SCIENCENEWS.ORG | VOL. 208 NO. 8 | AUGUST 2026

ScienceNews

SKELETONS IN THE CLOSET

Research institutions still hold human remains collected without consent. What's next?

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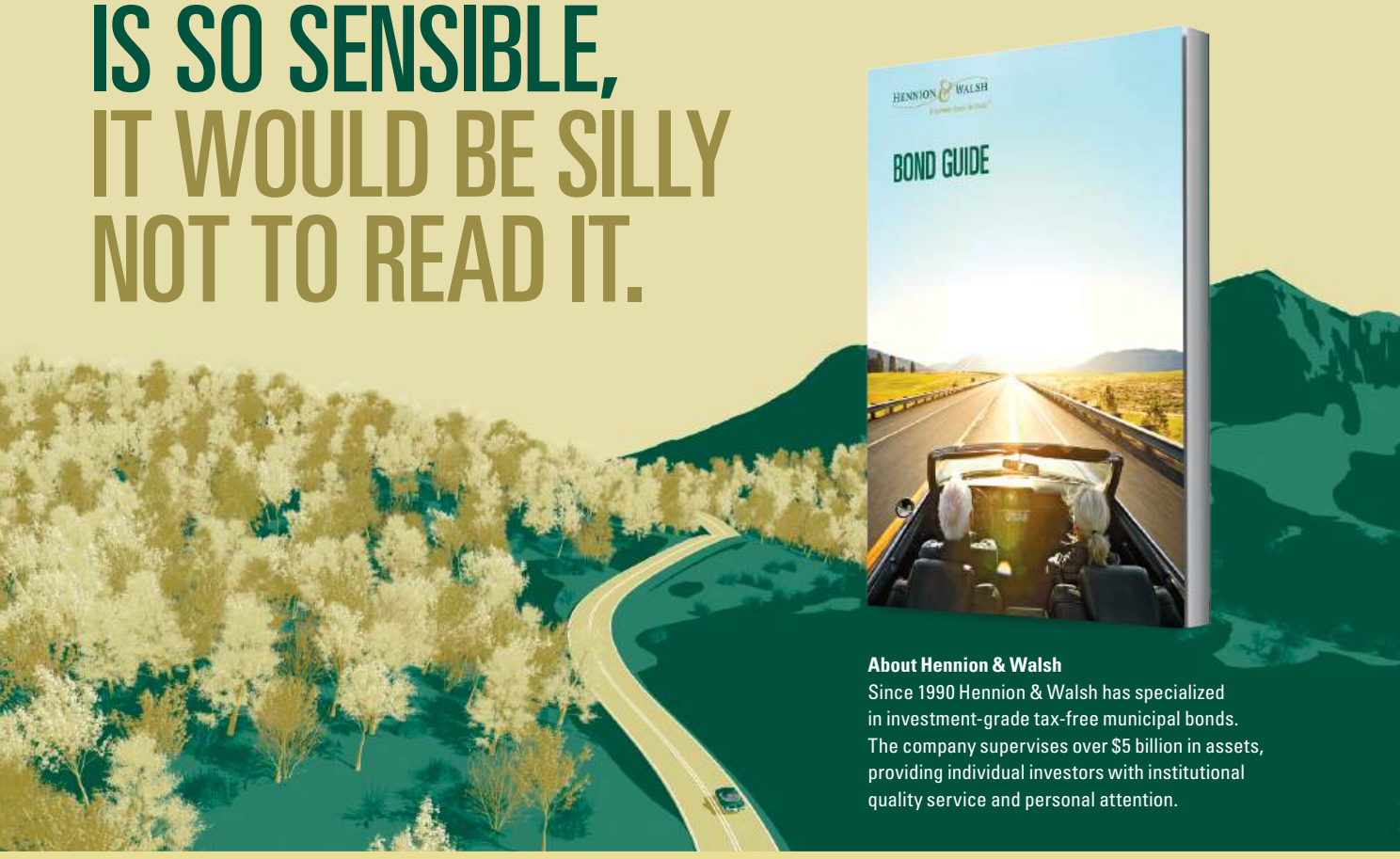

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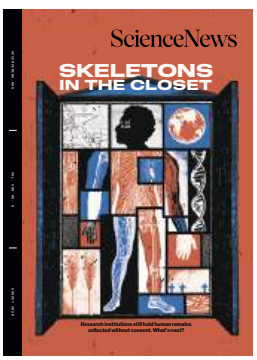
News



On the Cover

Anthropologists grapple with the ethical management of human remains.
ILLUSTRATION BY BEN JONES

YU FANG/CHINESE ACADEMY OF AGRICULTURAL SCIENCES

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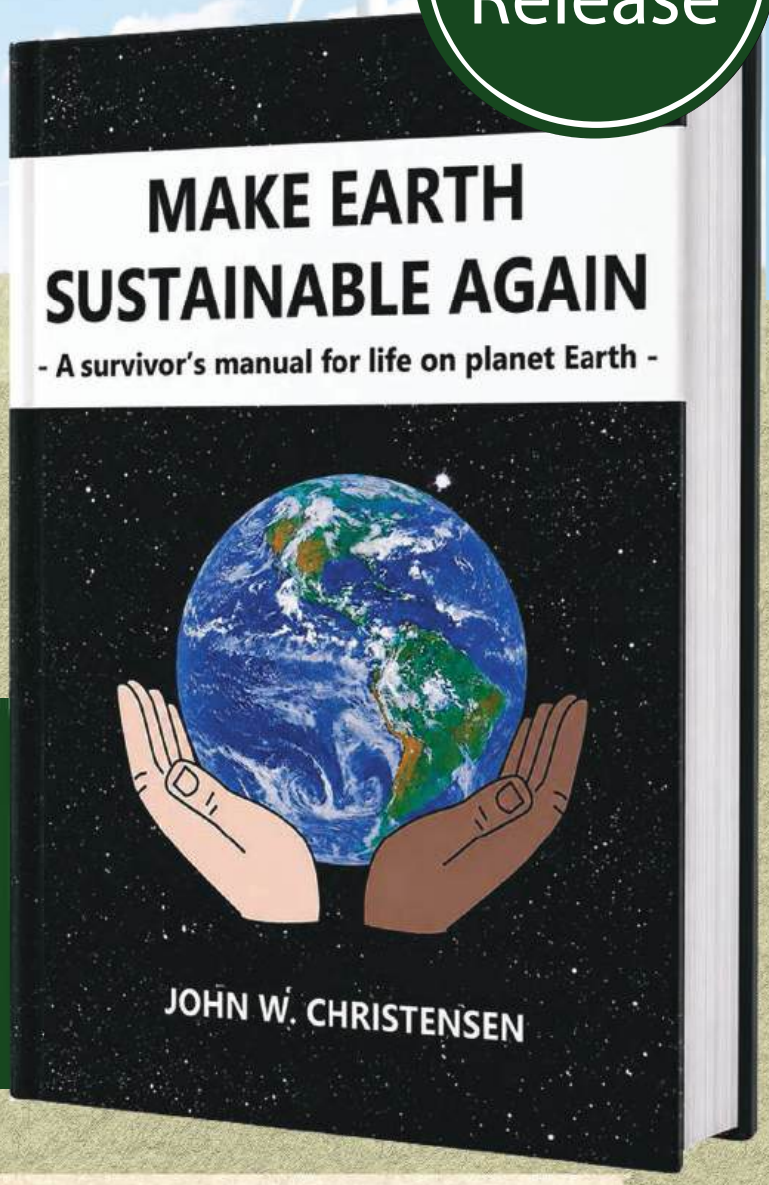

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FROM TOP: NASA; GLENN HARVEY

Who owns your bones?

Scientific research on the workings of the human body has led to major advances in longevity and health. But there have also been dark chapters. The 19th century Viennese physician Joseph Hyrtl became famous for his knowledge of human anatomy — knowledge earned by paying grave robbers to provide subjects for his private collection. He was far from the only researcher at the time to do so.

For centuries, major museums and research institutions have stored, studied and displayed human remains. Some, like Hyrtl's, were almost certainly obtained unethically. The retention of remains has become increasingly controversial as various groups challenge the ethics of using people's bodies as tools of research and public education without consent.

In 1990, Congress passed the Native American Graves Protection and Repatriation Act. It requires institutions to return remains and artifacts to lineal descendants, tribes or other culturally affiliated organizations, or gain consent for their continued use.

Progress has been slow, and controversies over remains held by scientific institutions continue to erupt, extending beyond those from Indigenous communities.

In 2021, journalists in Philadelphia reported that the University of Pennsylvania was holding human remains from the 1985 MOVE bombing, in which city police dropped a bomb on a row house serving as headquarters for a group calling for Black liberation. Eleven people were killed, including children. Some of the MOVE families' remains were used to teach anatomy classes. Although those remains were returned to family members, more have since been found.

Fatimah Jackson, a biological anthropologist recently retired from Howard University in Washington, D.C., is leading a new effort spurred by the MOVE revelations to prevent such abuses (Page 50). "We're the ones who are holding this material," she told freelance contributor Michael Marshall. "The scientists are the culprits here."

Jackson aims to guide universities and museums in ethically managing remains in their possession that were obtained without consent, including those from African American cemeteries.

And there's progress on other fronts. Medical schools now rely on people who choose to donate their bodies after death for anatomy studies. Some schools hold ceremonies honoring donors when the class ends. Hrytl no doubt would have been amazed.

Nancy E. Shute

Nancy Shute
Editor in Chief
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★★★★
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Science News (USPS 484680, ISSN 0036-8423) is published 12 times per year, monthly by the Society for Science & the Public, 1776 Massachusetts Ave. NW, Washington, DC 20036.

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MARINA KOREN

FREELANCE SCIENCE JOURNALIST

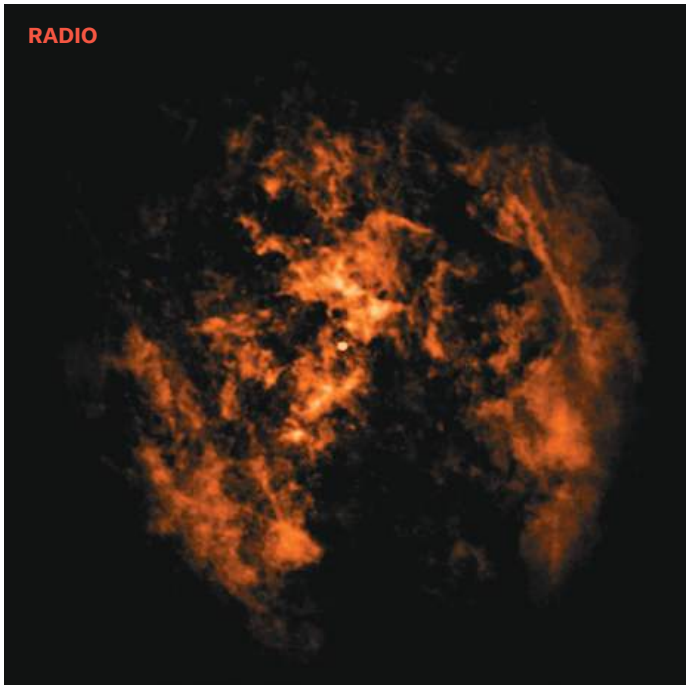
● PLANETARY SCIENTIST Candice Hansen-Koharcheck had been a go-to source for Marina Koren since she began reporting on space in 2016. Back then, Koren barely knew what a planetary scientist did, but Hansen-Koharcheck happily answered the journalist's basic questions without making her feel ignorant. Hansen-Koharcheck spent her decades-long career imaging outer space, so when she died in April, Koren was inspired to commemorate her life with a photo essay (Page 42). "This person was behind so many of the most exciting endeavors in space exploration," Koren says. "I thought people should know about her." Koren's favorite images from Hansen-Koharcheck's portfolio are close-ups of Jupiter taken during NASA's ongoing Juno mission. "Without her pushing for JunoCam to be a part of this mission, we'd have a completely different view of Jupiter," Koren says.

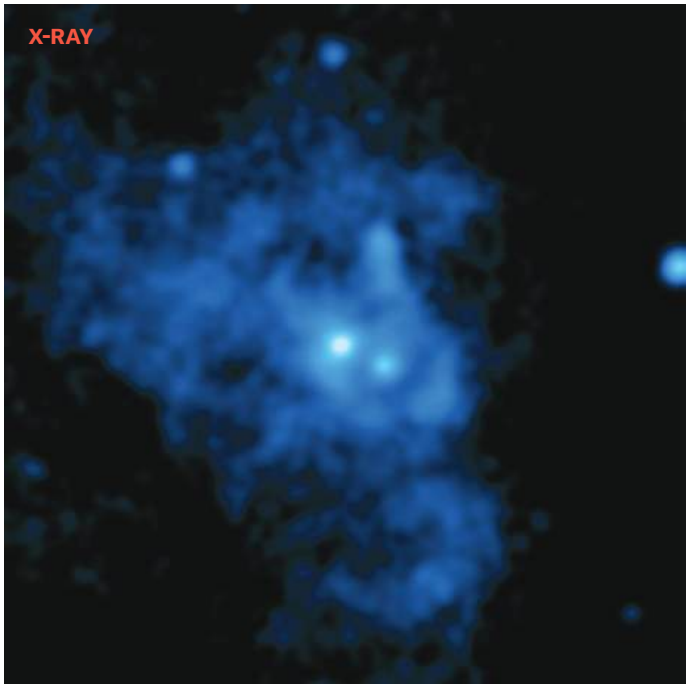
Elie Dolgin
Freelance journalist Elie Dolgin reports on a promising new search for treatments for Huntington's disease, a neurodegenerative disorder that's passed down in families (Page 34). Scientists are targeting a process called somatic expansion, which causes the underlying genetic mutation to get worse over time. Drugs that could halt this expansion, the thinking goes, could stop symptom progression or even onset. If successful, they will "hopefully open new avenues for therapeutic interventions for diseases that work similarly to Huntington's, such as spinocerebellar ataxia and myotonic dystrophy," Dolgin says.

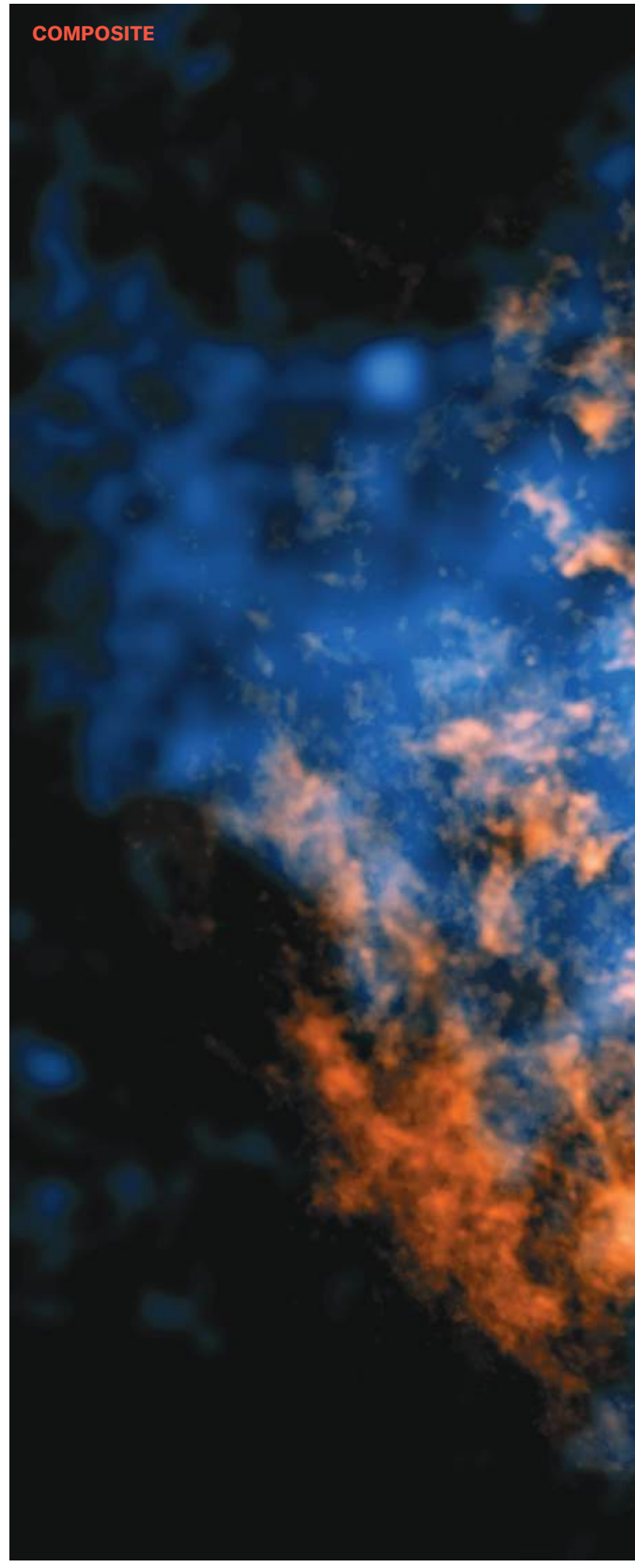
Brody Price
Laying out a feature is an art unto itself. Art director Brody Price takes many things into consideration when designing pages: Which colors convey the right tone for the topic? Which visuals are needed for effective storytelling? Those questions were especially challenging to answer for this issue's profile of Fatimah Jackson due to the sensitive nature of her work. The biological anthropologist developed guidelines for institutions for repatriating human remains obtained without consent (Page 50). "I tried to approach the story with reverence and respect for the individuals behind the remains," Price says.

Fang-Mackey Family
Lilly Fang was planning a birthday party for one of her children in spring 2025 when the software engineer realized something odd. Her kids, herself and then her husband, Lester Mackey, were all turning odd-number ages that year. Now, the family has turned this discovery into a *Science News* math puzzle (Page 64). "Our kids like to find patterns in anything that piques their interest," Fang says. But it took the family a while to figure out the answers to their questions, says Mackey, a computer scientist and alumnus of the Society for Science's prestigious science fairs.

CLOCKWISE FROM TOP LEFT: RONALD CHANG; COURTESY OF E. DOLGIN; THAXTER PRICE; NORA SANDERS







ANATOMY OF A COSMIC WIND
Radio observations (orange) of carbon monoxide near Sagittarius A* (bright dot) show a cone-shaped cavity nearly devoid of the cold gas (white lines, right). The geometry of the void, plus previous X-ray observations (blue) showing hot gas in the same cavity, suggest a hot wind from the black hole swept the cold gas away.

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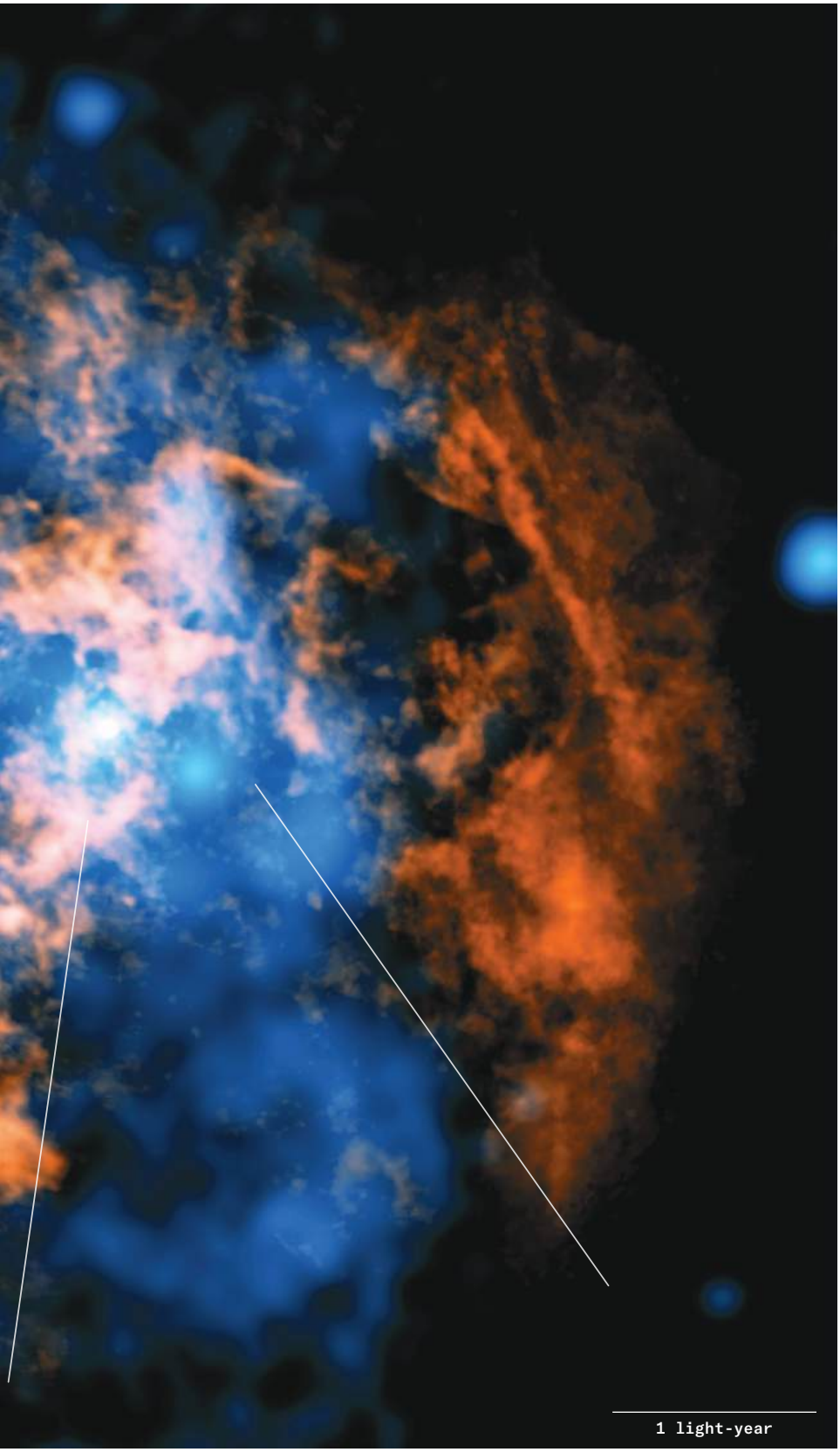

ASTRONOMY

A GENTLE BREEZE FROM A GIANT BLACK HOLE

By Mara Johnson-Groh

● **After more than 50 years** of searching, astronomers have found evidence of a mild wind blowing from the supermassive black hole at the Milky Way's center. New observations reveal a cone-shaped path leading from Sagittarius A* — one that could be carved only by a hot wind, researchers report in the *Astrophysical Journal Letters*.

As black holes consume gas and dust, their munching heats nearby debris. Radiation then pushes material outward, creating wind. Strong winds can slow or trigger star formation around active supermassive black holes. But for most of their lives, black holes are quiet, says Mark Gorski, an astronomer at Northwestern University in Evanston, Ill. The findings show that even quiet ones like Sagittarius A* affect their surroundings. ✖

RADIO: ALMA/ESO, NAOJ, NRAO; X-RAY: CXC/NASA, M. GORSKI/NORTHWESTERN UNIV.; IMAGE PROCESSING: K. ARCAND AND P. EDMONDS/SAO/CXC/NASA

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News

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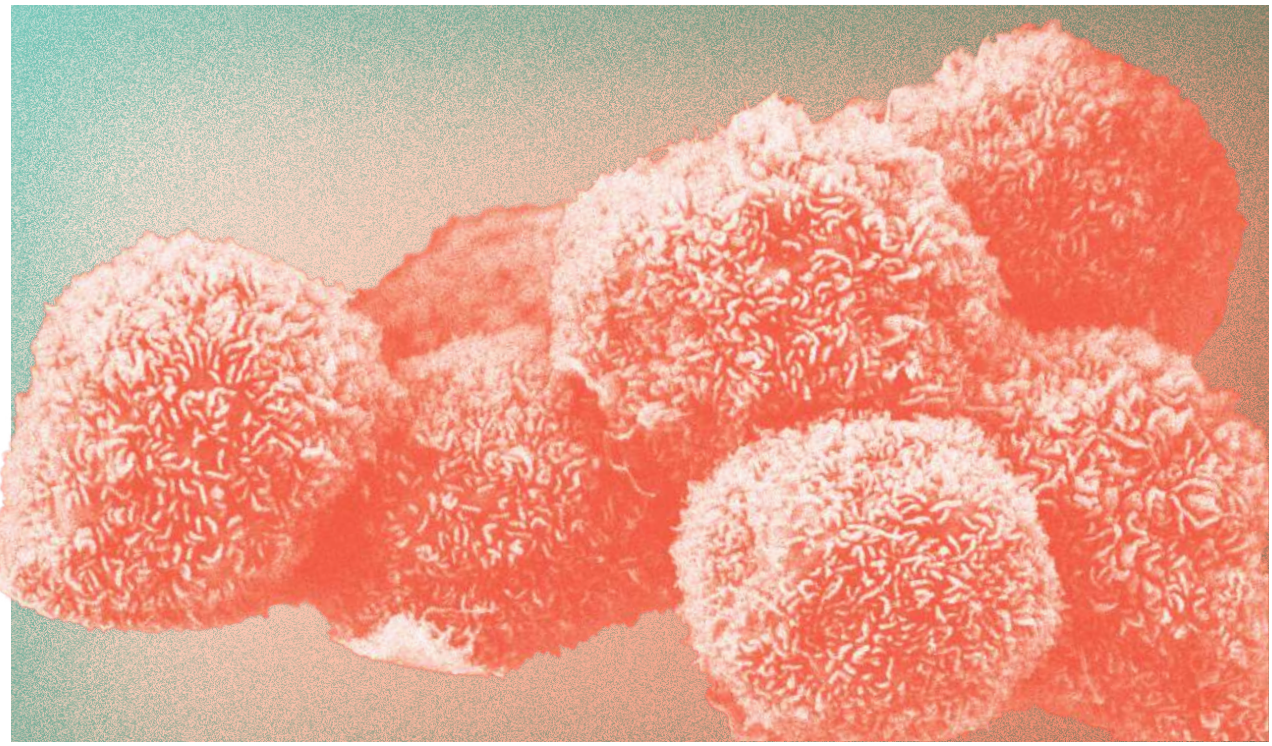
ARCHAEOLOGY

THE ICEMAN HAS A LITTLE LIFE IN HIM YET

● An iconic Copper Age messenger from the Italian Alps may not be perfectly frozen in time. Ötzi the Iceman's 5,300-year-old naturally mummified body (feet shown) harbors four ancient yeasts that scientists were able to grow in a lab. While the glacierlike conditions in which the remains are stored seem to keep many of the yeasts in suspended animation, some species could be active, the team reports in *Microbiome*. The findings could help scientists determine extra interventions needed to keep Ötzi's remains from decomposing. — *Tom Metcalfe*



HEALTH & MEDICINE

A new pancreatic cancer pill could be a game changer

By Meghan Rosen

● **An experimental pancreatic cancer drug** has ignited hope in a field desperate for new treatments.

Compared with traditional chemotherapy, a pill called daraxonrasib nearly doubled the survival time of people with advanced pancreatic cancer, scientists reported to multiple rounds of applause at the annual American Society of Clinical Oncology meeting in Chicago. Half of patients on the new drug lived 13 months or longer after treatment. In comparison, the median survival time was less than seven months for those on chemo.

An extra six months of life might not sound like much, but "it's a drastic improvement," says oncologist Andrew Coveler of the Fred Hutchinson Cancer Center in Seattle. Survival time for people with advanced cancer that's spread beyond the pancreas is often less than a year.

↑ A new pill switches off the "grow" signal for pancreatic cancer cells, shown here in a colorized scanning electron microscopy image.

The new results are "a huge deal for so many reasons," says gastrointestinal medical oncologist Benjamin Musher of Baylor College of Medicine in Houston. Beyond how well daraxonrasib appears to work, it lays the foundation for perhaps even more effective therapies. "This is opening the door to a whole new world of research in pancreatic cancer," he says.

Despite the excitement, Musher and other experts are careful to point out daraxonrasib's limitations. "This is not a panacea," says Anirban Maitra, a pancreatic cancer researcher at NYU Langone Health in New York City. "We have not cured pancreas cancer. I cannot emphasize this enough."

Though pancreatic cancer is relatively rare, with about 60,000 new cases diagnosed in the United States each year, it is infamously

ANNE WESTON/EM STP/THE FRANCIS CRICK INSTITUTE, SCIENCE SOURCE

deadly. Diagnosis often comes late, after the disease has advanced, and only 3 percent of people with advanced cases are still alive five years later. "It's a truly devastating disease, and we haven't had many therapy options," says Shubham Pant, a medical oncologist at the University of Texas MD Anderson Cancer Center in Houston and one of the trial's investigators.

Once the cancer has spread beyond the pancreas and weaseled its way into other tissues, it's usually not possible to operate. An experimental mRNA cancer vaccine currently in the works might one day help, though it's still in the early stages of testing. So doctors rely on chemotherapy, which doesn't extend patients' lives much. The disease is somehow able to resist the medicine's toxic effects.

Pancreatic cancer is commonly fueled by malfunctioning RAS proteins. These proteins typically act like a light switch, toggling on or off to promote cell growth or rest. But in pancreatic cancer, the switch is stuck in the "on" position. "It's always telling the tumor cells to grow," Pant says.

Scientists have been trying for decades to design drugs that target the pushy proteins. "It's been a very hard nut to crack," Maitra says. RAS proteins don't have the kind of molecular grooves that make it easy for drugs to latch on, Coveler says. They're like the smooth surface of a ball bearing, he says.

Daraxonrasib, made by the California-based biotech company Revolution Medicines, skirts the issue by using a different strategy: It brings together other proteins to make a molecular glue. First, the drug sticks to cyclophilin A, which is abundant in cells. That duo gloms onto RAS, blocking the protein's grow command. Imagine the drug and its partner bear-hugging RAS, Maitra says, "like an embrace of death."

In a Phase III clinical trial with 500 patients to evaluate how well the drug worked, researchers compared people given a daily daraxonrasib pill with those given standard intravenous chemotherapy. All patients had advanced disease and had undergone previous therapy. In addition to extending patients' lives, daraxonrasib also helped some patients control their pain, Pant says. He remembers one man who felt so well after treatment that he was able to start golfing again. "He had a great quality of life," Pant says. "He was just out golfing all the time."

Overall, daraxonrasib's side effects tended to be less severe than what patients endured with chemotherapy. During the trial, only about 1 percent of patients stopped taking the drug due to side effects versus 11 percent on chemo, the team reported at the meeting and in the *New England Journal of Medicine*.

Side effects can include diarrhea, nausea, painful mouth inflammation and vomiting, an earlier trial found. In the new trial, the most common severe side effect was rash, occurring in nearly 14 percent of patients.

Researchers had previously shown that roughly 90 percent of patients on daraxonrasib develop some form of rash. It can range "from quite mild to quite fierce," says Musher, who is working on a separate clinical trial that combines daraxonrasib with chemotherapy. The rash typically starts on the face and can flare across the scalp, chest and back. A regimen of creams and pills can help prevent irritation and soothe angry skin, something clinicians will need to learn when the drug gets approved, Musher says. And for him, drug approval is a question of "when" not "if."

"We haven't won the World Series, but it's a home run in a disease where there have been a series of strikeouts."

— Anirban Maitra

Daraxonrasib has not yet been approved by the U.S. Food and Drug Administration, but the FDA is granting access to seriously ill pancreatic cancer patients who have no other treatment options.

Maitra believes the drug will change how cancer doctors practice medicine. Daraxonrasib is "a major home run," he says. "We haven't won the World Series, but it's a home run in a disease where there has been a series of strikeouts."

Medical oncologist Brian Wolpin of Dana-Farber Cancer Institute in Boston said the next step is to make treatment even better. His team is seeking RAS-targeting drugs that provide longer-lasting benefits and potentially a cure.

"Let's all get to work," Wolpin told the crowd. "Let's figure this out." ✖

PHYSICS

GOLD'S PRISTINE STAYING POWER

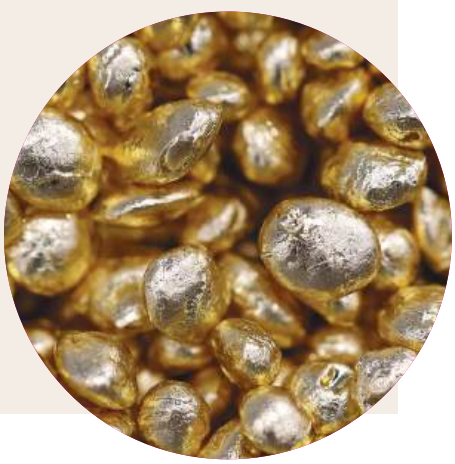
By Emily Conover

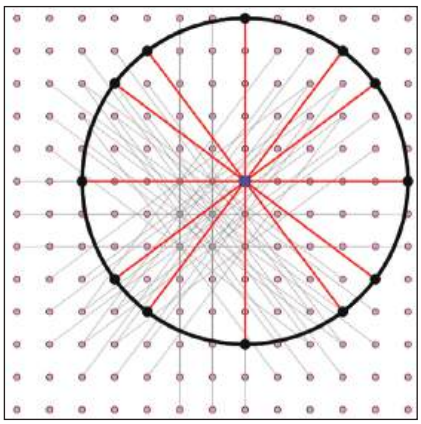
● Steel rusts over time; copper goes green. But gold seems impervious to the elements. Scientists have now discovered a new detail behind how gold stays so pristine.

Atoms on gold's surface rearrange into a geometry that hinders oxidation. Without that rearrangement, gold would begin to tarnish in seconds, scientists report in *Physical Review Letters*.

Metals oxidize when their atoms split the surrounding air's oxygen molecules and those freed oxygen atoms form compounds that stick to the metals' surfaces. So the team calculated how well gold's surfaces could split oxygen.

When a new surface of gold is exposed to air, atoms shift from their original arrangement within their lattice. Different arrangements can occur on gold's exterior, and the team studied two for which atoms laid out in squares reorganize into hexagons. It turns out that the square arrangement is better at splitting oxygen than the hexagonal one. For the hexagonal structure to split oxygen, it would need to distort back into a square, a hurdle that forestalls oxidation. Gold oxide itself is unstable, so even if the square structure could be maintained, only a thin layer of oxide would form. ✖





ARTIFICIAL INTELLIGENCE

AI CRACKS AN ERDŐS MATH PROBLEM, STIRRING DEBATE

BY KATHRYN HULICK

Think about placing dots on a flat surface. You want as many pairs as possible to be separated by the same distance. For any amount of dots, what's the greatest possible number of pairs that can be exactly that far apart?

The question, called the unit distance problem, seems simple. The answer is tricky. Eighty years ago, mathematician Paul Erdős proposed what he thought was the answer, but no one had been able to prove or disprove his conjecture. At least, not until now.

Researchers at OpenAI fed the conjecture to an AI model and walked away. When they returned, they discovered the model had disproved it.

"It's a beautiful piece of math-

↑ Paul Erdős said to maximize pairs of points at the same distance from each other, arrange them on a regular grid so that as many as possible fall onto circles. Adding more points would increase the number of pairs slightly, he thought.

FROM LEFT: BLOOMBERG CREATIVE/GETTY IMAGES; KAI WILLIAMS

ematics," says Melanie Matchett Wood, a mathematician at Harvard University who contributed to a paper in which outside experts reviewed the AI's result. The discovery, posted on OpenAI.com, bolsters hopes that AI can contribute to scientific understanding.

But the AI proof relied on perseverance rather than creative insight and has raised concerns about how mathematics will be done going forward. A group of experts has published a declaration calling for tight guardrails around AI in mathematical research. At the time this magazine went to press, the declaration had more than 2,500 signatures.

OpenAI's model isn't publicly available yet, but the company says it is a general-purpose large language model trained for reasoning. It did not use any math-specific tools or software. And "we didn't guide the model in any particular way," says Sébastien Bubeck, a researcher at OpenAI.

The original prompt, composed by AI, described the conjecture and instructed the model that a complete solution must either prove or disprove it. Mathematicians had long believed the conjecture was true. Yet the model tried to disprove it instead.

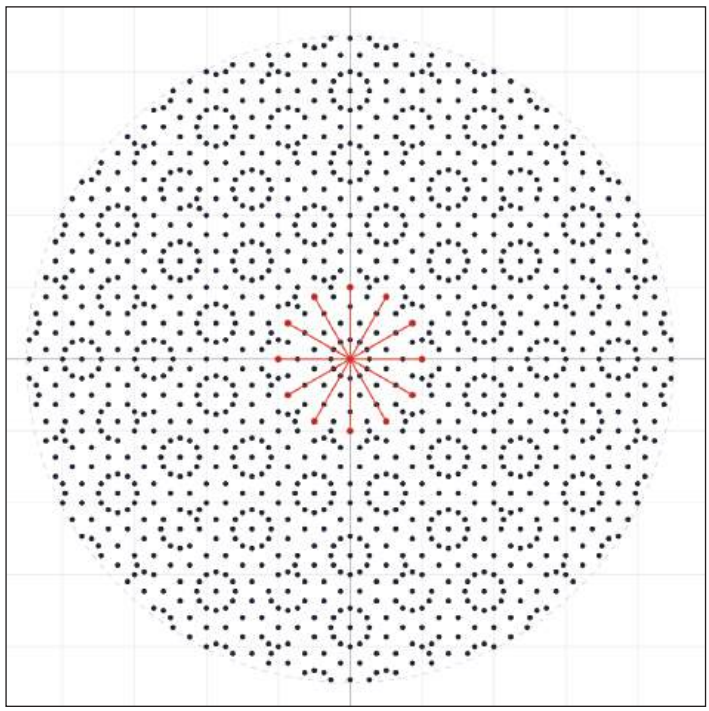
Wood sees the result as a breakthrough in mathematics. The AI came up with a counterexample using tools from two of the oldest and most foundational mathematical fields: algebra and number theory. It seems that these areas shouldn't have anything to do with this geometry question, Wood says. But the result "shows that tools from one part of mathematics can be applied really fruitfully in this other area of mathematics." She thinks this result will inspire mathematicians to think of new ways to apply those same tools.

Wood isn't convinced, however, that this is a breakthrough in AI. When she read the solution, it seemed to her that the latest, publicly available AI models could have come up with it. (In fact, one researcher posted on X that he had reproduced the proof using a publicly available model.)

Mathematician Thomas Bloom of the University of Manchester in England had a similar reaction. He and colleagues noted in the paper from outside experts on the achievement that it would have been "truly incredible" if the AI had managed to prove the conjecture, as that kind of solution would require creative insight.

Bubeck concedes that "this proof isn't exactly the spark of genius that we see sometimes in mathematics." AI still struggles to make leaps of discovery. But the tech can patiently slog through a huge number of unlikely strategies.

Still, the declaration notes that AI threatens our ability to produce responsible, verifiable and ethical mathematics. In this case, the model's proof happened to be relatively easy for a human CONT. ON PAGE 16



A model from OpenAI produced a counterexample to Erdős' grid arrangement, which isn't easy to visualize. It involved building a complicated grid in a high-dimensional space, then projecting it onto a flat plane. But this picture gets at the gist of the idea. It shows an arrangement of points constructed in a similar way. ↓

KAI WILLIAMS/CHATGPT, BASED ON AN IDEA BY WILL SAWIN

CONT. FROM PAGE 15 expert to verify, Bloom says. But he has seen people on the internet who have used AI to generate hundreds of pages of math that they can't understand or even read. "It could be right. It could be nonsense. Who's going to be able to check?" he says.

If mathematicians knew the probability that an AI-generated proof was correct, that would help. But OpenAI does not share all the times its model failed to solve an open problem in math or, even worse, produced an incorrect solution.

Bubeck says the team ran the Erdős prompt through the same model multiple times, and the AI produced the correct solution half of the time. His colleague Lijie Chen says that the new model is better than current models at generating an "I cannot solve it" response when it runs into difficulty. But data to support these claims have not been released or peer-reviewed. And OpenAI will not reveal how much time the model spent working on its solution.

Those who signed the declaration have other concerns. Right now, AI generates mathematical reasoning without showing what work inspired the ideas. That clashes with mathematicians' standard practice of giving credit where it's due. "It's not clear that there's a way for it to reasonably attribute the source of the ideas," Wood says.

Access is another concern. If the most powerful tools are expensive and private, mathematics could become less democratic and some people may question why they should learn it at all, Bloom says.

Still, he, Wood and some other mathematicians are cautiously optimistic. AI, Wood says, "is going to become an indispensable tool in mathematics." ✖

ANIMALS

Royal jelly isn't the only thing that makes a queen bee

By Anirban Mukhopadhyay

● **A queen bee** may be shaped by more than her famous royal diet.

The wax of the peanut-shaped chamber where the queen develops has distinct physical and chemical properties that help steer a queen's development, researchers report in *Nature*. By analyzing the composition of the chamber and the larvae it harbors, the team challenges the long-held notion that royal jelly alone — fed to select larvae — makes a queen.

"The discovery is very cool and thought-provoking," says biologist Thomas Seeley of Cornell University. "To me, queen cells have long seemed important because odors from a developing queen may permeate the wax walls, marking them as very special spots that workers recognize and don't accidentally damage."

But for entomologist Boris Baer, the cells posed a question born of years observing his own colonies. "Bees spend so much time and energy constructing these cells that it made little evolutionary sense if they were merely larger food containers," says Baer, of the University of California, Riverside. "Could the cell itself contribute to queen development?"

Baer and colleagues studied the western honeybee (*Apis mellifera*) and eastern honeybee (*Apis cerana*), comparing queen- and worker-cell wax, the workers that build the cells and how larvae fare in each environment. CONT. ON PAGE 18

↑ **Worker bees cling to the wax-covered cell of their future queen in this artistic rendering.**

YU FANG/CHINESE ACADEMY OF AGRICULTURAL SCIENCES

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CONT. FROM PAGE 16 Queen-cell wax is softer, less dense and chemically distinct from worker-cell wax. Compared with bees that build worker cells, the bees that build royal cells "spend longer constructing these cells, run hotter than other bees and show distinct patterns of gene [activity]," Baer says. That suggests the bees that build royal cells are specially adapted to modify the wax they work with.

The team let queens-to-be develop on royal jelly for four days in artificial cells, then replaced the wax caps on the cells with wax from either natural queen or worker cells. Up to about two-thirds of larvae under worker-cell wax died, compared with roughly a third under queen-cell wax. The worker-cell larvae also developed into smaller pupae, while those reared under queen-cell wax more closely resembled bees left undisturbed in their natural cells.

Worker bees do more than feed queens, Baer says; they actively engineer them. "These superorganisms mobilize specialized workers that collectively shape the next generation," he says. "The division of labor in bees might be much more complex than we have acknowledged."

Exactly how that engineering works remains unclear. Apiologist Kai Wang says the distinct chemical scents that he, Baer and colleagues found inside the cells are especially intriguing. "Are they influencing the developing queen's senses, preparing her for mating and life after emergence?" asks Wang, of the Chinese Academy of Agricultural Sciences in Beijing. "Are some produced by the larva herself? And could the future queen be actively communicating with the workers constructing her chamber?" The team plans to trace when during development the wax environment exerts its effects. ✖

↗ This hailstone, collected in North Dakota in 2025, is about as wide as a half dollar.

Q. ZHANG/PEKING UNIV.

CLIMATE

GLOBAL WARMING MAY BE SUPERSIZING HAIL

BY YUJIA HUANG

On April 28, a fierce hailstorm battered Springfield, Mo., dropping ice chunks the size of baseballs, with some even larger than grapefruits. The giant hail smashed cars, wrecked homes, injured people and even killed an emu.

In a warming world, ice falling from the sky might seem more likely to melt away before it hits the earth. But hailstones may instead grow larger and more destructive in many parts of the world, researchers report in *Nature*.

"The study provides an interesting and timely contribution to understanding how climate change may affect hail hazards," says climatologist Davide Faranda of the French National Center for Scientific Research in

Paris. “It combines physical reasoning with climate model projections.”

Hail forms when strong storm winds lift moisture high into cold clouds. There, water droplets freeze around tiny particles and grow until they become too heavy for the winds to hold up.

To see how that process may change in a warmer world, researchers in China and the United States built a computer simulation of how hailstones grow inside clouds based on atmospheric conditions, such as temperature, moisture and wind. The team trained the simulation on data from more than 14,000 real-world hailstorms around the globe from 2014 to 2021, then estimated how those storms might change under future climate conditions.

Large hailstones are expected to become more common, making hailstorms more damaging, the simulation suggests. That pattern reflects two competing effects. Warmer air can hold more water vapor, giving hailstones more material to grow. But that same warm air can also melt small hailstones before they hit the ground.

“Large hailstones melt too, but they can still reach the ground as sizable chunks of ice,” says study coauthor Qinghong Zhang, a meteorologist at Peking University in Beijing. “Smaller hailstones are affected more. They may melt completely and turn into raindrops.”

The danger isn’t equal everywhere; risks will vary by region. Places farther from the equator could get hit harder, while hail damage in tropical and subtropical regions may actually ease, the team found. That’s partly because by the end of this century, temperatures are expected to rise more sharply at higher latitudes. The extra warming can also strengthen updrafts inside storm clouds, allowing hailstones to grow larger.

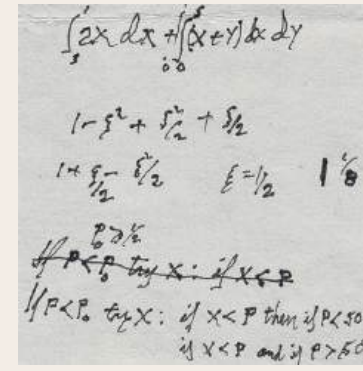
“This is the first study to make a quantitative estimate of hail hazard events worldwide,” Zhang says.

The broad conclusion is plausible and fits with earlier work, Faranda says. But he is less certain about the quantitative results and regional forecasts.

“Hail is an extremely local phenomenon,” Faranda says. “Global climate models cannot explicitly resolve hailstorms.” That means studies based on broader weather patterns still come with uncertainty.

Zhang acknowledges those uncertainties. Still, she says, the team successfully tested its results against hailstorms recorded over the last several decades in China and the United States. Those checks suggest the uncertainties are manageable.

For now, the study offers a clear warning: If temperatures keep rising, larger and more damaging hail will probably become a greater threat in many regions. ✖



RICHARD P. FEYNMAN ESTATE, COURTESY OF RALPH LEIGHTON

MATH

FEYNMAN’S FORMULA FOR EATING WELL

By Emily Conover

● A famous physicist’s scribbles reveal the answer to a common dilemma: When dining out, is it better to stick with an old favorite or try something new?

The late Richard Feynman pondered this question with a lunch companion in the 1970s. He turned it into a math problem and solved it on the spot, in his inscrutable handwriting. The solution remained undeciphered until scientists recognized in the scrawl a solution to a class of mathematical questions, which allowed them to make sense of the rest of the scribbles. Feynman found the optimal solution, the team reports in the *Proceedings of the National Academy of Sciences*.

Start by giving a score to a dish or restaurant to indicate quality. The aim: Maximize the score over a set number of nights. Feynman found an equation for a threshold against which to compare the best dish or place you’ve tried so far. Each night, check your favorite against it. If your fave scores above the threshold, return to it for the remaining nights; if not, try something new. The threshold isn’t fixed. It starts high and falls over time. Early on, it pays to hold out for something extraordinary. By the last night, you have little to gain from searching, so you should settle for anything better than average. ✖

HAYLEY WALL

NEUROSCIENCE

Measures of age-related memory loss may be flawed

By Diana Kwon

● **One of the major cognitive changes** that happens as we age is difficulty recounting details of past experiences. But two recent studies suggest that older adults' memories may be richer than previously thought.

The notion that describing previous experiences becomes more difficult with age was built on decades of lab research, says psychologist Matthew Grilli of the University of Arizona in Tucson. Lab-based studies of autobiographical memories, which typically involved experimenters interviewing people about past events, have shown that older adults can recall fewer details than younger ones. Researchers have found this effect to be more pronounced in people with dementia, leading many to view this shift as a sign of cognitive decline.

Grilli and colleagues wanted to test if the trend held up outside the lab. The researchers asked 24 adults aged 18 to 28 and 50 adults aged 61 to 81 to wear a smartphone with an app that randomly recorded 30 seconds of audio five times per hour for 14 hours a day. After 10 continuous days of recording, the team sifted through the files for moments when people shared autobiographical memories. Participants also came into the lab for interview-based assessments.

In everyday conversations, older adults showed no significant difference in their ability to recount details compared with their younger counterparts, the team reports in the *Journal of Experimental Psychology: General*. The findings suggest that researchers "may need to take a step back from the assumptions that we make about how autobiographical memories may or may not change in older age based on laboratory research, which might not be capturing the whole picture," Grilli says.

These results are supported by another study from Grilli's team published in the *Proceedings of the National Academy of Science*. The researchers assessed autobiographical memory recall in more than 3,800 adults aged 18 to 89 using a different smartphone-based technique. Participants were pinged throughout the day and prompted to share their thoughts. Older adults reported their past thoughts with more specificity and vividness than their younger counterparts.

Researchers aren't sure why these differences between the laboratory and everyday life appear to only show up in older adults. Cognitive neuroscientist Jessica Andrews-Hanna, a coauthor of both studies, says one reason may be that the lab

setting is less familiar to older participants than to younger ones, who are often students on the campus where this type of work is carried out. On top of that, experimenters are often young adults themselves, which means older adults may feel the need to give more context before describing their memories.

It's still unclear whether the participants' memories were accurate, says Andrews-Hanna, also of the University of Arizona. Perhaps the ways that people experience and share memories are more sensitive markers of disease-related cognitive decline than their accuracy.

"We need more studies on aging that go outside the laboratory and look at memory and related function in a more naturalistic context," says Harvard psychologist Daniel Schacter. Factors such as a person's narrative style can also explain differences in autobiographical recall reported in the lab.

Brian Levine, a neuropsychologist at Baycrest Health Sciences hospital in Toronto, emphasizes that aging indeed affects memory. But the way older adults use their memories may differ from younger adults, he says. "When we bring them into the lab and test them on things that are not optimized to their needs, then they're going to look more impaired than they really are." ✖

"We need more studies on aging that go outside the laboratory and look at memory ... in a more naturalistic context."

—Daniel Schacter

PARTICLE PHYSICS

HERE'S WHAT WOULD HAPPEN IF YOU BROKE OPEN A PHOTON

BY MARIA TEMMING

If you think breaking a Nature Valley granola bar makes a mess, try a photon.

As a fundamental particle, the photon can't really be cleaved into smaller pieces. But theoretically, if you tried to crack one in half, an infinite number of new photons would crumble out, researchers report in *Physical Review Letters*.

Physicist Daniele Faccio's first-glance reaction to that conclusion was: nonsense. Then he read the study. "The technique is legit," says Faccio, of the University of Glasgow in Scotland.

The analysis hinges on the fact that photons are both pointlike particles of light and extended waves. That got Johannes Skaar and his colleagues at the University of Oslo in Norway wondering: What would happen if you had a device fast enough to snip the wave of a single photon in half?

Using quantum equations, the team modeled a scenario in which a photon is traveling toward a mirror. The front half of the light wave hits the mirror first and bounces back in the direction it came from. But if the mirror is suddenly removed, the back half of the light wave is free to pass through.

This would spew out a complex mix, or superposition, of possibilities with different numbers of photons. Removing the mirror infinitely fast would conjure infinite photons out of thin air. Infinite speed is of course impossible. But even pulling the mirror away more slowly, Skaar says, "you end up with a possibility of several photons, or a bunch of photons." You're just much more likely to create small groups than huge swarms.

By quantum standards, the result is not that weird, Skaar says. Physicists knew that disturbing supposedly empty space could knock new photons loose. In this case, the energy fed into the system by moving the mirror could spawn new light particles. To Skaar, the oddest outcome is what you'd see if you observed the system from different perspectives. If you could see both sides of the mirror at once, you'd witness the messy eruption of up to infinite photons. But if you could see only one side of the mirror, you'd see either a single photon or a vacuum, depending on the side. Skaar hopes to probe that difference more deeply in future work. ✖

PLANETARY SCIENCE

HOW DID NEPTUNE GET ITS ODDBALL MOON?

By Lisa Grossman

● Neptune's moon Nereid may be the sole survivor of an ancient moonpocalypse.

Billions of years ago, Neptune captured its largest moon, Triton, from the Kuiper Belt in the outer solar system. That cosmic wrecking ball pulverized most of the planet's original moons and banished Nereid to the margins, Caltech planetary scientist Matthew Belyakov and colleagues argue in *Science Advances*. This idea counters the long-held assumption that Nereid formed in the Kuiper Belt and was later pitched into its present orbit.

The team analyzed data from the James Webb Space Telescope and found that Nereid's makeup doesn't match well with those of any similar objects in the Kuiper Belt nor with any of its sibling moons — smaller, rubbly bodies that orbit a lot closer to Neptune and that seem to have been through a lot of collisions.

"Nereid always is an outlier," Belyakov says. Maybe the moon's origin story is an outlier, too.

Computer simulations of Triton's chaotic arrival at Neptune didn't reproduce Nereid's exact present-day orbit. But about 20 percent of them produced a close-enough outcome to make the hypothesis plausible, Belyakov says. ✖

NASA, ESA, CSA, STSCI; IMAGE PROCESSING: JOSEPH DEPASQUALE/STSCI, NAOMI ROWE-GURNEY/NASA-GSFC

PALEONTOLOGY

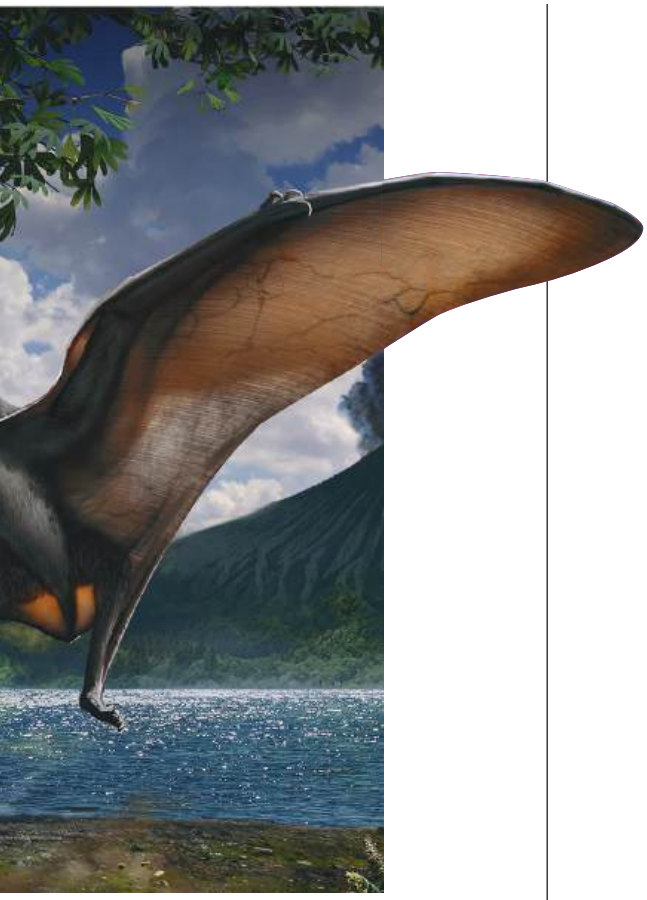
Some pterosaurs may have boasted bold iridescence

By Taylor Mitchell Brown

● **Fossilized filaments** are giving Earth's first flying vertebrates a shiny new makeover.

At least one species of pterosaur shimmered in iridescent greens and magentas, scientists report in a paper posted on bioRxiv.org. The discovery reshapes what we know about the formidable flying reptiles, hinting at heightened metabolisms and hidden courtship displays.

"This is one of the most intriguing and surprising fossil



COURTESY OF ZHAO CHUANG

↖ Artists have long imagined pterosaurs in vivid hues. A new fossil analysis suggests at least one species really did shimmer in iridescent greens and magentas.

discoveries of the past few years," says paleontologist Steve Brusatte of the University of Edinburgh, who was not involved in the work.

The study focuses on a previously unexamined specimen of *Sinopterus dongi*, a small pterosaur with a wingspan of up to nearly 2 meters. The 120-million-year-old fossil, unearthed in Northeast China, preserves soft tissue and offers an unusual glimpse into how pterosaurs might have looked.

"Soft tissue preservation at this level of fidelity is incredibly rare," says paleontologist David Martill of the University of Portsmouth in England, who was not involved in the work.

Previous research had found that pterosaurs' pycnofibers — small filaments similar to dinosaur protofeathers — had pigment-containing structures called melanosomes. That led paleontologists to envision pterosaurs with colorful patterns on their crests and other body parts. But the finding that the reptiles might be iridescent is new.

Iridescence occurs when an object reflects different colors depending on the viewing angle, creating a kaleidoscopic spectacle. It has evolved many times in the natural world — in insects, birds and even some plants and slime molds. In many cases, the shimmer comes from layered structures that scatter light and split it into myriad colors.

An international team of scientists scrutinized the fossil using scanning electron microscopy and other techniques. The pycnofibers contained ordered layers of melanosomes. These layers resemble the melanosome-bearing structures that produce iridescence in modern bird feathers.

The diversity of melanosomes within these fibers matches that found in warm-blooded birds and mammals. That similarity suggests these pterosaurs may also have had high metabolisms and complex mechanisms to regulate their own body temperature, Brusatte says.

Using computer simulations, the researchers determined the fibers probably produced deep greens and magentas.

These are "the same colors that you find in pigeons, starlings and a whole host of other birds," Martill says. "It will really give the paleoartists something to go on ... but it also means that we have to go back and analyze other things," such as soft tissues preserved in fossil feathers and dinosaur skin.

In birds, iridescent plumage is widely known for its role in courtship rituals. Birds will dance in idiosyncratic ways, parading around their shiny plumage in an attempt to entice mates. The discovery of iridescence in pterosaurs suggests the reptiles may have done something similar.

"We often think that simple protofeathers in dinosaurs evolved for insulation, like hair did in mammals," Brusatte says. "Now we must consider the possibility that even the simplest dinosaur feathers arose as display structures." ✖

ENVIRONMENT

EVEN CAREFUL SCUBA DIVERS CAN DAMAGE CORAL REEFS

BY JAKE BUEHLER

Scuba divers may be battering coral reefs more than they think. Video analyses of hundreds of divers show that more than 80 percent of damaging physical contact with reefs is unintended or simply unnoticed, researchers report in *Conservation Letters*. The findings show that even routine diving practices aren't harmless.

Scuba diving is often framed as one of the "good" ways to use reefs because it isn't extractive, says marine conservationist Bing Lin of the University of Sydney. Fish aren't torn from their homes and divers get to enjoy seeing them in the wild.

However, divers can damage reefs by kicking or bumping corals or by disturbing wildlife. "What's less understood is just how invisible much of this damage is to the people causing the harm," Lin says.

Lin's team compared what divers thought they were doing underwater with what they were actually doing. From December 2022 to January 2024, the team collected survey and video data from 732 divers at sites across Indonesia and the Philippines. The researchers filmed divers on coral reefs, logging their behaviors when they touched or damaged a reef. The team also asked divers to estimate how often they thought they had contacted a reef.

A scuba diver accidentallly kicks up sediment at a coral reef restoration site in Bali, Indonesia. ↙

B. LIN

Divers touched reefs an average of about once every four minutes. About 60 percent of the touches were unintentional. "Reef damage was pervasive, but usually not malicious," Lin says.

Overconfidence and a lack of situational awareness seem to be the driving factors. About 75 percent of divers rated themselves above average in diving ability and reef avoidance — all while touching reefs five times as much as they had estimated. Wildlife sightings made things worse, more than doubling the rate of damaging contact.

On some popular reefs with thousands of divers and snorkelers per day, the accumulating damage could have "substantial ecological impacts," Lin says.

Marine ecologist Fabio Favoretto of the University of Plymouth in England notes that about 15 percent of divers in the study never touched a reef at all. "That's the proof that this is fundamentally a fixable problem by training and regulation," Favoretto says. An important next step is to see whether diver behavior is linked to measurable effects on reef health, he says. What actually happens to a reef over five or 10 years if it's touched once every four minutes during dives?

Favoretto and Lin both say that hanging up one's fins isn't a solution. Tourism plays an important role in reef conservation. "Ultimately, the goal is not to stop people from diving, but helping people dive better," Lin says. ✖

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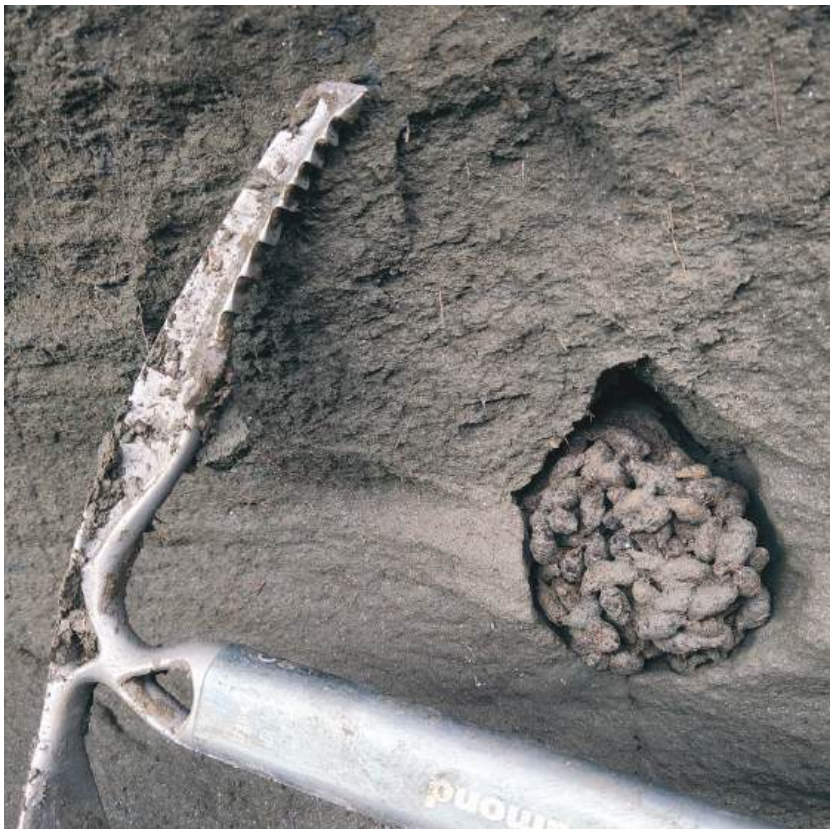

ANIMALS

Frozen squirrel poop reveals deep secrets of the Ice Age

By Bethany Brookshire

↑ This small pile of squirrel poop, frozen in Yukon permafrost for thousands of years, contains DNA from hundreds of species that lived in Ice Age ecosystems.

SCOTT COCKER

● **Ancient squirrel poo doesn't stink.** At least not at first.

But that changes when you begin to break down the pellets. Melting them makes it clear that they are not mineralized, stony fossils, says biomolecular archaeologist Tyler Murchie of the Hakai Institute in Canada. "There's no mistaking," he says. "This is a very poopy-smelling lab."

The fresh fecal smell is a sign of science. The pellets, collected from gold mining sites in the Yukon Territory in Canada, contain fragments of DNA from the squirrels' diet that paint a picture of the animals' ecosystems in new detail, Murchie and colleagues report in *Nature Communications*.

The fecal pellets are some of the many ancient remains found during mining operations, Murchie says. As miners melt the permafrost, they expose gold as well as precious bones and naturally preserved remains of ancient creatures.

There are mammoth bones and steppe bison bones, Murchie says, and as work progresses, eventually "you see these large exposures on the valley walls, there's all these little pockets." Those holes are the remains of burrows dug by the ground squirrel (*Urocitellus*). The burrows are literally frozen in time. "They have little latrine areas and tunnel networks and caches of food," Murchie says.

Ancient ground squirrels are wonderful paleontology assistants. "They really function as little naturalists or archivists, going on the landscape like pack rats, gathering up all these bits of plant material, seeds, leaves, twigs, et cetera," Murchie says. But while scientists had carefully cataloged the contents of the burrows, no one had looked at the frozen poop the squirrels left behind.

The researchers plugged their noses, thawed the pellets and extracted DNA from 13 squirrel coprolites from the Yukon. The oldest poop turned out to be almost 700,000 years old, with others ranging from 80,000 to 17,000 years old. That places the squirrels in the Pleistocene epoch.

Scientists usually assume that Pleistocene squirrels were the same type of squirrels present in the Yukon today. But "there's been a population turnover of some sort," Murchie says. The oldest sample also could be from a new species. "It sits in its own evolutionary branch," he says. "The closest thing to it is squirrels that are in China now."

The poop held other surprises, too. "I thought the DNA would mostly just be the squirrel plus their gut microbiome," Murchie says. But there were fragments from the squirrels' diet, too.

Ground squirrels are not picky eaters, and genetic material popped up from grasses, willows, beetles, grasshoppers, woolly mammoths, steppe bison, wolves, ancient horses and many other species.

There were probably not packs of ground squirrels ravenously chasing down mammoths, says Jacquelyn Gill, a paleoecologist at the University of Maine in Orono who was not involved in the study. Instead, the squirrels were probably scavenging.

"It could have been a component of the diet," Gill says. "They could have been chewing on bone for [the] calcium."

Rodents today frequently do the same. "I remember watching one on my fence eating a bone," Murchie says. "It was definitely a different perspective on them after that." Their omnivorous tendencies allowed Murchie's team to reconstruct the mitochondrial genomes of 23 animals: 12 squirrels, two bison, three horses and six mammoths.

The other species in the scat offer a detailed picture of the ecosystem, Gill says. "A mammoth bone will tell you a mammoth was here," she says. "These ground squirrel coprolites are telling you we had a ground squirrel here eating these plants, living among these insects, sharing the landscape."

And while it's easy to want to study a mammoth tusk, it's a little harder to see the promise in poop, she says. "I think you have to have a little bit of imagination and maybe a little humility." ✖

A. SALAMA *ET AL*/*SCIENTIFIC REPORTS* 2026

↗ A scientist measures vibrations inside a chamber in the Great Pyramid of Giza.

ARCHAEOLOGY

THE GREAT PYRAMID SURVIVES EARTHQUAKES ON GOOD VIBES

BY SKYLER WARE

Egypt's Great Pyramid of Giza has resisted earthquake damage for millennia, and scientists finally know why.

The pyramid tends to vibrate at a different frequency than the surrounding soil, which prevents excessive shaking during an earthquake. Combined with the structure's sturdy shape and mass-distributing internal design, this difference has kept the pyramid intact, researchers write in *Scientific Reports*.

"It's no surprise that they are very seismically resistant," given the pyramids' long history, says civil engineer Sherif El-Tawil of the University of Michigan in Ann Arbor. But the new work offers important insight into why the pyramids are so resistant to seismic damage, he says.

The Great Pyramid, the last of the Seven Wonders of the Ancient World still standing, was built in Giza around 2600 B.C. to serve as the tomb of the Pharaoh Khufu. It contains some 2.3 million blocks and took over two decades to construct.

Though Egypt generally has low seismic activity, it does occasionally experience strong earthquakes. Over the years, the Great Pyramid has suffered only minimal damage.

To uncover the origins of this enduring stability, geophysicist Mohamed ElGabry and colleagues monitored subtle vibrations at 37 points within and around CONT. ON PAGE 28

CONT. FROM PAGE 27 the pyramid. Intentionally shaking it could be damaging, so the team instead relied on tiny disturbances caused by far-off ocean waves, traffic or other human activities to set the structure vibrating.

At about three-quarters of the measurement sites inside the pyramid, the structure naturally vibrated back and forth at frequencies between 2 and 2.6 times per second. The narrow distribution of frequencies indicates that stress is evenly distributed throughout the pyramid. The surrounding soil vibrated more slowly, oscillating about once every two seconds.

Because the natural frequencies of the building materials and the soil are different, the pyramid is less likely to absorb energy from the soil's vibrations, decreasing the risk of damage during a quake.

Spaces inside the pyramid also help mitigate shaking. Ancient Egyptian builders constructed pressure-relieving chambers above the king's chamber, where the pharaoh was entombed, to distribute the pyramid's weight and protect the burial chamber in the event of a collapse. Those chambers also decreased the strength of the vibrations at points higher up in the pyramid.

"All of this is really amazing to look at from today's engineering point of view," says ElGabry, of Egypt's National Research Institute of Astronomy and Geophysics in Cairo. "But it's more amazing and more impressive when you look into the tools and available resources we had 4,600 years [ago]."

However, the findings can't confirm whether ancient Egyptians intentionally designed the pyramids with earthquakes in mind, the researchers say. ✖

ANGELA WEISS/AFP/GETTY IMAGES

↑ Chemists have devised a way to detect new illicit fentanyls that aren't yet on official watch lists.

CHEMISTRY

A new method could ID emerging fentanyl varieties

By Rachel Berkowitz

● **Underground laboratories** do a criminally good job of synthesizing new forms of dangerous drugs that slip under the radar. But a new method could spot variants of the powerful opioid drug fentanyl even before they've been cataloged by authorities.

Measuring a suspicious pill's traits and comparing them to computer-generated reference values could reveal chemical signatures of previously unseen fentanyl variants, researchers report in a paper posted at bioRxiv.org. Although the system isn't yet ready for use by law enforcement officials, the ability to look beyond molecules on the official books could one day help them stay a step ahead of opioid traffickers.

Used in anesthesia and pain medication, fentanyl is up to 100 times as potent as its cousin morphine. Just two milligrams, an amount that fits on the tip of a pencil, is potentially lethal. In the United States, there were more than

72,000 overdose deaths in 2023 alone.

Fentanyl contains no natural ingredients, and underground labs can tweak its structure just enough to avoid detection while retaining the drug's heroin-like effect. That's made it profitable to lace pills with this powerful substance that many users don't know they're consuming.

The only way experts can know if a pill contains a fentanyl variant is by comparing it to a reference library made by analyzing pure chemical compounds in the lab. But calculations indicate that there are more than a billion possible forms of fentanyl; experts know of only about 60,000. Forensic and toxicology labs can't keep up.

"It's become a whack-a-mole problem," says biochemist David Wishart of the University of Alberta in Edmonton, Canada, who was not involved in the study.

Bioanalytical chemist Tom Metz and colleagues set out to eliminate the need for traditional reference libraries. In prior work, they used two customized instruments to identify chemical features shared by fentanyl compounds and to distinguish between many unrelated molecules that share fentanyl's molecular mass.

All fentanyls have a common core chemistry, but labs can vary surrounding chemical groups. "It's like a Christmas tree — nearly always a [conifer] of some sort, but each household will decorate it differently," says Metz, of Pacific Northwest National Laboratory in Richland, Wash. The instruments give clues to the precise elements that make up molecules, how they're structured and the shape they adopt during analysis.

Now that the researchers knew which measurements would thoroughly profile fentanyls, they dreamed up a database of hypothetical variants. They computationally broke apart each of the roughly 60,000 known fentanyl and fentanyl-like molecules into a few different fragments, then recombined those fragments to create over 1 trillion molecules.

The team eliminated nonsensical and implausible molecules from the digital catalog, such as ones unlikely to penetrate the brain's protective barrier. Finally, with help from machine learning, the researchers predicted what real-world chemical measurements of the dreamed-up structures would look like. They combined the data with that of the 60,000 known structures to make their final digital library of over 1 billion analogs.

Since the researchers could not test street drugs, they created a mock fentanyl pill with traces of 12 commercially available varieties (along with a chemically similar nonopioid decoy) cut with typical street pill ingredients like caffeine. They completed the feature-identifying measurements, then handed the raw data and the computer-generated library to another analytical chemist who'd never seen the mock pill, with a prompt: "We suspect there's fentanyl in this sample. Can you tell us which, if any, analogs are in it?"

The answer was a resounding "Yes."

After multiple cycles of narrowing down possible matches, the blinded chemist identified six of the mock pill's fentanyl components perfectly. The chemist narrowed another four down to a few possible candidates, no pure compound library needed. The remaining two lacked the signatures used for flagging or could not be fully teased apart. The results have not yet been peer-reviewed.

The approach is a "tremendous first step," but it relies on customized instruments that are unavailable to most forensic or national security laboratories, says A. Way Fountain III, a chemist at the University of South Carolina in Columbia. And the technique should be tested with other classes of drugs or molecules to show where improvements are needed, Fountain says.

Such tests are under way. Among several classes of molecules that Metz and colleagues are studying, they have identified common features in nitazenes — a new family of lab-made opioids that are becoming prevalent in overdose cases.

Wishart thinks the work will help modernize the forensic community's approach to identifying unknown compounds. Relying on a reference library of pure compounds "is still very 19th century thinking," he says.

Molecular pharmacologist Gary Miller agrees. "Reference-free identification could be revolutionary from a scientific standpoint," says Miller, of Columbia University. "These data demonstrate that the approach can work." ✖

HEALTH & MEDICINE

AN EXPERIMENTAL DRUG MAY HELP GLP-1 USERS SAVE MUSCLE

BY MEGHAN ROSEN

When people drop weight on GLP-1 meds, they can also lose muscle. But a proof-of-concept antibody might help preserve it.

When taken with a powerful weight-loss drug, the experimental antibody, called apitegromab, let patients hang on to lean body mass, metabolic disease researcher Richard Pratley and colleagues report in *Nature Medicine*.

Apitegromab hasn't yet been approved by the U.S. Food and Drug Administration and is available only via intravenous infusion. It's not something consumers will get their hands on any time soon, says Pratley, of the AdventHealth Translational Research Institute in Orlando, Fla. But the work cracks open a door to saving muscle that might otherwise be lost.

GLP-1 drugs can spark rapid weight loss, and 25 to 40 percent of that isn't fat but lean body mass: muscle, organs and blood. Though GLP-1 users may lose some muscle when they drop weight, the harms of this are probably less than people think, says Randy Seeley, an obesity researcher at the University of Michigan in Ann Arbor. For most users, maintaining enough muscle "isn't a problem," he says.

That view is supported by GLP-1 clinical trials. Losing lean body mass doesn't seem to impact function for most participants, Pratley says. But it's unclear how that data translates to real life. Apitegromab could be useful for some people.

Pratley's team tested the antibody in 102 people who were overweight or obese. All got weekly injections of the GLP-1 drug tirzepatide for 24 weeks; half also got a monthly antibody infusion. People in both groups lost about the same amount of weight, but those who got the antibody lost half as much lean mass on average and scored slightly better on preliminary strength tests.

Ultimately, the goal is to fine-tune medications to patients' specific needs, Pratley says. ✖

↖ **Apitegromab, an experimental antibody depicted here as a dumbbell, seems to help people who take weight-loss drugs hold onto lean body mass.**

LEANNE RULE

VOL. 208 NO. 08 | AUGUST 2026

THE HEALTH CHECKUP

A SPLASHY SUNBLOCK LANDS ON U.S. SHELVES

BY MEGHAN ROSEN

Glowy oils, tinted creams, misty sprays and chalky sticks: Like many people this summer, my pool bag holds a stunning variety of sunscreens. They come in different forms, but all rely on a handful of the same sun-shielding ingredients. In the United States, this ingredient list hasn't changed in 20 years — until now. The U.S. Food and Drug Administration has added a new chemical, bemotrizinol, to the list of permitted sunscreen ingredients.

"I think we should all be super excited," says dermatologist Tanya Hathaway. Bemotrizinol is more long-lasting than some other sunscreen ingredients, it's not readily absorbed into the bloodstream and it offers a new line of defense against the kind of solar radiation that ages skin. This sunscreen ingredient has been available in Europe for more than 25 years, says Hathaway, of Fred Hutch Cancer Center in Seattle. It has since become available in Japan, Canada, Australia and elsewhere. Bemotrizinol's entrance into the U.S. market could open the pipeline for other sunscreen ingredients regularly used around the world. The FDA's new rule, which goes into effect August 9, "is a great first step," Hathaway says. Sunscreens with bemotrizinol could hit shelves by late summer.

Sunscreens rely on a couple of tricks to shield people's skin. Mineral sunscreens use ingredients such as titanium dioxide and zinc oxide to create a protective barrier that reflects the sun's rays like a mirror flicking away light. But the minerals can leave a white sheen on the skin — think of a lifeguard's nose, says dermatologist Jennifer Stein of NYU Langone Health in New York City. "One of the problems with zinc and titanium is they don't rub in so well," Stein says. "And if people don't like the way their sunscreen smells or feels, they're not going to use it."

Previous chemical sunscreens, which often rub in a little better, use molecules such as avobenzone and oxybenzone to absorb the sun's radiation. These molecules are not very stable. When sunlight hits them, Stein says, they start to break down — hardly ideal for a product that's supposed to be used in the sun. Bemotrizinol also absorbs the sun's rays, but it is more stable than its chemical compatriots, which means people may not need to apply bemotrizinol formulas as often as other sunscreens.

The new ingredient is also adept at blocking a type of radiation from the sun called ultraviolet A1, which penetrates deeply into the skin. UVA1 can cause damage called photoaging, Hathaway says, which includes wrinkles, skin laxity and pigment changes. Current U.S. sunscreens do a good job of blocking UVB, which causes burns, but we've had fewer ways to block UVA, Hathaway says. Bemotrizinol "really expands that UVA spectrum of coverage."

The ingredient may also ease some people's sunscreen skepticism. In Stein's experience, patients who avoid sunscreen tend to worry about chemicals passing through the skin. That does happen with avobenzone, oxybenzone and some other chemical ingredients. "If you apply them to large areas of the body repeatedly throughout the day, they can get absorbed into the bloodstream," Stein says. But "there have never been any studies that have shown that current ingredients on the market are unsafe," Hathaway says.

Unlike other chemical ingredients, bemotrizinol is a big molecule, which makes it less able to slip through skin. Today, only three sunscreen ingredients — the minerals titanium dioxide and zinc oxide, and now bemotrizinol — are generally recognized as safe and effective, a category the FDA calls GRASE.

Even better than a fancy new sunscreen, though, are the low-tech options. "The more clothing you're wearing," Stein says, "the less you have to depend on sunscreen." ✖

Actual Size
38.1 mm

JUST RELEASED!

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No coin embodies the spirit of the 1920s and 30s more than the Peace Silver Dollar. First struck in 1921, the Peace Dollar was a celebration of the end of WWI and was the silver dollar of choice for prohibition bootleggers and bank robbers like John Dillinger and Bonnie and Clyde. The hefty Peace Silver Dollar holds a special place in American history, as the silver dollar helped America through the Great Depression.

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Features

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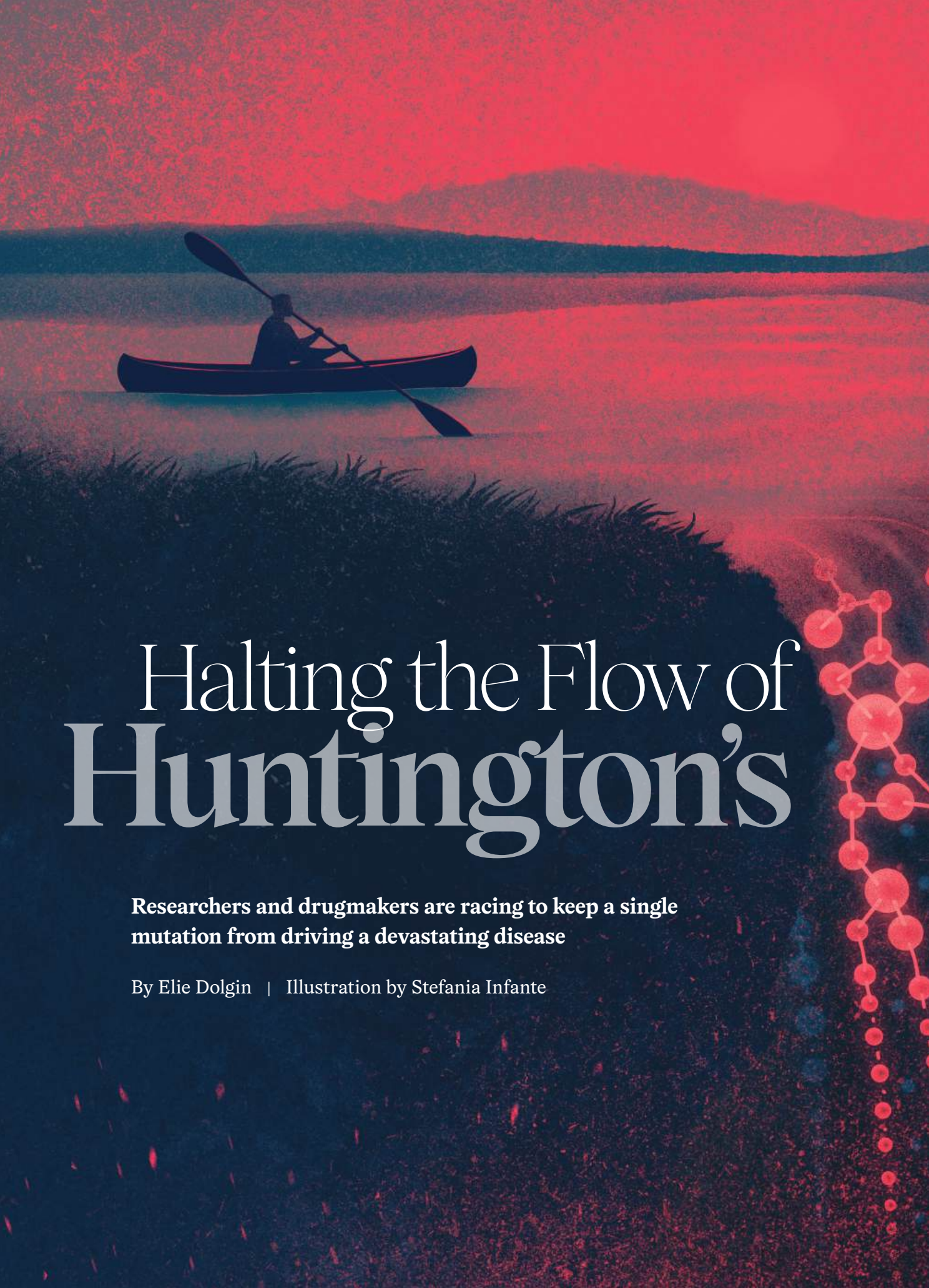
HEALTH & MEDICINE

BETTER WORLD A-COMIN'

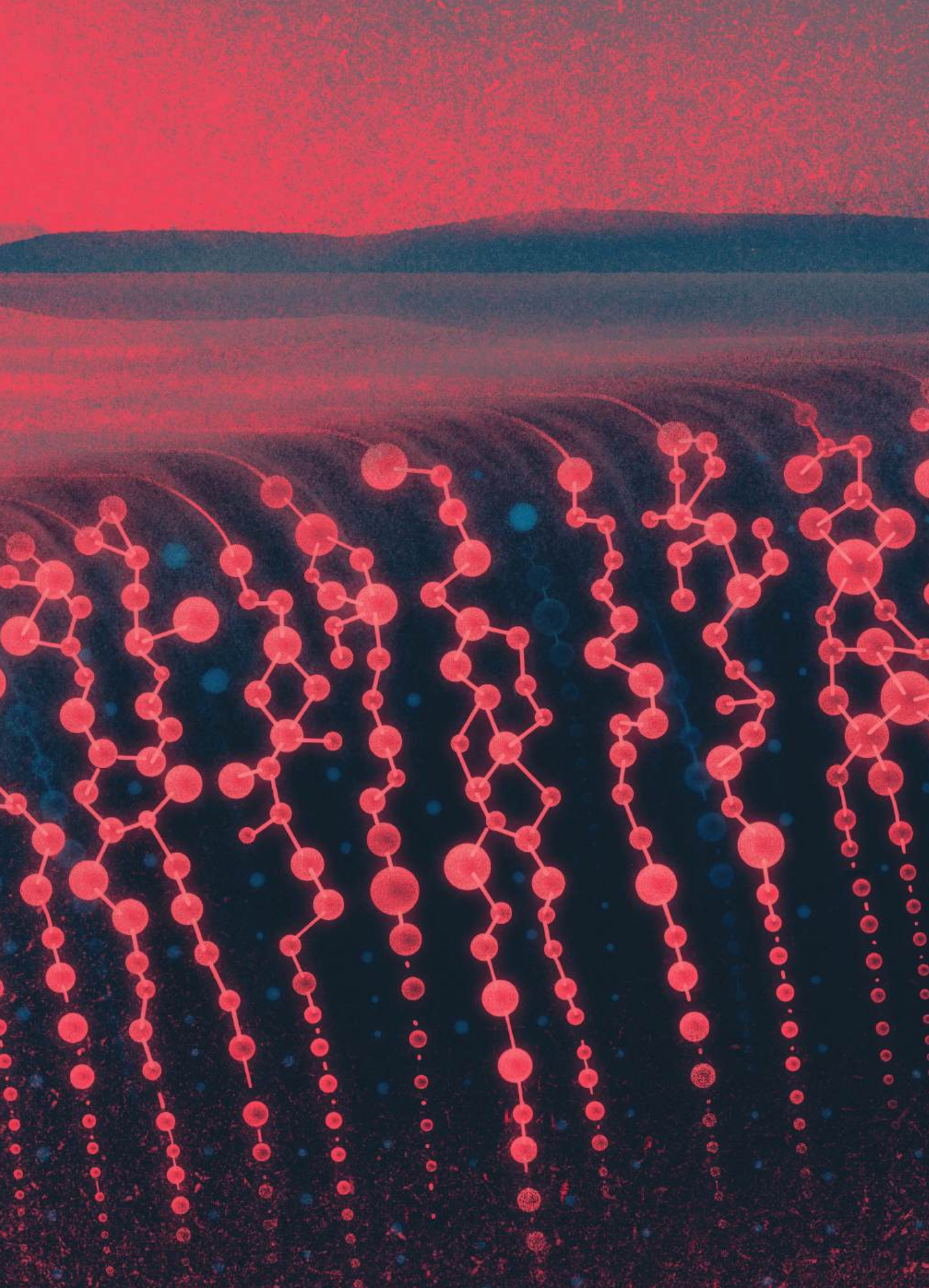
● Legendary folk singer Woody Guthrie, shown with his son Arlo and former wife Marjorie receiving an award from the U.S. Department of the Interior, died of complications from Huntington's disease in 1967, about a year and a half after this photo was taken. Marjorie dedicated her life to raising awareness of the disease, founding what became the Huntington's Disease Society of America. Her efforts helped lead to the discovery of the gene for Huntington's. Now, experimental treatments are targeting the specific mutation (see Page 34). — *Cassie Martin*



Halting the Flow of Huntington's

Researchers and drugmakers are racing to keep a single mutation from driving a devastating disease

By Elie Dolgin | Illustration by Stefania Infante



Jeff Carroll was in his mid-twenties, fresh out of the U.S. Army, when a genetic test confirmed his worst fear: He was going to develop Huntington's disease.

He had watched his mother's illness for years; the tremors first, small enough to explain away, then the involuntary movements that looked almost like dancing. The test revealed that the same mutation that caused her disease lived in him: a stretch of three DNA letters in a gene called *HTT*, repeated over and over again dozens of times.

Carroll himself was not sick yet. He would not get sick for many years. But the countdown had begun. In fact, it had likely been running all his life, deep inside vulnerable neurons in his brain. There, the mutant *HTT* gene was slowly growing longer, its internal repeats piling up toward a threshold that, once crossed, would tip the cell into disarray.

The first outward signs of Huntington's often begin with mood changes and subtle cognitive effects. The hallmark jerky movements come later. Eventually, and relentlessly, patients lose the ability to speak, swallow or move. Most people die within a decade or two of symptom onset.

Huntington's disease is rare, affecting roughly 1 in 20,000 people worldwide. Yet because each child of an affected parent has a 50 percent chance of inheriting the mutation, the disease can haunt families for generations. It had already claimed Carroll's grandmother and would take his mother at age 54. Without a therapy capable of altering its course, Carroll — along with three of his five siblings who also inherited the mutation — seemed fated to follow his ancestors into an early grave.

Carroll decided the only rational response was to become one of the scientists seeking ways to beat the disease. He was midway through his undergraduate studies when he learned, in 2003, that he carried the mutation. He pressed on, earning a Ph.D. and completing postdoctoral training before establishing his own research group devoted to understanding and slowing the disease stalking him from within. Now a neurobiologist at the Allen Institute in Seattle, Carroll is part of a new initiative launched in June dedicated to tackling fatal neurodegenerative diseases. He is leading the Huntington's strand of that work, with a major focus on somatic expansion, the lifelong growth of specific repeating DNA sequences. In Huntington's, this takes the form of CAG (encoding the nucleotides cytosine, adenine and guanine) in the *HTT* gene.

Once dismissed as a biological curiosity, somatic expansion is now viewed as a key force shaping when and how Huntington's and other diseases progress. For the field, this shift in thinking has opened a new therapeutic frontier. For Carroll, however, it has landed as a gut punch.

As Carroll now understands all too well, the mutation he inherited isn't static. It is still creeping longer, tacking on new CAG copies inside the very neurons it is destined to destroy. The genetic chain reaction

has already produced subtle motor and cognitive symptoms he regards unmistakably as Huntington's.

"It sucks as you think about it," says Carroll, who will turn 49 in August. "Not only do I have this horrible mutation, but it's getting worse over time."

That's the bad news. But fortunately for Carroll and others like him, the research into somatic expansion has revealed an unlikely silver lining. The expanding DNA repeat and the destruction of neurons do not appear to proceed in lockstep. Researchers now believe there may be a lengthy window, perhaps decades long, during which the repeats accumulate while much of the brain remains structurally and functionally intact.

A new generation of therapies aims to intervene at that gap between the disease's molecular march and its neurological consequences. The first drug candidates designed to target somatic expansion are slated to enter clinical testing later this year. If those medicines can slow or halt the process before too many neurons cross the point of no return, researchers hope they may preserve much of the vulnerable brain tissue that remains in people like Carroll.

"It's a beautiful intervention," Carroll says. "There's a good reason, based on the best science, to still be hopeful."

← Neurobiologist Jeff Carroll was diagnosed with Huntington's in 2003. He now leads research into the disease, with a focus on somatic expansion, at the Allen Institute in Seattle.

GENETIC GAMBIT

Huntington's has long carried a grim distinction: It is among the most clearly defined genetic tragedies in all of medicine.

Unlike complex disorders shaped by dozens of DNA variants, Huntington's follows a brutally simple rule: Inherit one copy of mutant *HTT*, with its stuttering stretch of CAG repeats, and the disease will come.

Though the first signs of disease typically appear between the ages of 30 and 50, exactly when the illness strikes depends largely on the number of CAG repeats a person starts with. Any repeat count above 40 virtually guarantees the disease will develop. But the genetic hand dealt at birth also sets a rough timetable, with longer CAG tracts hastening symptoms and shorter ones buying more years of healthy life.

Scientists identified *HTT* and its corresponding protein, huntingtin, in 1993 — the culmination of a decade-long hunt involving research teams from around the world. The discovery remains one of the most celebrated achievements in human genetics research, a moment that finally revealed the cause of Huntington's disease. It immediately raised hopes that an effective treatment would soon follow.

It did not.

The premise was hard to fault. Normally, the huntingtin protein encoded by *HTT* does vital work: guiding embryonic development, then helping neurons function in the adult brain. But the misshapen version churned out by the mutant gene builds up inside neurons and poisons them. So, the thinking went, clear out the toxic protein and the disease should loosen its grip. Yet one drug candidate after another stumbled in clinical trials.

The sharpest blow came in 2021, when late-stage trials of a drug called tominersen — once

the field's leading hope—had to be halted after investigators found the therapy not only didn't work, it appeared to make patients worse.

Then came an unexpected revival of the huntingtin-depleting strategy. Last year, a gene therapy from the Dutch company uniQure became the first treatment to slow the progression of Huntington's disease in a clinical trial. The therapy is designed to permanently dial down huntingtin production with a single infusion into the brain. When compared with matched controls from a database of untreated patients, the gene therapy showed at long last that "you can shift the dial" on the course of Huntington's, says Sarah Tabrizi, a neurologist at University College London who served as lead scientific adviser on the trial.

But it wasn't just the clinical results that convinced Tabrizi the effect was real. "It was the molecular evidence that nailed it for me," she says. Spinal fluid analyses showed that levels of a protein called neurofilament light, which is released by dying neurons, had been reduced—the opposite of what typically happens as Huntington's progresses.

The result was significant, though not without caveats. The therapy may have slowed the disease but did not stop it. Delivering it demands an hours-long brain surgery that only neurosurgeons at select medical centers can perform. And the evidence came from a small trial with no placebo-control arm, a methodological limitation that led some experts to question the strength of the conclusions.

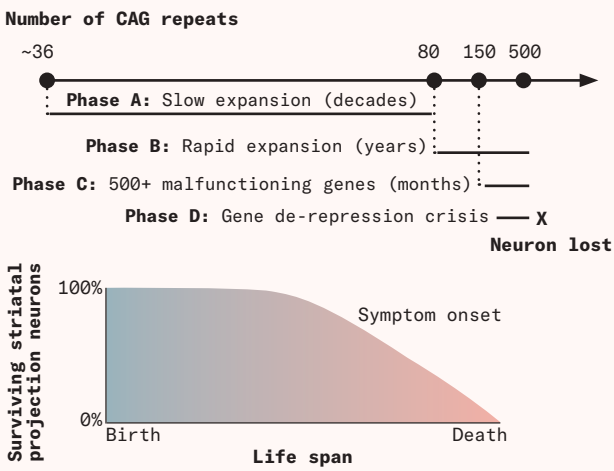
In June, the U.S. Food and Drug Administration agreed that the data could support a new-drug application after all. Yet even this milestone reinforced a growing suspicion that the field has been aiming too far downstream. Lowering huntingtin may help, researchers say, but it may not fully address the deeper biology driving the disease.

That does not mean abandoning the protein-lowering strategy. Carroll sees it as a necessary piece of the puzzle. Were the uniQure therapy approved today, he says, he would take it himself, and "I would endorse my family doing it."

For those wary of an invasive brain operation, other *HTT*-targeting therapies are coming, too. This year brought promising early-stage data on once-daily pills designed to throttle production of the toxic huntingtin protein.

Yet the field's center of gravity has continued to move upstream, toward the unstable DNA mutation and the cellular machinery that keeps making it worse.

THE STEADY MARCH OF HUNTINGTON'S



People with Huntington's go through distinct phases of somatic expansion (top), as CAG repeats accumulate. The disease severely affects specific neurons in the striatum (bottom), a brain area that controls motor function and decision-making, among other crucial tasks. As CAG repeats pile up, these neurons can undergo a de-repression crisis, when many normally repressed genes start to encode proteins, leading to neuron death.

REPAIR RUN AMOK

Almost as soon as the *HTT* gene was identified and genetic testing for Huntington's became available, scientists noticed something that didn't add up: Patients carrying nearly identical numbers of CAG repeats could develop symptoms years, sometimes decades, apart.

One person might start declining rapidly in his 40s while another might keep her faculties largely intact into her 60s. Same mutation, wildly different fates. Clearly, something else was at work.

R. E. HANDSAKER *ET AL*./*CELL* 2025

That something began coming into focus around a decade ago, when an international consortium led by neurogeneticist Jim Gusella of Massachusetts General Hospital in Boston identified some of the first genes implicated in determining when Huntington's strikes.

The research kept pointing to the same place: genes involved in DNA repair, the process cells use to correct the small copying errors that pile up as they read and maintain the genome. Subtle variations in these genes seemed to shift the age at which Huntington's symptoms would begin, pushing onset earlier or later than the inherited repeat length would predict.

A new picture began to emerge. Though the mutant *HTT* gene might determine who develops Huntington's, the DNA repair machinery seemed to help determine when the disease would set in. "It all fits together," Gusella says.

The DNA-repair proteins encoded by these modifier genes normally patrol the genome for mistakes and damage. The mutant *HTT* gene, however, turns this protective machinery against itself. Its repetitive DNA sequence can confuse the very proteins tasked with preserving genetic integrity, causing some to make the sequence longer than they found it. The result is gradual accumulation of CAG repeats.

But even as the repeating DNA sequence gets nudged ever longer by the cell's own repair machinery, the brain seems remarkably capable of absorbing the damage. It is only after the repeats cross a critical threshold that huntingtin turns lethal, pushing neurons into dysfunction and death.

Molecular biologist Nathaniel Heintz at the Rockefeller University in New York City and colleagues first laid out this logic in 2024 in *Nature Genetics*. That study showed the disease unfolds in two stages: first the slow expansion of the repeat, then a distinct toxic phase once the repeat has grown long enough.

"It's like going over a waterfall."

— Steve McCarroll

In parallel, neurogeneticist Steve McCarroll at Harvard Medical School and colleagues had been examining individual neurons taken from post-mortem human brains. Reporting in February 2025 in *Cell*, they measured the exact number of CAG repeats inside each cell. What they found mapped almost perfectly to what the earlier gene-hunting studies had hinted at.

Once the repeat reached roughly 80 copies, its growth began to accelerate. Beyond about 150 copies, gene activity inside affected neurons started to go haywire. Degeneration followed soon thereafter. And within months of crossing that threshold, neurons entered a rapid downward spiral toward death.

"It's like going over a waterfall," McCarroll says — calm for a long while, until suddenly the plunge is unavoidable.

A NEW TARGET EMERGES

Even before McCarroll's team confirmed this tipping point, the discoveries about somatic expansion had steered Huntington's researchers toward a new set of drug targets: components of the DNA-repair pathway that govern how quickly the CAG repeats expand.

Much of the drug discovery action has converged on a gene called *MSH3*, which encodes a key component of the DNA repair machinery. The protein recognizes small mismatches in DNA strands and recruits other repair proteins that can inadvertently lengthen the CAG sequence.

Natural variation in *MSH3* impacts how quickly the sequence expands and, by extension, how quickly Huntington's symptoms progress, Tabrizi and colleagues reported in 2017 in *Lancet Neurology*. That finding, along with similar activity in patients with myotonic dystrophy, another rare disorder caused by triplet DNA repeats, elevated *MSH3* from a molecular curiosity to a prime therapeutic target.

"If we can save Jeff's brain, then we've done our job."

—Ed Wild

In the years since, several research teams have shown that blocking *MSH3* activity in mice can slow — and in some cases nearly stop — the runaway repeats inside neurons.

As the evidence mounted, so did interest from industry, ushering in the first wave of biotech ventures built on the premise that Huntington's might be held at bay by preventing the mutant gene from growing more dangerous over time.

Interfering with a DNA-repair gene raises an obvious concern: Could weakening that system allow mutations to accumulate elsewhere in the body? People who naturally inherit two defective copies of *MSH3,* for example, develop clusters of intestinal polyps that can raise the risk of colorectal cancer. But inheriting only one defective copy seems to have little effect on overall health. That pattern has reassured many researchers that partial suppression of *MSH3*, especially if confined largely to the brain, would not cause significant harm.

Anastasia Khvorova, a chemical biologist at the UMass Chan Medical School in Worcester, has studied long-term *MSH3* suppression in mice. Up to a year of therapeutic silencing produced no obvious biological changes in these short-lived rodents, she and colleagues have found.

The findings don't necessarily guarantee that the treatment is safe for people, Khvorova says. "But if you're talking to Huntington's patients, I think this risk is something people are willing to take."

PROMISE AND PERIL

As yet, no company has translated the promise of stopping somatic expansion into a proven therapy, in part because the road to clinical testing has been anything but straightforward.

Triplet Therapeutics was among the first to try. The Cambridge, Mass.–based startup, which was founded in 2018, burst onto the biotech scene with tens of millions of dollars in venture financing, plus a who's who of scientific advisers including Gusella, Tabrizi, Carroll and other leaders in the field.

Scientists at Triplet quickly zeroed in on a type of drug known as an antisense therapy, which uses short strands of synthetic DNA or RNA to block production of a target protein. They developed one such drug that turned down *MSH3* expression in the brains of mice and monkeys, setting the stage for what would have been the first human trial of a therapy targeting somatic expansion. "We had everything ready," says neuropsychiatrist Irina Antonijevic, who led Triplet's drug development efforts.

But the company needed fresh capital to fund a clinical trial. And just as Antonijevic and colleagues were preparing to raise it, the disappointing results from trials of tominersen and another Huntington's antisense drug rattled confidence across the field. Investors suddenly grew reluctant to place more bets on Huntington's research, especially another antisense drug candidate.

Triplet shut down in 2022. The collapse came down to bad timing, not any crack in the biological case for targeting *MSH3*, says Antonijevic, who is now chief medical officer of the startup Trace Neuroscience. "I have no doubt about the science," she says.

Genetic evidence continues to tie the repair genes to how fast the disease unfolds, Gusella's consortium reported last June in *Nature Genetics*. And mouse studies have validated the idea that switching off the genes blunts runaway DNA growth and eases the disease's mark on the brain, according to a study published in 2025 in *Cell*.

Now, Latus Bio of Philadelphia and Boston has secured millions in funding and is on track to become the first company to test the strategy in humans using a new type of gene therapy. Possible approaches to slowing repeat expansion vary. Among companies targeting *MSH3*,

some like Latus Bio are developing biological drugs that must be injected directly into the brain or spinal fluid to reach their target. Others are pursuing small-molecule pills in which the drug is so tiny that it can easily cross from the bloodstream into the brain, no invasive procedures needed.

Other targets within the DNA-repair pathway are under investigation as well, including proteins that genetic and molecular studies suggest should be boosted, rather than blocked, to keep repeat expansion in check.

A DETERMINED PUSH FORWARD

None of this means the original strategy of targeting the huntingtin protein is obsolete. Such a therapy could still play an important role late in the disease course, after the DNA repeats have already expanded and when neurons are faltering but not yet gone, says David Howland, head of preclinical biology at the CHDI Foundation, a nonprofit research organization dedicated to Huntington's.

The ideal, Howland suspects, may be to pair the two approaches in a one-two punch — first freezing the genetic expansion to stop the disease at its source, then clearing the toxic protein already damaging neurons.

But a combination attack on Huntington's is probably years away. Drugs that achieve each half of the strategy generally have to be established on their own before the two can be brought together. In the meantime, researchers face a choice about where to place their bets.

"Five years ago, probably 95 percent of people would have said huntingtin-lowering is the top priority target by a long way," says neurologist Ed Wild of University College London. Now, Wild says, that consensus has dissolved, and experts are hard-pressed to say whether lowering huntingtin or modulating DNA repair should come first.

41,000

The number of U.S. patients with symptomatic Huntington's disease

50 percent

The chance that a child of someone with Huntington's will inherit the mutation

>200k

People in the United States at risk of inheriting the Huntington's gene

And the implications reach well beyond a single disease. A drug that freezes repeat expansion in Huntington's could, in principle, work against myotonic dystrophy, spinocerebellar ataxia and a wide range of other diseases that follow similar genetic dynamics.

"Huntington's is the disorder for which we have the most compelling data," says Darren Monckton, a geneticist at the University of Glasgow in Scotland who studies these conditions. "But all the mouse and tissue-culture data say it's the same players [in the DNA-repair pathway] and absolutely we should be thinking about targeting them to treat these other disorders as well."

For the scientists closest to this work, success or failure won't be measured only in trial data. It will be measured in people, and one person in particular: Jeff Carroll.

"If we can save Jeff's brain, then we've done our job," says Wild, who has known Carroll since they were graduate students. "If we can't, then we probably should have stayed a few more nights late at work."

Carroll himself holds a clear-eyed view. He still has research to do and a life to live. Despite his symptoms, he is producing some of the strongest science of his career, colleagues say. In the last year, his lab reported that one type of huntingtin-depletion therapy can also curb the expansion of repeats, a finding that knits together the field's two leading bets.

He is also a father to twin 20-year-olds — both conceived via IVF and with genetic screening to spare them the Huntington's mutation — and he helps shoulder the care of his siblings who also inherited it. "It's very clear that Jeff's reserves are far from empty," Wild says.

But they are steadily depleting. Inside Carroll's brain, the mutant DNA continues lengthening. The slow neurological countdown set in motion by his genetic inheritance is advancing, even as the science is trying to stop it. Through it all, he keeps moving forward — for himself, for his family and for the tens of thousands of others living with the same diagnosis.

"We have to hurry," Carroll says. "We have to keep pushing." ✖

Elie Dolgin is a science journalist who specializes in biomedical research and drug discovery.

SOURCE: HUNTINGTON'S DISEASE SOCIETY OF AMERICA

Going to space?

As the Artemis II astronauts began their passage over the moon's farside in April, they captured this view of Earth dropping below the lunar horizon— a nod to Apollo 8's indelible 1968 "Earthrise" photo.

Pack a camera.

Planetary scientist Candice Hansen-Koharcheck knew that images can make remote worlds feel near **By Marina Koren**

Humankind's journey around the moon in April produced a spectacular photo album of our corner of the universe. Here was the crescent Earth, a luminous sliver surrounded by complete darkness, behind the graphite-colored moon. There was the moon's farside rippled with craters, the way raindrops draw rings across the surface of a lake. There was the moon again, this time a shadowy marble levitating in space, encased in the soft glow of sunlight.

Only four people basked in these spellbinding views. But by capturing and sharing the snapshots, NASA's Artemis II astronauts made myself and many others back on Earth feel momentarily, dazzlingly weightless.

That's the magic of a good space picture. A single frame can shrink the distance between here and way out there, compressing the wonders of our celestial neighborhood down to the scale of human experience.

When I think about

poignant,

otherworldly

photography,

I think of Candy.

Candice Hansen-Koharcheck was not an astronaut. She was a planetary scientist. But through nearly 50 years of robotic space missions, she touched almost every planet in the solar system and many of its moons. She was the first person to lay eyes on the "Pale Blue Dot," an iconic portrait of Earth from afar that inspired Carl Sagan's memorable description of our planet as "a mote of dust suspended in a sunbeam."

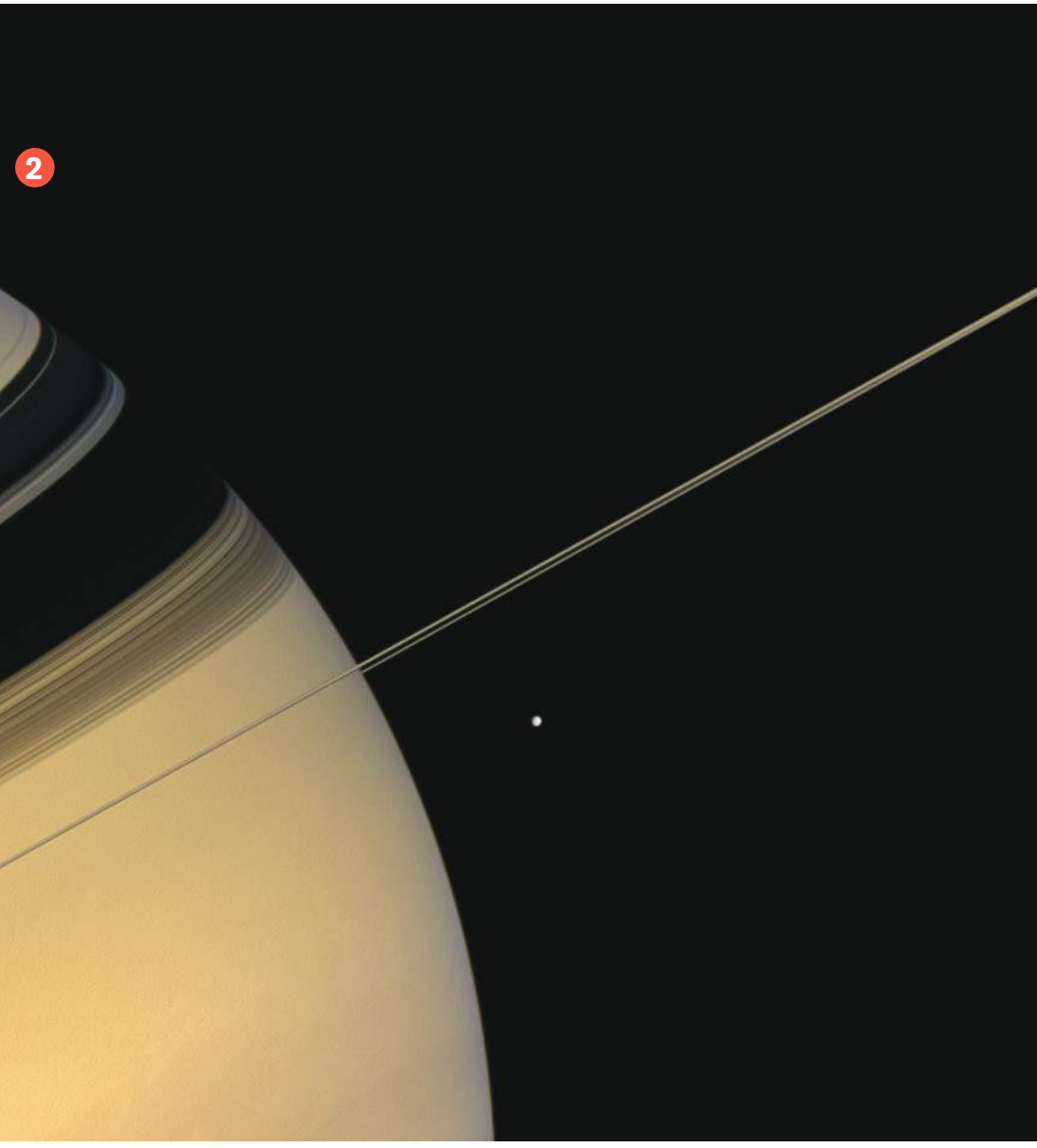
Hansen-Koharcheck died of cancer at age 72 on April 11, the day after the Artemis II crew returned home.

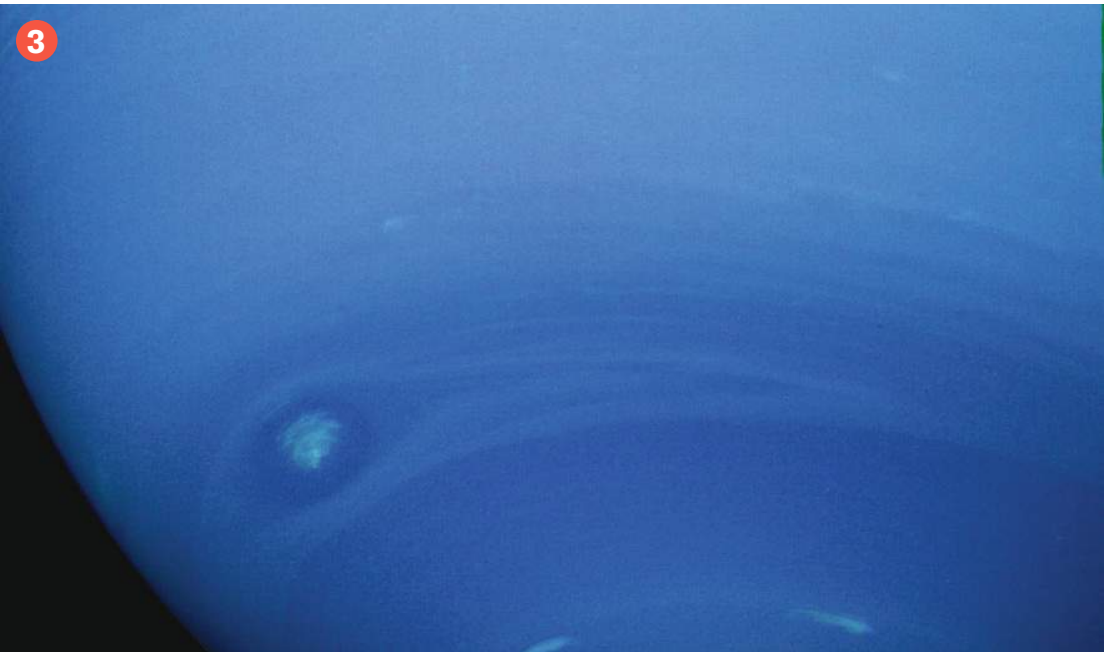
As a space journalist, I'd interviewed Hansen-Koharcheck many times over the years. We talked about the Cassini mission's exploration of Saturn before the spacecraft plunged into the planet's atmosphere and the workings of the Voyager probes that still call home from interstellar space. Most often, we'd marvel at pictures of Jupiter from the Juno mission, whose camera team she led. "Even with the best possible telescope, you'll never get a perspective like this from the Earth," she said.

Hansen-Koharcheck knew well the power of space photography and the purpose of bringing it home. Most human beings will never travel beyond Earth, at least not in the foreseeable future. The camera may be the most meaningful piece of equipment on any space mission.

Why go to outer space if you're not going to show everyone else what you witnessed?

Looping around the moon, the Artemis II astronauts created a modern-day version of one of the most iconic space pictures in history: the Apollo 8 mission's "Earthrise," taken in 1968. While an





1 Candice Hansen-Koharcheck points to something on an image of Neptune's moon Triton from Voyager 2 while sitting in her office at NASA's Jet Propulsion Laboratory in 1989.

2 Saturn's iconic rings, seen edge-on, cast a shadow on the planet in this photo of the giant world and one of its icy moons, Mimas, taken by the Cassini spacecraft in 2007.

3 A white swirl of clouds mars the cobalt blue of Neptune's atmosphere in this snapshot from Voyager 2, taken in 1989 when the probe was over 4 million kilometers from the planet.

4 The HiRISE camera aboard NASA's Mars Reconnaissance Orbiter captured this contrast-enhanced view of a 9-kilometer-wide impact crater amid rugged terrain in 2016, as the spacecraft passed over an equatorial region of the Red Planet.

uncrewed spacecraft had previously snapped a similar shot, the Apollo one was in full, glorious color. The arresting image walloped the public with the existential reality of the world as a vulnerable planet, and it helped ignite the modern environmental movement.

Soon after the Apollo missions recast the moon as a real place — not just a mystical orb in the sky — NASA sent two uncrewed spacecraft, Voyager 1 and Voyager 2, deeper into space in 1977. Hansen-Koharcheck joined the mission right at the start, her first job out of college. Over 12 years, the probes pirouetted past Jupiter, Saturn, Uranus, Neptune and an assortment of their moons. Hansen-Koharcheck designed every flyby's camera sequences: timings, filters, exposures.

"Voyager really belongs to everyone in the world," Hansen-Koharcheck told me in 2019. "It wasn't like there was some lucky person on a spacecraft standing at a porthole who had a better view than anybody else. We all experienced that same view of passing through our solar system and seeing things for the first time." It seemed as if the world was crowded into a darkroom, watching the results materialize.

The "Pale Blue Dot" came along in 1990, as Voyager 1 flew toward the solar system's edges. Sagan, Hansen-Koharcheck and others on the imaging team at NASA's Jet Propulsion Laboratory in Pasadena, Calif., had spent years trying to convince the top brass that one final look back was worth it before the cameras shut off.

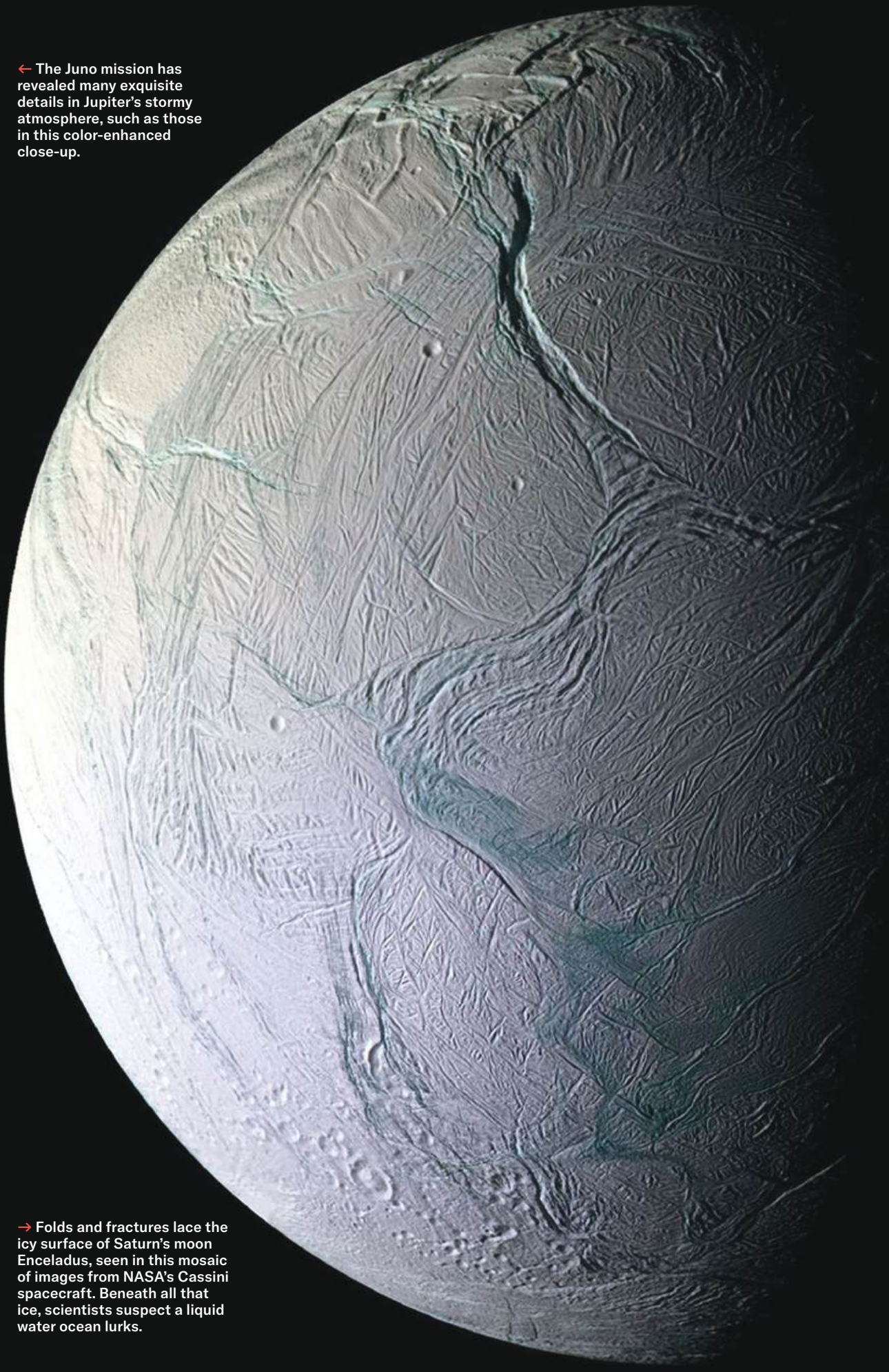
Hansen-Koharcheck was in her office when the dusky image, beamed across roughly 6 billion kilometers, appeared on her computer screen. "It was really quite overwhelming to think about," she told *National Geographic* in 2020. "That our little spacecraft was so far away, that this was a picture of home, and somewhere in that little bright speck, I was sitting at my desk."

Hansen-Koharcheck's research interests took her to the ethereal plumes of Saturn's moon Enceladus, the icy shell of Jupiter's moon Europa and the hazy atmosphere of Pluto. As deputy principal investigator on HiRISE, the camera aboard NASA's Mars Reconnaissance Orbiter, she investigated seasonal shifts in Martian polar regions.

"It's hard not to think about her anywhere you go in the solar system," says Alfred McEwen, a planetary geologist at the University of Arizona in Tucson who also worked on HiRISE, which has been photographing Mars since 2006.

x

Why go to outer space if you're not going to show everyone else what you witnessed?



← The Juno mission has revealed many exquisite details in Jupiter's stormy atmosphere, such as those in this color-enhanced close-up.

→ Folds and fractures lace the icy surface of Saturn's moon Enceladus, seen in this mosaic of images from NASA's Cassini spacecraft. Beneath all that ice, scientists suspect a liquid water ocean lurks.

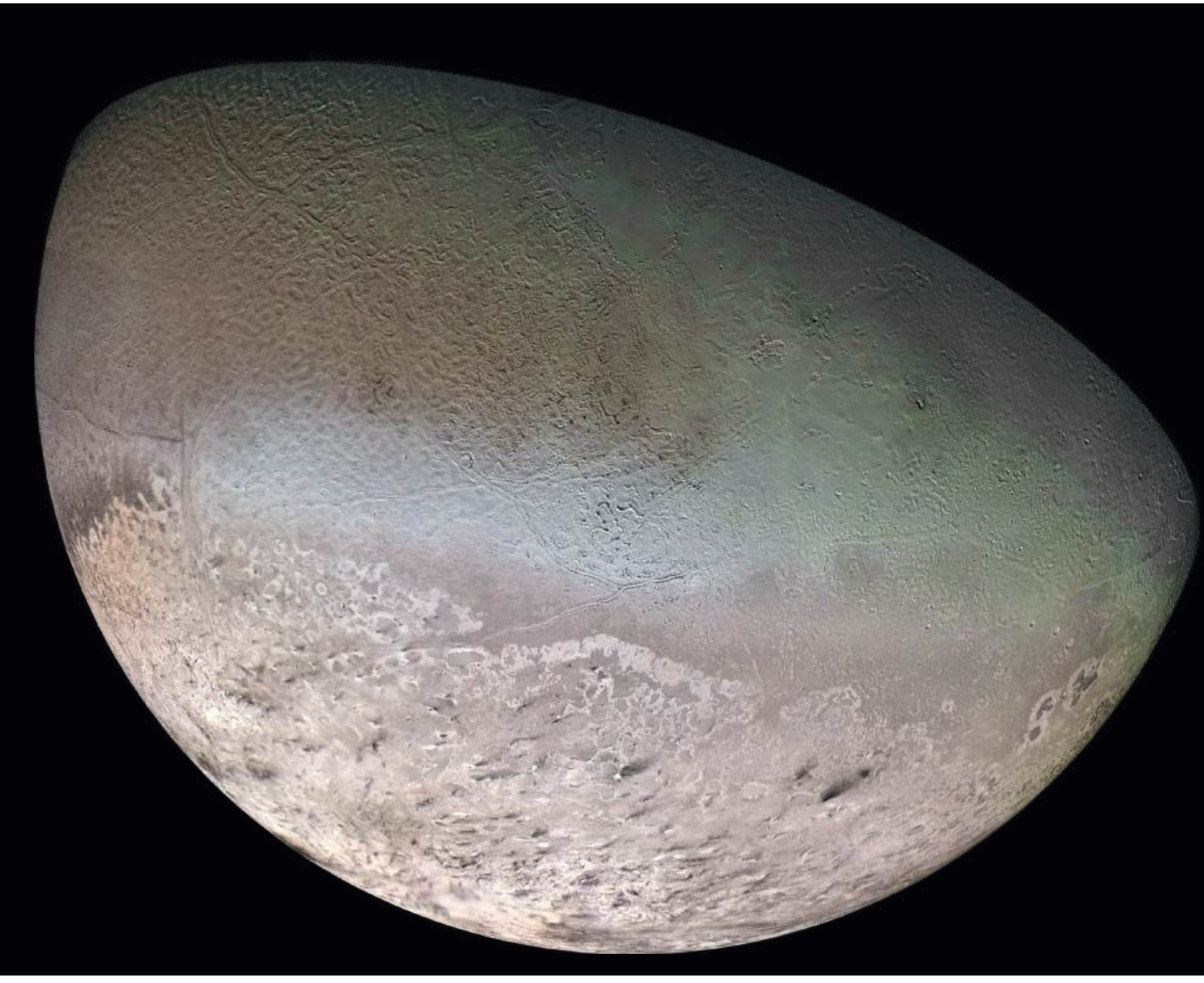
x

The best space pictures make the unfathomable feel familiar and the abstract like home.

→ Good space cameras bring observers tantalizingly close to celestial phenomena, such as Jupiter's Great Red Spot, seen in this 2018 photo from the Juno spacecraft.

↘ As NASA's New Horizons probe departed Pluto in 2015, it spun around to look back, capturing an image of the remote world backlit by the sun, revealing a hazy atmosphere.

↓ In 1989, the Voyager 2 probe pulled the curtain back on Neptune's largest moon, Triton, shown in this mosaic from the spacecraft.



Hansen-Koharcheck often brought non-scientists along. The HiRISE team solicited public suggestions for spots to photograph on Mars; so did her group on JunoCam, the Juno mission's camera. The raw data went online quickly, letting enthusiasts process the images to reveal Jupiter's stormy atmosphere in exquisite detail. The clouds, rendered in soft beiges and blues, made me think of cream swirling in coffee; they reminded Hansen-Koharcheck of her grandmother's crochet patterns. The best space pictures make the unfathomable feel familiar and the abstract like home.

In recent years, Hansen-Koharcheck was excited about two spacecraft bound for Jupiter's icy moons — NASA's Europa Clipper and the European Space Agency's Juice mission. And she still dreamed of a dedicated mission to Uranus, says Fran Bagenal, a planetary scientist at the University of Colorado Boulder who worked with her on Voyager and Juno. Hansen-Koharcheck was especially eager for a return to Neptune's largest moon, Triton, where Voyager 2 spotted geyserlike activity in 1989. Scientists suspect a liquid ocean lies beneath its frigid surface, which would make Triton one of the most distant ocean worlds in the solar system.

For Hansen-Koharcheck, there was always more to learn, more to see. In 2017, the Juno mission came within just 9,000 kilometers of the cloud tops in Jupiter's Great Red Spot, revealing the fiery eddies within. I asked her what it felt like to come so close to the colossal storm. She pointed me to an artist's illustration on JunoCam's website: a brown-haired child standing on a colorful precipice, angelic feathers draped across her back, gazing up at a massive, glowing planet. "I would say, emotionally, this captures it for me," she said. "Just moving in closer and closer and seeing this world. And as you get closer, you don't know what you're going to see. But you know it's going to be fantastic." ✖

Marina Koren is a science journalist based in Washington, D.C.

IMAGE CREDITS: EARTHSET–NASA; HANSEN-KOHARCHECK–DAVID PARKER/SCIENCE SOURCE; MARS–JPL-CALTECH/NASA, UNIV. OF ARIZONA; SATURN–JPL/NASA, SPACE SCIENCE INST.; NEPTUNE–JPL/NASA; JUPITER CLOUDS–DATA: JPL-CALTECH/NASA, SWRI, MSSS, PROCESSING: KEVIN M. GILL, © CC BY; ENCELADUS–JPL/NASA, SPACE SCIENCE INST.; TRITON–NASA, JPL/NASA, USGS; GREAT RED SPOT–SWRI, MSSS, JPL-CALTECH/NASA, GERALD EICHSTÄDT, SEÁN DORAN; PLUTO–NASA, JOHNS HOPKINS UNIV. APPLIED PHYSICS LAB., SWRI


graduated cylinder

What remains

Many universities and museums possess human remains collected without consent. Biological anthropologist **Fatimah Jackson** is leading an effort to prevent history from repeating.

By Michael Marshall

ILLUSTRATION BY BEN JONES

"Shock and awe." That was Fatimah Jackson's reaction when police dropped a makeshift bomb onto a residential building in Philadelphia on May 13, 1985. The brick-faced row house was the communal home of MOVE, an organization that advocated for Black liberation, natural living and animal rights. After a daylong standoff and shootout, during which MOVE members refused to leave the building, the city's mayor gave police the go-ahead to drop an explosive device. In the ensuing inferno, 61 houses burned to the ground — and six adults and five children were killed.

The consequences of that day are still playing out. In 2021, local journalists reported that the University of Pennsylvania had been keeping human remains from the bombing — tentatively identified as 14-year-old Katricia "Tree" Africa and 12-year-old Delisha Africa — in a cardboard box on a shelf, without their relatives' knowledge or consent. The remains, originally entrusted to the school by the Philadelphia Medical Examiner's Office, had also been used as teaching materials.

Although the remains of Delisha and Tree have since been returned to their families — and the school and city apologized — the MOVE incident is one example in a long line of researchers keeping human remains without consent, says Jackson, a biological anthropologist who retired in 2024 from Howard University in Washington, D.C. "We're the ones who are holding this material," she says. "The scientists are the culprits here."

Spurred by the MOVE revelations, the American Association of Biological Anthropologists in 2021 created a task force to craft new guidelines for the ethical study of human remains, with Jackson as cochair. It was an intimidatingly large project, she says, but too important not to take on. "Even after the Civil War, people in Black communities were talking about the grave robbers that would come and steal the bodies, and these bodies would show up in white medical schools," she says. "We had a lot of pain, but we didn't have any solutions for the pain."

Other experts see Jackson as a natural choice for the project. "Across the course of my own career, I've seen major shifts in the ways that we talk about ancestors, the ways that we perceive their treatment, and the necessity for consultation with contemporary descendants or experts," says Jennifer Raff, an anthropological geneticist at the University of Kansas in Lawrence. Jackson, she says, "was, and remains, one of the most influential voices in this movement."

Biological anthropologist Fatimah Jackson is setting new standards for the ethical use of human remains.

Research over remains

Anthropologists and anatomists have been studying human remains without consent for centuries. In the 1800s, physician Samuel George Morton infamously collected hundreds of human skulls, launching a new field of science — and propping up his views on white superiority in the process. The Smithsonian's National Museum of Natural History alone holds the remains of about 30,700 individuals. Many come from paupers' graves, slave cemeteries, Native American burial sites or countries formerly under Western colonization, most notably India.

"Especially in the early days of American physical anthropology, there was an expectation that science and scientific research was paramount and took precedence over any other consideration," Raff says.

Those practices started to come under fire in the 1970s. Maria Pearson — a member of the Turtle Clan of the Yankton Sioux — challenged how research institutions handled Native American remains and demanded that they be returned to their communities. Her work led to the first state law protecting Native burial sites, in Iowa, in 1976 — and ultimately the passage of the Native American Graves Protection and Repatriation Act in 1990.

NAGPRA led to the repatriation

> Fatimah Jackson "was, and remains, one of the most influential voices in [the] movement."
>
> **— Jennifer Raff**

FROM LEFT: COURTSEY OF HOWARD UNIV.; GEORGE WIDMAN/AP PHOTO

The 1985 MOVE bombing leveled 61 homes and killed 11 people, including five children.

of thousands of Native American remains. But it was not without controversy. After the 8,400-year-old nearly complete skeleton of an individual known as the Ancient One, or Kennewick Man, was discovered on the banks of the Columbia River in 1996, five Columbia Basin tribes requested the remains under the new law; researchers won a lawsuit to study them, instead. Years of legal battles and forensic testing ensued. In 2017, the remains were at last returned to the tribes and buried in a hidden location in the Pacific Northwest.

The disposition of other remains, including those of enslaved individuals, has been equally fraught. The country's oldest — and largest — cemetery for people of African descent was unearthed in Manhattan during the construction of a government building in 1991. Only after sustained protests did officials decide to preserve the site, eventually creating the African Burial Ground National Monument.

Scientists' traditional treatment of human remains has been "very much a free-for-all," Jackson says. But that has started to change. By 2024, at least three major scientific organizations had published guidelines for the care of these remnants: the American Association for Anatomy, the American Anthropological Association and the Smithsonian Institution.

Contemporary anthropologists are still wrestling with their forebears' legacy. "We have these collections that have been assembled by the early physical anthropologists," Raff says. They contain a mixture of skeletons, artifacts and DNA. "We are struggling to figure out what is the most ethical way to care for them, to study them. In many cases, it's not to study them. In many cases, it is to return them to contemporary descendants."

African American anthropology

Jackson was born in 1950 and grew up in Denver. Her family had been among the first settlers of nearby Dearfield, an African American homesteading town that was abandoned after the Great Depression. In Denver's Five Points neighborhood, Jackson lived among African Americans, Mexican Americans and Japanese Americans who had recently returned from World War II internment camps. In her redlined community, "a lot of people were getting the short end of the stick," Jackson says.

In the late 1960s, Jackson enrolled at the University of Colorado Boulder — and found herself regularly in white company for the first time. It was an inspiration of sorts. "I was interested in biological anthropology because of the differences in morphology I saw in the people around me," she says.

After two years, on the recommendation of a trusted professor, Jackson transferred to Cornell University to continue her education in anthropology. She studied genetics, nutrition and parasitology. She also met her future husband, nutrition scientist Robert Jackson. By coincidence, she had first seen his picture in the newspaper in Colorado: He had been one of several African American students who took over Cornell's Willard Straight Hall in a 1969 protest demanding a Black Studies program.

The pair married, and Jackson graduated in 1972. They moved to Tanzania that same year, where Robert did postgraduate fieldwork and Jackson took a three-year leave of absence from her studies. She had the first two of their six

children and worked as a teacher to support the family.

Then Jackson's life changed irrevocably. She contracted malaria in 1974, and the infection was brutal: She temporarily lost her eyesight and could not walk or speak.

Close to death, Jackson made a promise to God that if she lived, she would study malaria. Her prayers were answered, and she recovered. In 1975, she began a Ph.D. at Cornell. But by the time she was ready to go back into the field, Tanzania and Uganda were at war. Reluctant to return to what was now a war zone, she moved to Liberia, where she wrote a dissertation on genetic adaptations to malaria in Liberian mothers and children.

When she finally returned to the United States in 1980, Jackson noted the racism — both overt and subtle — that was part of daily life. "People would refuse to get on the elevator with us," she said. And her chosen field of anthropology had often dismissed the experiences of disenfranchised groups. "Race science was still very strong in anthropology at that time," she says, with many authors still publishing pseudoscientific studies about racial differences in characteristics such as intelligence.

For Jackson, that bias came to a head in the groundbreaking Human Genome Project. The project, which ran from 1990 to 2003, used DNA from 20 people: one of blended ancestry and 19 of primarily European ancestry. Though Jackson supported the project, she also saw it as a continuation of 20th century practices in which African Americans were routinely excluded from research that collected baseline scientific and medical data.

The project's leaders "wanted to characterize the human genome, but which genome?" Jackson asks. In 1994, she helped organize fellow academics to write a manifesto, in which they called for the project to include more samples from African Americans — and more African American researchers.

That experience inspired Jackson to develop a more inclusive approach to genomics. In 2009, while at the University of Maryland, College Park, she published a paper on a new technique she called ethnogenetic layering, which can identify the patterns of genetic variation within complex populations.

"African Americans are an amalgamation of a lot of different African groups, with admixture from certain Native American groups and specific European groups," Jackson says. Her new method tried to figure out where different genetic variants had come from, how they interacted with environmental and cultural factors, and how they affected disease risk. Instead of treating African Americans as a single group, it helped reveal the richness of their genetic diversity, Jackson reported in the *Annals of Human Biology*.

"She's made a lot of contributions, I think, in general to thinking more critically about groups of people in ways that aren't focused on racial categorization," says evolutionary biologist Benjamin Auerbach at the University of Tennessee in Knoxville.

In 2018, Jackson and her team at Howard University published a study detailing how institutionalized racism and enslavement may have affected African Americans' biology via epigenetics. Previous studies had shown the process — in which environmental cues can change gene expression without altering the underlying DNA — can be triggered by elevated levels of the stress hormone cortisol. That work in the *Journal of Clinical Epigenetics* suggested potential

Howard University students examine skeletal remains from the 19th and 20th centuries for signs of cardiovascular diseases.

COURTESY OF F. JACKSON

epigenetic shifts in African American populations — and proposed that scientists start searching for them. Subsequent studies have found evidence of faster epigenetic aging among African Americans who have repeated exposure to racism.

Then in 2021, the MOVE bombing erupted back into the spotlight.

Caring for ancestors

The revelation that the MOVE victims' remains had been kept without their relatives' consent, stored carelessly in a box and used as teaching props caused widespread outrage.

The biological anthropologists' association chose Auerbach to lead its new task force to systematize the ethical management of human remains; he immediately asked Jackson to be his cochair. As director of the Cobb Biological Anthropology Laboratory at Howard University, and with her decades of community engagement, Jackson was the ideal partner, Auerbach says. "She's really good at thinking about nuance."

Not that it was easy. In the wake of the MOVE revelations — and the racial reckoning inspired by the Black Lives Matter movement — some researchers publicly advocated for the end of all studies involving human remains. Jackson and her colleagues wanted to take a more thoughtful approach. "The goal was to broaden the discussion and not to trivialize what was done," Jackson says. Instead, the task force sought to balance the value of scientific research with respect for the communities it affected. "The community has a right to be involved in the research," she says.

First, the task force narrowed the scope of the project to focus on just one group. It chose African Americans, whom Jackson says are overrepresented in legacy human tissue collections. Then, it sought input from people in that community.

"Since we didn't have any money for the task force, I had to use my students," says Jackson. She taught 52 of them how to run focus groups and ask descendant communities how they wanted their ancestors' remains treated. Over four years, her students spoke to more than 3,000 African Americans from 35 states.

"You need to ask me before you work on my grandmother's bones," said one respondent, a young man from Orange Mount, Tenn. Another, an older man from Chicago, said: "Once we give our approval [for the research], that is not the end of it. We need to be kept abreast of everything else."

Auerbach also surveyed anthropologists across the United States to find out what skeletal materials they had — and what they hoped to do with them.

The result is a set of recommendations published in the *American Journal of Biological Anthropology* in March. In addition to requiring "explicit consent" from relevant communities, the guidelines call on institutions to inventory remains in their legacy collections, ascertain the identities of descendant communities and engage those groups in dialogue. Both researchers and community members said they wanted mutual formal partnerships to help them make decisions about ancestral remains.

> "You need to ask me before you work on my grandmother's bones."
>
> **— Anonymous Focus Group Participant**

Raff cautions that the next steps could be tough. Different communities will want different outcomes, she says. And the mistrust born of abuses such as the Tuskegee experiments — in which African Americans who had syphilis were not told of their disease status and left untreated — echo to this day.

"We're lucky," says Raff, "that we have very thoughtful researchers like Dr. Jackson, who are going out and doing the work of talking to communities and finding out what is it that they think we should be doing."

But the current environment for scientific research in the United States is making that work harder, Jackson says. "The guidelines were created in a more progressive political situation." She worries that the Trump administration's slashing of research funding for social sciences and social justice could hamstring the work of her group and others.

"At least we provided the groundwork," Jackson says of the project. She hopes others will build on what she's done. "You're starting to hear the voice of the people. It's been a long time coming, but it's the change we've been waiting for." ✖

Michael Marshall is a science journalist and author based in Devon, England.

Fundamentals First Fund

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2/21/24 IPO	Market Open:	$10.00
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Dividends Paid:		$0.32

Abstract

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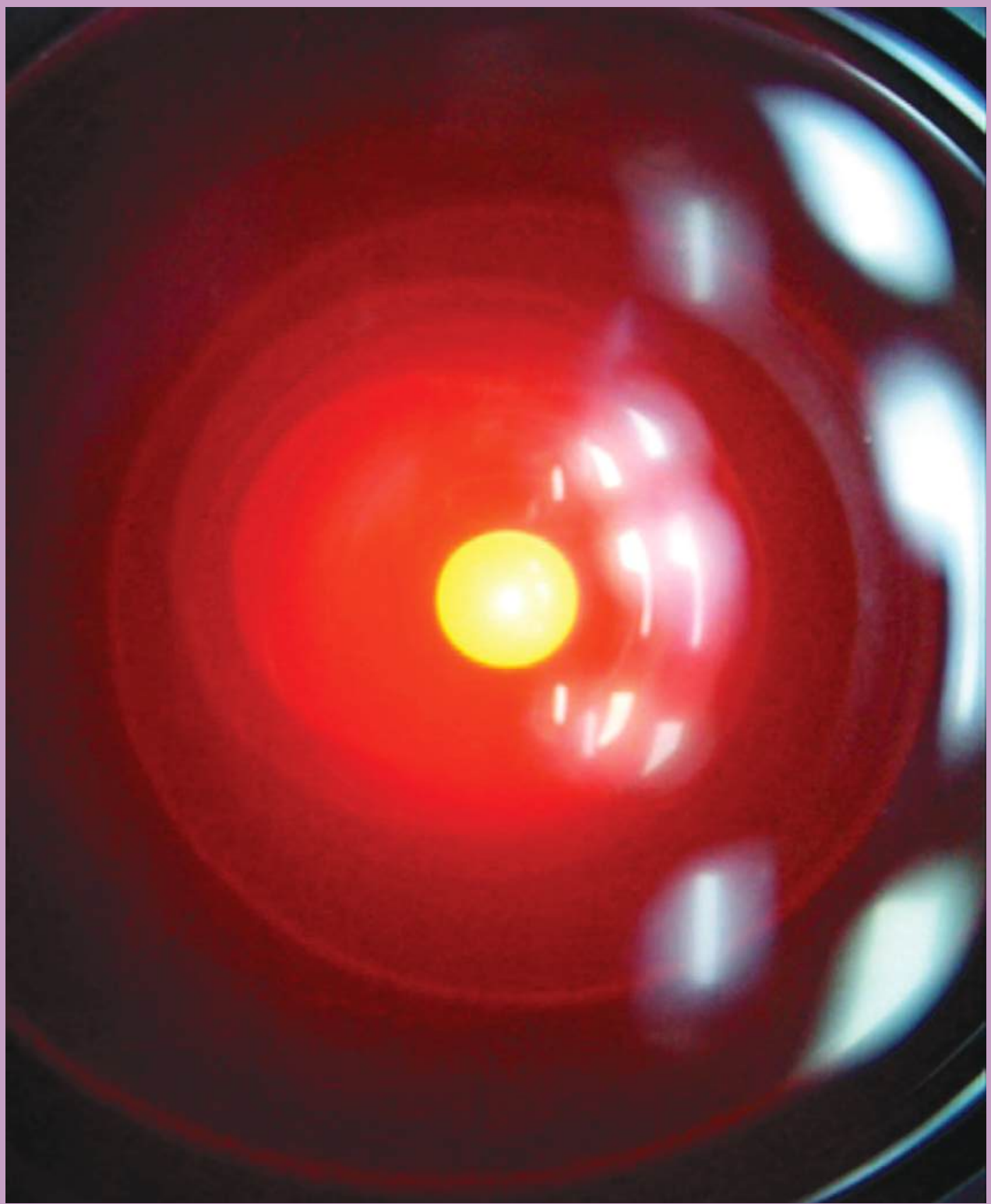
Curiosities

Artificial intelligence agent HAL goes from helpful to homicidal in *2001: A Space Odyssey*. What would it take for AI to helm a real space mission? See Page 62.



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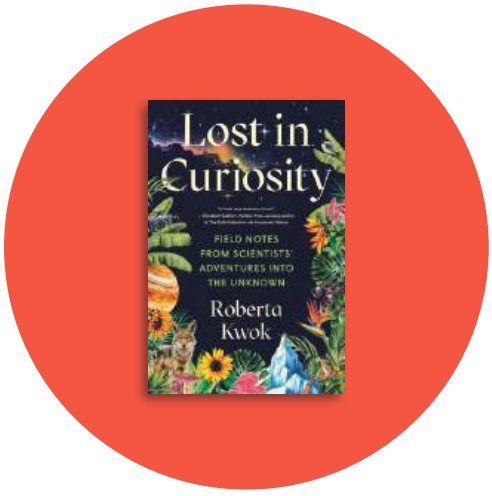

TALES OF SCIENTIFIC DISCOVERY ARE NEVER AS TIDY AS THEY SEEM

By Shi En Kim

LOST IN CURIOSITY | *Roberta Kwok*
Sourcebooks | $27.99

Earning my Ph.D. was the hardest thing I've ever done, and I remember the first time I wanted to quit. I was a first-year at the University of Chicago, and while taking images of my nanomaterial with a fancy microscope, I accidentally rammed the material into the instrument's $10,000 detector. I spent the next hour tearfully scrolling eBay for a replacement part and wondering how I could afford it. I was fortunate — the instrument was unscathed. But it was the first of many scares, failures and mishaps that battered my short-lived scientific career.

Stories like mine fill journalist Roberta Kwok's debut book, *Lost in Curiosity*, which looks at science's unglamorous side: the hair-pulling, gut-wrenching, head-scratching, "whoopsie" parts that are often omitted from tales of discovery.

Kwok corrects that narrative glossing over. The book opens with glaciologists racing to study ice melting in Greenland. Over many years, bad weather, a helicopter mix-up and a raging COVID-19 pandemic grounded the team. The ups and downs are as funny as they are tragic, as thrilling as they are painful. Readers will laugh, cringe and cry.

For people who may be unfamiliar with the scientific process, Kwok reveals its true nature: Doing science is hard, and it's a team effort. Though the book's title whimsically evokes passionate nerds losing themselves in the joy of their curiosity-driven pursuits, often they are simply lost, trying to formulate a plan B for the next experiment. That's just how science is — nature doesn't give up its secrets easily.

Readers get a taste of the myriad questions U.S.-based scientists are chasing, such as the effects of housing discrimination on biodiversity and the race to detect the first exomoon. Uncertainty, failure and persistence are common themes.

While *Lost in Curiosity* succeeds in selling the science, it fails in taking a stand. At a time when politics is deeply entangled with science, the book is jarringly apolitical. Since the start of the second Trump administration, the federal government has drastically curbed funding for science, eroded the independence of research institutions, ignored facts and perpetuated misinformation. I was left wondering how the scientists I came to adore in this work may have been affected.

But it's clear from Kwok's storytelling that these passionate people are incredibly resilient. That gives me hope. If there's anyone who can keep sciencing through this chaotic political climate, it's them. ✖

Helping Teachers Bring Science to Life

Brianna Jenkins, a STEM educator with Jackson City Schools in Jackson, Ohio, is dedicated to helping her students engage with science through real-world investigation. By incorporating data collection, analysis and hands-on experiments into her classroom, she encourages students to think critically and develop practical problem-solving skills. As a recipient of a 2026 STEM Research Grant from Society for Science, Jenkins is excited to bring new technology into her classroom that will help students experience the scientific process firsthand.

Society for Science's STEM Research Grants program supports U.S. middle school and high school educators who are engaging students in authentic scientific research. Each grantee receives either $1,000 in preselected STEM research kits or up to $5,000 in funding to purchase specialized STEM equipment tailored to their classroom needs.

Each STEM kit includes two Arduino Starter Kits, two PocketLab Voyagers, a Water Monitoring Kit and a Soil Test Kit. These materials enable educators to introduce their students to hands-on, inquiry based projects focused on electronics programming, data collection, local ecosystems and soil analysis.

"On behalf of Jackson City Schools and our students, I am incredibly grateful to be a STEM Research Grant recipient," Jenkins says. "This grant allows our students to monitor and record real-time quantitative observations during class experiments. This is incredibly important in STEM education, as our students work through the rigorous steps of data collection and analysis using measurements they collect firsthand."

Since 2017, Society for Science has distributed over $1 million in STEM Research Grants to educators across all 50 states, and Washington, D.C., helping students across the country develop technical skills to explore their questions.

SOCIETY FOR SCIENCE

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Society for Science is a nonprofit organization best known for our award-winning journalism, world-class STEM competitions and STEM outreach programming. For more than a century, our mission has been to promote the understanding and appreciation of science and the vital role it plays in human advancement: to inform, educate, and inspire.

MAXIME MOUYSSET

REMOTE WORKERS FEEL ISOLATED. IN-OFFICE WORK IS NOT A FIX

BY SUJATA GUPTA

Remote work in the United States has skyrocketed since the COVID-19 pandemic. In 2019, people worked from home for just 7 percent of all their workdays; by 2023, that figure had quadrupled to 28 percent. As a remote worker for this publication since 2019, I have been part of this sea change. And I understand the feelings of both empowerment and isolation that can accompany working alone.

Whether remote work is, on balance, a perk or a problem is something remote workers like me often question. So, too, do employers, though we frequently seem to land on opposite sides of this polarizing issue. While I've been working from my home office some 800 kilometers from my company's Washington, D.C., headquarters, many institutions, including the federal government, have been issuing return-to-office mandates.

Social scientists have waded into the issue. Research on remote work and hybrid work — in which the week is split between working from home and the office — is still nascent, and signals point in many directions. Into that fray enters a big, new paper in *Science*.

Economist Natalia Emanuel and colleagues crunched the data from more than half a million U.S. workers across five surveys from 2011 to 2024 (excluding the peak pandemic years of 2020 and 2021). Compared with before the pandemic, the share of people who spend all day alone has increased by 4 percentage points. A third of that increase could be due to days worked remotely, the team says. About 1 in 3 workers are in remote-capable jobs.

These employees not only experience more isolation than their nonremote peers, but they also experience greater mental distress. People in this group are nearly 5 percentage points more likely to see a mental health professional (a result not linked to remote workers' increased ability to schedule visits during the workday). For the subset of remote-capable workers who live alone, distress levels are especially high. On a standardized distress scale, they went from experiencing distress "some of the time" on average before the pandemic to

experiencing distress "most of the time" after it.

Remote work can benefit some groups, like working caregivers and people with disabilities. So it's easy to assume their mental distress might go down when working at home. But that's not the case, says Emanuel, of the Federal Reserve Bank of New York in New York City. "We actually see the entire distribution shifting to more distress."

The researchers cannot show that increased isolation caused the increased mental distress. But even minor social interactions, such as chatting with a barista or smiling at a coworker, can improve well-being, other studies have found.

Not everyone is convinced by the new findings. "To the extent there are mental health downsides from isolation at home, there are more than offsetting upsides in terms of less stress, time with family and quality of life," says economist Nicholas Bloom of Stanford University.

Nor does the methodology let researchers differentiate between fully remote and hybrid workers, says sociologist Mattia Vacchiano of the University of Geneva. Depending on the evidence one looks at, a hybrid arrangement is either the remote work sweet spot or a source of family conflict for workers craving predictable workweeks, he says. With hybrid arrangements accounting for about 65 percent of all remote work, distinguishing outcomes between the groups is key.

If remote work is harming mental health, then action is needed to protect workers. But that action should not take the form of getting everybody back to the office, experts caution.

Commutes, for instance, incur social and mental health costs, says economist Cevat Giray Aksoy of King's College London. His research has found that working from home saves employees an average of more than an hour a day. But most of that extra time goes to more work and caregiving, Aksoy says. "The right lesson is not 'everyone back to the office' but 'design work better.'"

Employers and scientists need to move beyond simplistic narratives that remote work is good or bad, Vacchiano says. "This story [in the *Science* study] is simple. But the reality might not be."

The study hasn't resolved the conflicting signals emerging from research on remote work. But scientists are reaching some consensus on how to help workers. "Employers should treat social connection as part of the job design," says Emma Zang, a family and health policy expert at Yale who coauthored a perspective about the *Science* study.

1 in 3

The number of U.S. workers in remote-capable jobs

1 hour/day

The time saved by working from home

Hybrid employees might end up working in empty offices, speaking to geographically distant colleagues on Zoom. That's a task readily accomplished at home, experts say. Employers should also avoid "proximity bias," Aksoy says, or situations where people in the office get better assignments and faster promotions than those working fully remotely.

The key is coordination and consideration of individual needs, Zang says. Work may require both deep focus time and informal interactions that can foster innovation. Solutions can include having hybrid employees come in on the same days or at the same time on a given day. Such flexibility can meet the needs of workers in various life stages, from new hires and early-career workers who might benefit from more in-person time to workers with caregiving responsibilities. For fully remote workers who rarely interact with colleagues face-to-face, employers could subsidize membership to coworking spaces or support opportunities for in-person time with colleagues.

As for me, I began reporting this story nervous that the bottom line would be: Remote work harms mental health, so get back to the office. But I'm now reminded of something my mentor liked to say. "There are solvable problems and unsolvable ones. Focus on the former." Getting remote work right is a solvable problem—if employers take heed.

The global shift toward remote work is "one of the largest social experiments in modern history," Zang and Yale sociologist Rourke O'Brien write in their perspective. All the more reason to get it right. ✖

IN SPACE, THE UNKNOWNS ARE STILL TOO TRICKY FOR AI TO FLY SOLO

BY AARON TREMPER

In the movie *WALL-E*, some of the last Earthlings travel through the Kuiper Belt on the starship *Axiom*. For 700 years, a fully automated crew of robots has cared for them after our planet became uninhabitable. Running the ship is AUTO, an artificial intelligence system working to keep humans away — forever. Here at home, space agencies such as NASA are using AIs to explore the solar system. They are piloting rovers on Mars, preventing satellite collisions and training astronauts for spaceflight. But for now, spacefaring humans would be ill-advised to rely so heavily on AI.

Today's AIs are much more prone to mistakes and failure than what you see in fiction, says Daniele Gammelli, a roboticist at Stanford University who studies how to integrate AI systems into robots that interact with their environments.

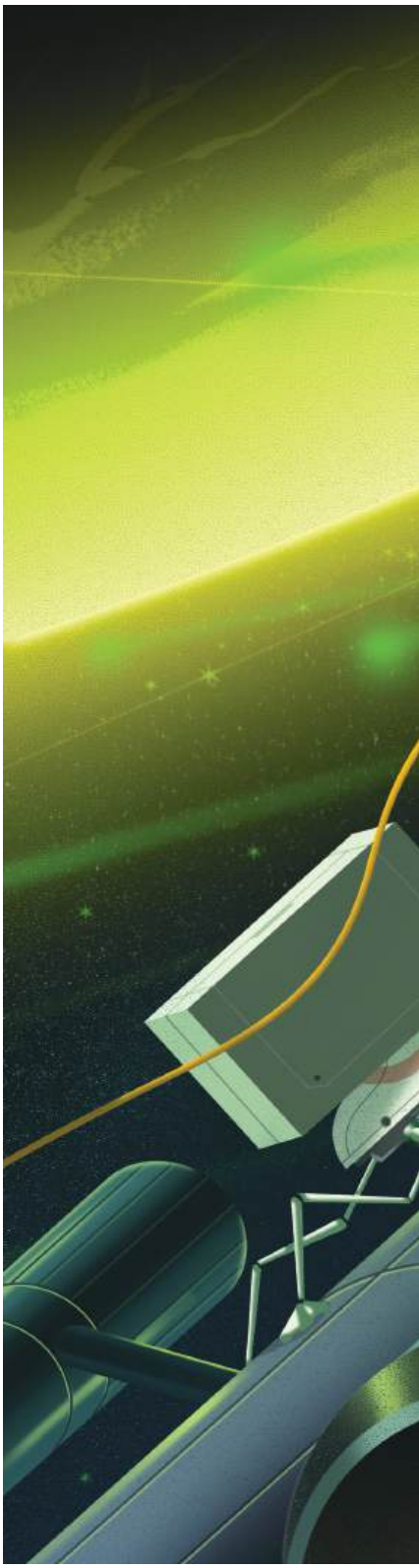
AI systems in space robots would need to complete multistep tasks in all sorts of scenarios without making up inaccurate information. In space, Gammelli says, "you have virtually no room for error."

The title robot in *WALL-E* is a trash-compacting machine that abandons his duties to follow another robot, EVE. His greatest strength is, arguably, his ability to handle change. The robot escapes a self-destructing pod using a fire extinguisher. When his wheel or eye malfunctions, WALL-E can replace the damaged part. All of this is learned from experience and done without additional programming.

Such versatility is an example of artificial general intelligence, AI that can think and learn across different situations and take on tasks that it hasn't been programmed for. AGI doesn't yet exist.

Adapting to unforeseen situations is a big goal for future spacefaring robots, Gammelli says. Between extreme temperatures, radiation and space debris, space is an ever-changing environment. "The kind of scenarios you are forcing on your robot are, by definition, things that nobody has ever seen," he says.

Today's AIs excel at single or closely related tasks, and with repetitive and predictable work. Their top skill "is processing a huge amount of data very efficiently," says Sanjoy Paul, a computer scientist at Rice University in Houston who researches how AI can assist



GLENN HARVEY

with space missions.

Martian rovers use this type of AI, all without human input. For instance, Perseverance employs AI algorithms to scan minerals and determine if rock samples are worth collecting. A human sorting through that kind of data could get overwhelmed, Paul says. "AI can cut through all the details ... and highlight those things for humans to take a look at."

To handle multistep tasks, nearly all space robots rely on "autonomy stacks," Gammelli says. Separate modules responsible for different actions are linked. One AI model might detect rocks or obstacles using cameras or sensors. This info would be passed on to another module to interpret and determine appropriate actions. Other modules would then carry out physical maneuvers to get the job done.

Aboard *Axiom*, robots handle everything. Custodial robots scrub and polish. Utility bots do repairs and maintenance. Hover chairs cart the ship's residents to their destinations. *Axiom*'s passengers live sedentary lives, watching videos and drinking food shakes.

In reality, "you still need humans in the loop," Paul says. While AIs continue to improve, they remain unpredictable. "If your life depends on it, would you really bank on AI? Probably not," he says.

Machines like rovers should eventually be able to make their own mini-goals that align with the overall mission, Gammelli says. That ability would allow bots to better handle unforeseen situations and free up humans to attend to more crucial tasks and decisions. "We want these robots to be as independent as possible," he says. Though maybe not as independent as mission-quitting WALL-E. ✖

A SEQUENCE OF ODD EVENTS

BY THE FANG-MACKEY FAMILY

In Spring 2025, we — Lilly Fang, Lester Mackey and our two children — noticed that something odd was happening to our family. First, Noah turned the odd age of 9 in May. Then Isaiah turned odd — or, more specifically, 7 — in August. Next, Lilly turned odd, 41, in November and finally, Lester turned odd, also 41, in February. This led us to wonder ...

1 Does every family experience an "odd cycle," in which the age of each member flips from even to odd in turn?

2 With a list of birthdays and birth years for all members of a given family, what is a simple way to determine whether (a) the family will experience an odd cycle and (b) in what order the members will flip from an even age to an odd one?

3 Why was Noah the first to turn an odd age and not Lilly (who was born first) or Lester (who has the earliest birthday each year)?

4 Is there a simple way to check 2(a) and 2(b) without calculating anyone's age?

FROM TOP: CAVAN IMAGES/CAVAN/GETTY IMAGES PLUS; SNEZHANA KUDRYAVTSEVA/ISTOCK/GETTY IMAGES PLUS; TATIANA SVIRIDOVA/MOMENT/GETTY IMAGES PLUS

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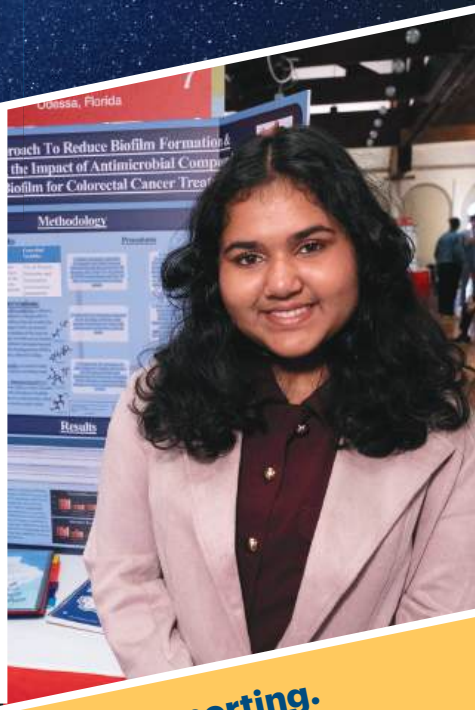

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