVac, respectively. One hundred million doses were administered. With that, we were able to provide proof of concept that you don't have to be a big pharma company to do big things. That has been very meaningful.

On the policy end, the focus has been getting people to care about neglected tropical diseases, which represent more than a dozen major chronic infectious diseases mainly prevalent in tropical areas. Together with a couple of colleagues from the United Kingdom, we wrote one of the first scientific papers using the term neglected tropical disease back in 2005.

That policy and advocacy experience positioned me well to push back against anti-vaccine groups, which became

another very important aspect of my life. I have four adult children, including Rachel, who has autism and intellectual disabilities. A few years ago, I wrote a book called *Vaccines Did Not Cause Rachel's Autism*, which wound up making me public enemy number one with anti-vaccine groups.

My life has this duality to it: being a working scientist—which includes keeping up with lab meetings, grants, papers, major revisions and resubmissions—and also having a foot in public engagement, including defending vaccines and explaining to the public why they should care about NTDs.

As an advocate for vaccine diplomacy, can you explain what that means and how we can encourage more international cooperation in public health?

During the COVID-19 pandemic, the thinking was that only the big pharma companies had the chops to fight the virus. And eventually the crumbs would filter down to the low- and middle-income countries. But that was a failed policy, right? It did not work well; we knew it was going to fail.

I have spent decades with this concept that we can make the early prototype vaccines in our research laboratories in Texas, but then transfer the technology and ownership to vaccine producers in low- and middle-income countries so that they can take the lead on owning the technology and scaling it up. That's what we did for COVID-19. It's what we do for our parasitic disease vaccines. We're hoping to do it for our hookworm vaccine, which is now looking very promising.

Even before COVID-19, I had the opportunity to test this out in 2015 when the Obama administration appointed me as U.S. Science Envoy. I worked with the U.S. State Department to build vaccine diplomacy initiatives with Muslim-majority countries in the Middle East and North Africa.

You have been a champion of science communication throughout your career. How can scientists more effectively communicate complex topics to the general public?

Unfortunately, I had to learn it by trial and error, and I often say it was more error than trial. I believe we should teach science communication in medical schools, Ph.D. programs and during postdoctoral training.

Dr. Peter Hotez speaks at an event hosted by Rice University's Baker Institute for Public Policy in Houston.

I have found that a lot of the stuff that's dogma around science communication turned out to be false. Everyone always told me, "Peter, you gotta talk to the American people like they're in the fourth grade. Don't make it complicated. You're gonna really have to dumb it down and not use jargon." Well, they were right about not using jargon, but dumbing it down was not very successful. It makes you sound condescending, which is a real turnoff for a lot of people. From my experience, Americans like it when you speak to them like educated adults, and they are willing to tolerate a considerable level of complexity.

You have been a leading voice for the efficacy of vaccines. Are you concerned about the mainstreaming of anti-scientific views?

In recent years, the anti-vaccine movement has gone from pushing phony stuff around autism to becoming embedded in a major U.S. political party. They began attacking biomedicine like climate change deniers were attacking climate science a decade ago. This has become a very dangerous movement. In my most recent book, *The Deadly Rise of Anti-science*, I point out that the anti-science disinformation vaccine machine is so powerful now that it convinced 200,000 Americans not to take a COVID-19 vaccine after vaccines were widely available during the COVID-19 delta wave, which led to their deaths.

They were victims of this anti-science disinformation machine. It's organized, it's deliberate, it's politically motivated, it's predatory and it's a killer. That's why I wrote the book. It's not a theoretical or academic discussion. Improving science communication and combating anti-science is now more important than ever.

There are many challenges facing the world today. What keeps you up at night? What gives you hope for the future?

What keeps me up at night is that I think we're in a new normal with pandemics. We've had SARS in 2002 and MERS in 2012. COVID-19 is just a part of that cadence. By that reasoning, we're going to have another major coronavirus epidemic or pandemic before the end of this decade. We're also at risk for emerging viruses transmitted by mosquitoes, such as dengue, Zika, chikungunya or even yellow fever.

It's happening because of a confluence of physical and social determinants: climate change working hand in glove with urbanization, poverty and human and animal migrations. This new normal is very troubling for me. Equally troubling is the fact that you've got groups with nefarious intent that want to prevent us from taking on these challenges.

What gives me hope is the strength of our research universities and institutions, seeing young people who still want to become scientists, and seeing the kinds of science outreach that Society for Science is doing. Seeing people still striving to do big things in science—that's what gives me hope.

EXHIBIT

Step into the world of elephants and rediscover our connection with them

For millions of years, over a dozen species of proboscideans, the group that includes mammoths, mastodons and elephants, roamed landscapes as varied as Arctic tundra and African savannas. Then, around 11,700 years ago, as the last ice age waned and human hunting picked up, this number dwindled to just three: the Asian elephant, the African forest elephant and the African savanna elephant — all of which are now endangered.

Losing elephants comes with a far-reaching and underappreciated impact, warns "The Secret World of Elephants." The exhibit, now on display at the American Museum of Natural History in New York City, explores that impact, as well as what modern science is revealing about elephant minds and bodies.

As ecosystem engineers, modern elephants transform their environments. Because these herbivores are so big, they eat a lot, poop a lot and travel far to graze, dispersing mounds of viable seeds far and wide. The beasts also trample fields, making room for a diversity of plants, and dig enormous water holes that other animals use too. In North American grasslands, the departure of mammoths and other large herbivores homogenized the land, covering rich landscapes with slow-growing plants and invasive weeds, says curator Ross MacPhee, a mammalogist at the museum.

The Secret World of Elephants
NOW OPEN
AMERICAN MUSEUM OF NATURAL HISTORY
New York City

An African elephant's skeleton and muscles are projected onto a life-size model in an exhibit that highlights new science on proboscideans.

The loss is cultural too. Though the exhibit is small, it's packed with displays focusing on how elephants shape — or have been incorporated into — our lives. The animals appear in early cave drawings and in religious stories. (This connection hasn't always benefited elephants, though, as humans have trained them as war steeds and circus performers.)

The exhibit aims to rekindle our connection with elephants by emphasizing that they, too, are smart and social creatures. They can recognize themselves in mirrors, a potential sign of self-awareness, and can use tools. Videos in the exhibit show elephants playing with and comforting one another. By pressing buttons on an interactive board, visitors can also hear calls that appear to convey fear, affection and annoyance.

Much of the exhibit's appeal comes from its exploration of how elephants differ from us. The animals, for instance, hear not just with their ears but also with their feet, picking up on the subtle vibrations of the ground beneath them. They also have a superior sense of smell, with more smell receptor genes than even dogs. And it's hard to fathom that the average adult male African elephant consumes some 150 kilograms of food every day.

It's amazing to see all that researchers can glean from what the animals leave behind. Chemical analyses of tusks, for instance, can reveal what an elephant ate and where it traveled during its lifetime. DNA samples, meanwhile, show unexpected evolutionary connections between elephants and animals that look nothing like them, including aardvarks and tenrecs.

By including grand visuals and tactile elements, the exhibit is appealing to visitors of all ages. During my visit, a group of schoolchildren admired a life-size model of an elephant and stroked a replica of the creature's wrinkly skin. I ran my fingers along the ridges of a fossilized mammoth tooth and better understood how they served as a tool for grinding up tough grasses.

Overall, the exhibit sparks both curiosity and alarm. "If we don't act quickly," a sign states, "elephants could be gone before we ever truly get to know them." The exhibit's curators hope that "The Secret Lives of Elephants" inspires visitors to help stave off the animals' extinction.

Supporting local nonprofits that protect elephant habitats is one way individuals can help, the exhibit suggests. Another is to abstain from purchasing ivory or other elephant products. Although it is illegal to sell ivory in the United States, the European Union and China, a thriving black market fuels rampant poaching, on such a scale that some elephants may be evolving to lose their characteristic tusks (SN: 11/20/21, *p.* 15).
— *Saima S. Iqbal*

ALVARO KEDING/© AMNH

NOVEMBER 4, 2023

A peek inside

Neutron tomography can help scientists capture 3-D images of the insides of fossils and artifacts without damaging them. The technique can uncover hidden features within dense material that X-ray scanning can't detect, ***James R. Riordon*** *reported in "Seeing into the past"* (SN: 11/4/23, p. 18).

Reader **Rob Janes** asked how the 3-D images are captured.

In neutron tomography, scientists blast beams of neutrons at an object they want to study, **Riordon** says. Detectors on the other side of the object record the proportion of neutrons that make it through without being reflected or absorbed along the way. Using that data, computer algorithms create virtual slices of the object, which can then be assembled to provide 3-D views of the object's interior, he says.

Reader **Heidi Wilson** asked whether neutron tomography has been used on ancient manuscripts that can't be unfolded.

X-ray computed tomography has been the go-to method for analyzing ancient manuscripts, **Riordon** says. The artifacts are generally made of low-density material, such as papyrus or parchment, that X-rays can effectively image.

One recent exception, reported in 2021 in *Archaeometry*, is a medieval amulet made of a folded lead scroll that contains inscriptions. Since X-rays cannot easily penetrate particularly dense materials like lead, the researchers turned to neutron tomography to virtually unfold the sheet and reveal the inscribed runes. "Although ancient metal manuscripts are comparatively rare, neutrons offer a way to read them without risking the damage that opening them up would cause," **Riordon** says.

Thinking about loneliness

Social scientists are learning more about how feeling detached from animals, places and routines — not just people — can cause loneliness. These revelations may lead to new interventions, ***Sujata Gupta*** *reported in "What is loneliness?"* (SN: 11/4/23, p. 24).

"While reading [this story], I found myself once again thinking about the demise of the front porch in our communities.... Having grown up in a small town in western New York that came into its own in the mid-1800s, front porches are to be found on most of the older homes," reader **Jim Sobek** wrote. "A few years ago, while in town for a week of bicycling, I spent an evening with cousins on their front porch. As we whiled away the evening in conversation, neighbors out for their evening walks stopped by to talk for a while and then went on their way, only to be replaced by other neighbors also taking their evening constitutionals. Warm greetings and friendly conversation were exchanged between neighbors who knew each other well."

Many modern homes "have large backyards, large decks, some pools and always-spacious air-conditioned interiors, but at best only a small covered concrete front stoop," **Sobek** continued. "The residents often do not speak with neighbors, and sometimes don't even know who their neighbors are." While these homes are more comfortable, **Sobek** wonders whether they have also contributed to the loneliness and loss of community that some people now feel.

Correction

In "Clear the air," the unit of the x-axis in the graph titled "Methane emissions in 2022, by source" was labeled incorrectly (SN: 11/18/23, p. 18). It should have been million metric tons, not metric tons.

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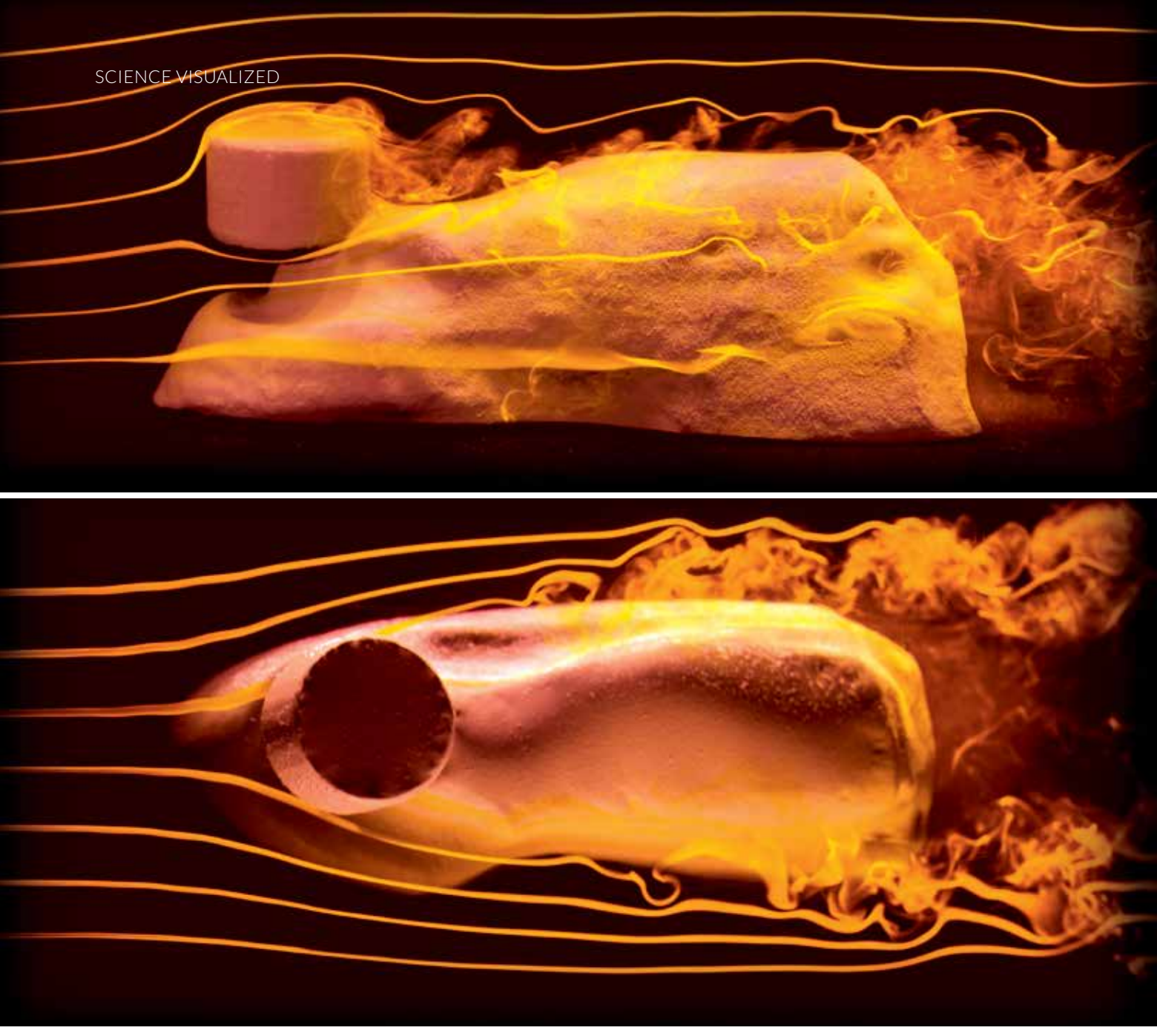

Before ancient Egyptians, nature may have sculpted sphinxes

Desert landforms called yardangs can resemble seated lions — so much so that some researchers think one might have had the honor of being carved to create the Great Sphinx of Giza.

The ingredients for these rock formations might be simple, researchers report in the November *Physical Review Fluids*. In a water tunnel, hand-sized globs of clay transformed into sphinx-shaped yardangs so long as two conditions were met: consistent prevailing fluid flow and a starting blob containing a mix of easily eroded and more resistant bits.

New York University mathematician Leif Ristroph and colleagues subjected hundreds of clay globs to muddy trials in a water tunnel. Each time, the team sculpted a stiff clay paste into a starting glob, embedded the glob with a chunk of hard plastic to represent harder rock, and plopped the glob into the tunnel to erode in a steady flow of water that mimicked wind. The setup reliably produced sphinxlike forms. The initial shape of the glob and placement of the plastic didn't matter much, so long as the plastic bit was in the windward half.

To get photos of the quickly dissolving shapes, the team coated 3-D printed replicas of the sphinxlike forms, like the one shown here from the side (top image) and from above (bottom), with clay laced with fluorescent dye. In the tunnel, the glowing clay — plus injected streams of dye — revealed whorling currents the team is now working to model mathematically. Those currents include a turbulent "mane" of eddies behind the sphinx's head that carves out a sloping, feline spine. — *Elise Cutts*

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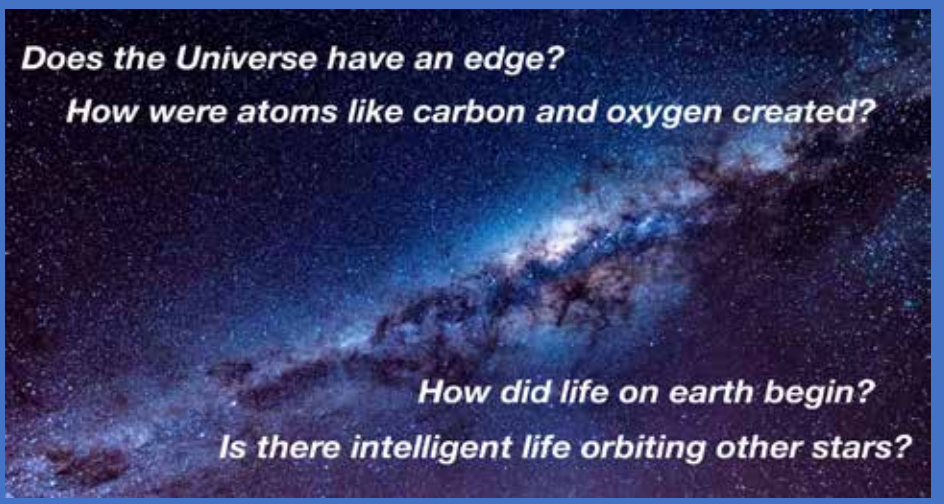

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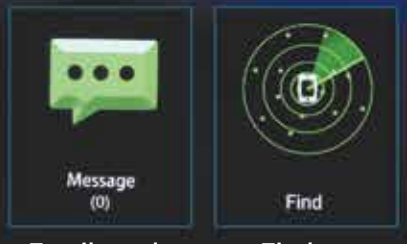

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