SCIENTIFIC AMERICAN

The Growing Threat of Landslides

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An abandoned plan to visit another star highlights the perils of billionaire-funded science

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SCIENTIFIC AMERICAN

FEATURES

ON THE COVER
Breakthrough Starshot, announced in 2016, promised $100 million in funding to send the first spacecraft to another star. The design called for tiny, superfast, chip-based spacecraft with "lightsails," spinning disks propelled by lasers. But the project's momentum has waned, and most of the money pledged never materialized.

Illustration by Eddie Guy

Photograph by Chris Gunn

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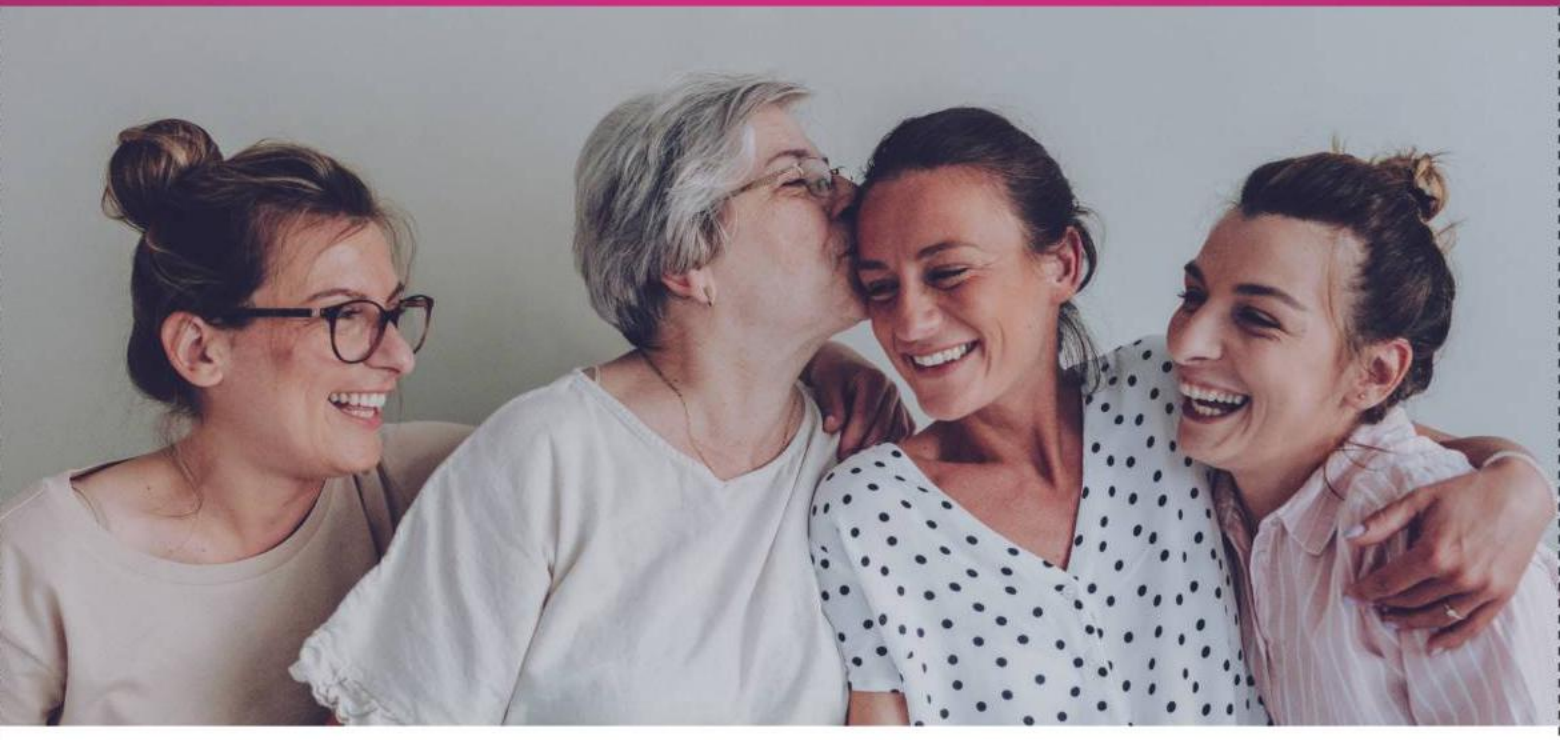

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Billionaire Science, for Better or Worse

A **POSTER-SIZE VERSION** of the front page of the very first issue of this magazine hangs in the lobby of Springer Nature's New York City office. I walk past it multiple times a day, and one line of text has stuck with me to the point that I keep bringing it up in meetings—so often that I fear my co-workers are getting tired of hearing it.

The line is at the top of the page, right under the words "*Scientific American,*" declaring the publication's mission: "The advocate of industry and enterprise, and journal of mechanical and other improvements." It grabs my attention because it reminds me that the first issue was created in large part to promote manufacturing and trade; it's pro-business, pro-capitalism, pro-wealth in ways that we don't see much in modern science journalism or in science as a whole.

Of course, there are some good reasons that in the 180 years since that first issue, science and industry have developed a complicated relationship, just as there are good reasons for some people to have a complicated relationship with capitalism; remember, *Scientific American* has been around so long that it predates Karl Marx and Friedrich Engels's *The Communist Manifesto* by three years.

During those two centuries, we've seen private industry give birth to entirely new areas of science. Edison and Westinghouse pioneered electrical engineering; thanks to IBM and Xerox and AT&T, I have the computer I'm writing this on. And corporate science hasn't just delivered new gadgets: the industrialization of drug discovery led to advances in pharmaceutical science and biomedicine that have saved countless lives.

Our cover story in this issue is a very 21st-century tale of how capitalism can help drive creation but doesn't always result in something quite as good as the invention of the transistor. Back in March 2017, another *Scientific American* cover story celebrated a project called Breakthrough Starshot, launched by Yuri Milner, a Silicon Valley billionaire who pledged to spend $100 million to send a cloud of tiny ships to Alpha Centauri. More than eight years later science journalist and *SciAm* contributing editor Sarah Scoles reveals that only a small fraction of that money ever materialized, and the project has effectively been lost in space (*page 24*).

It's a fascinating look at how billionaire science can go wrong, and I think it's full of important lessons about what may be one of the best hopes for science in the U.S. over the coming decades. Massive cuts to government funding of research and higher education are going to block off a lot of the traditional, academic paths to innovation, and even if those cuts are eventually reversed, it could take a very long time to rebuild what we'll have lost in the interim.

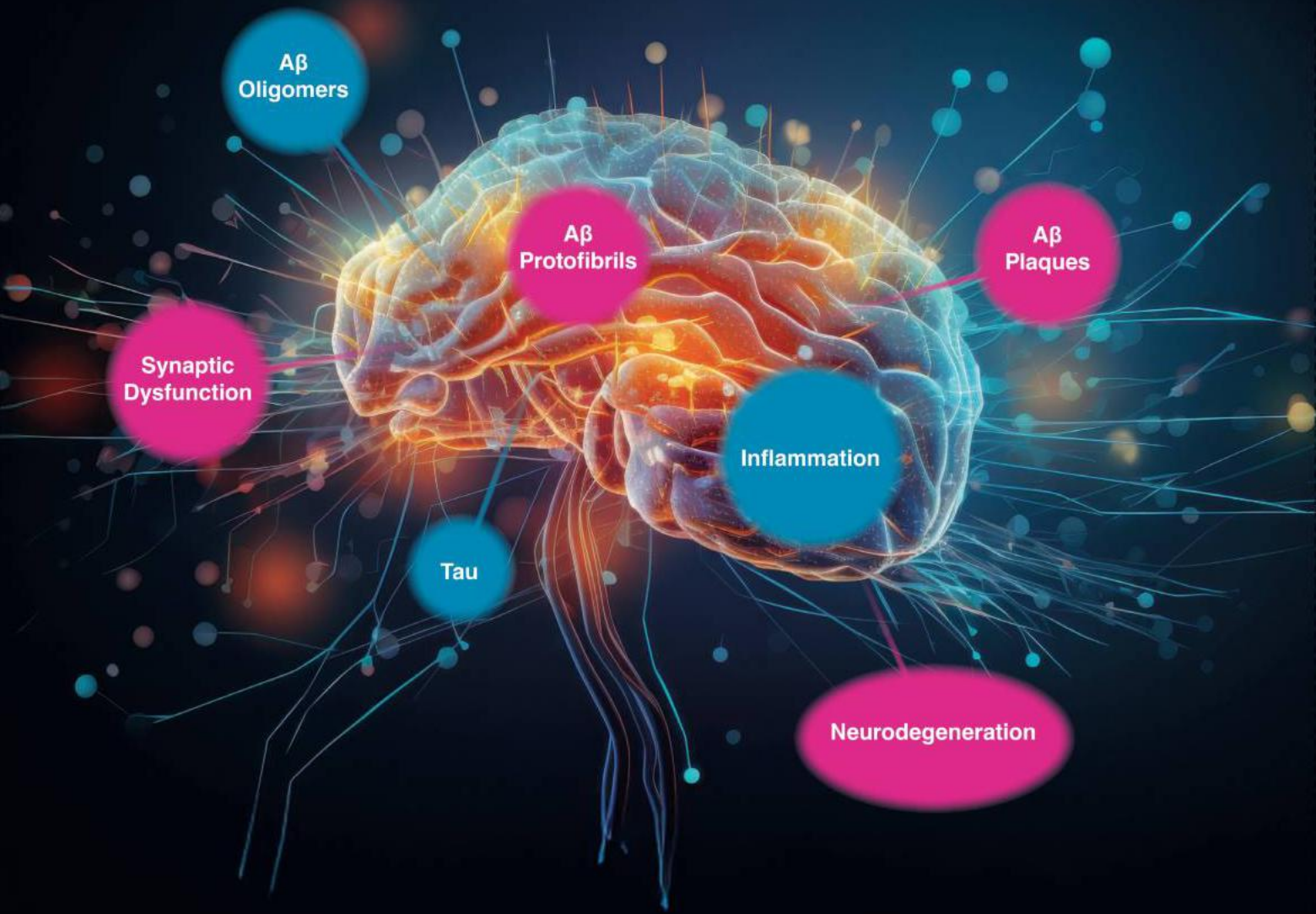
It seems to me, then, that a great many researchers are going to have to rely on business—and, yes, billionaires—in a way they haven't since before World War II. We know billionaire science can work: Milner's Breakthrough Listen project has already scanned thousands of stars in the most comprehensive search for alien intelligence to date, and Microsoft co-founder Paul Allen's Allen Institute has made big leaps in bioscience, including the creation of widely used open maps of gene expression in mouse and human brains.

As we enter a new era of slimmed-down, inadequately funded government science, the American scientific community is going to have to figure out how to maintain leadership in research and innovation—or whether that's even possible. Closer ties to business may be our best bet. We just have to learn how to tell when the money is too good to be true.

I'm curious about your thoughts on our cover story and about your take on billionaire science in general. E-mail us at editors@sciam.com to share your impressions, and visit us at ScientificAmerican.com to remain a part of the conversation. ●

David M. Ewalt
is editor in chief
of *Scientific American.*

Smoldering Alzheimer's Disease: A Constellation Of Pathologies



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CASSANDRA WILLYARD
DECODING BLOOD

PAGE S13

Alzheimer's disease has touched almost everyone's life in some way, says freelance journalist Cassandra Willyard, whose article in this issue's special report on Alzheimer's is about a recently approved diagnostic blood test for the disease. "It's a complicated subject because there's still controversy in the field about how it should be used correctly," she says. But Willyard, who has worked as a science writer for two decades, deliberately pursues stories with a lot of complexity. Sorting through nuance and presenting clear takeaways to readers is a satisfying challenge. For her entire career, "I've been very focused on medical topics like drug development and infectious diseases because I find it so fascinating and so relevant to what everyone goes through."

Watching federal funding for research get dismantled has been especially dismaying to Willyard because she's reported on the long trajectories of certain tests and treatments, such as the development of gene therapies and a possible vaccine for Lyme disease. "But talking to scientists helps me stay engaged and hopeful for the future," she says, "because they are excited about what they are learning."

REBECCA GELERNTER
THE DAWN OF POLAR BIRD MIGRATION

PAGE 42

Illustrator Rebecca Gelernter loves doing paleoart, "and I don't get to do it very often," she says. For this issue, she illustrated 10 ancient birds for a cladogram in the feature by Lauren N. Wilson and Daniel T. Ksepka about the dawn of bird migration. As Gelernter talks about skeletal reconstructions, it's easy to feel her joy at bringing fossil birds back to life. "I really like *A Field Guide to Mesozoic Birds and Other Winged Dinosaurs* [by Matthew P. Martyniuk] because it's structured like a bird guide, with notes on proportion and wingspan," she says.

Gelernter has been a "bird person" since she was 10 years old, and she studied ornithology in college. Then she discovered science illustration and enrolled in a graduate program, "which was one of the best decisions I've ever made."

The most fun part of the work is when Gelernter gets to problem-solve the gaps in knowledge, such as by designing plumage colors for dinosaurs. "I like adding a little crest here, some fun soft tissue there," she says. "Birds are just weird. They have all kinds of bizarre display structures, so it's hard to come up with something that's really unreasonable."

CHRIS GUNN
THE LIVES OF DEAD TREES

PAGE 52

For almost 25 years Chris Gunn (*above*) worked as a contract photographer for NASA, where he shot precious objects such as moon rocks brought back from the first Apollo landing and, as lead photographer for the project, captured three years of the James Webb Space Telescope's construction. That often meant working in clean rooms, with their rigid protocols and highly controlled conditions. So when Gunn entered the dense forests of Oregon to take pictures for journalist Stephen Ornes's story about a long-term study of decaying logs, it was an entirely different experience. "Having shot in locations with such stark geometric patterns for so long, going into the forest, initially I was like, 'Oh, my gosh, some of the trees are not straight,'" he says, laughing. "They are messing up my photograph!"

Gunn, who has lived in the Washington, D.C., area most of his life, had been seeking assignments that would both bring him closer to nature and communicate environmental change. "In so much of my previous work, I've been an outsider looking in on something, and this time I was really inside it," he says. Gunn likes his images to be super sharp, so he observed how light was falling through the canopy; controlling the exposure gave depth to his photographs. Although the subject was dead trees, "there was still so much life," he says. "It was magical from an imagery perspective."

LAUREN N. WILSON THE DAWN OF POLAR BIRD MIGRATION

PAGE 42

"Most kids go through a dinosaur stage," says paleobiologist Lauren N. Wilson. "I just never grew out of it." Wilson coauthored a feature with Daniel T. Ksepka in this issue about their discovery of the oldest known evidence for polar migration in birds. She says she found it fun to write about their research for a popular audience because she finally got to talk about what delighted her most: "The baby-bird fossils were *so* cute. Most of the bones I worked on were two millimeters or smaller."

When Wilson, who is now a Ph.D. student at Princeton University, went to Alaska for graduate school, she thought she'd spend her first summer identifying and describing bird fossils alongside Ksepka. "We started to get a good sense that some of this stuff was pretty significant," she recalls. "I e-mailed [Ksepka] nonstop for the next three years, saying, 'Wow, this is weird, look at this, what do you think?'" The result of their fieldwork was a "holistic study not just of the birds but of the whole ecosystem," she says.

Stories like this one are important, Wilson says, because we wouldn't be able to understand how abnormal the rate of global warming is today if we didn't know how things happened in the past. "We learned that birds have been nesting in the same area in Alaska for 73 million years," she says. "Then humans show up, and in the blink of an eye we're endangering that."

Chris Gunn

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May 2025

SPEEDY COMETS

"Dark Comets," by Robin George Andrews, describes a group of objects in our solar system with "unexplained" acceleration. That made me wonder: Is it possible that while dark energy appears uniform over galactic scales, it is actually more discrete at smaller scales such as that of the solar system? And if such packets of dark energy were to occur near or on one of these "dark comets," could they be giving those unusual bodies the mysterious acceleration? I guess that wouldn't really answer anything until we better understand dark energy, but it would be a place to look for clues.
MICHAEL K. MARTIN *VIA E-MAIL*

Andrews's article notes that the cause of the observed acceleration of some items passing through our solar system is unclear. The article considers whether outgassing might induce the acceleration, but no strong evidence for this option has been found. Unconsidered is another possible influence relating to magnetic fields.

An object that is made up of fused metals might accumulate an electrical charge. A charged item that travels through strong magnetic fields, as could be encountered close to the sun or Jupiter, might be expected to display acceleration without any visible emissions. Has anyone done the calculations to see if this might account for some of the anomalous acceleration?
SCOTT T. MEISSNER *VIA E-MAIL*

ANDREWS REPLIES: *Seeing as dark energy appears to be responsible for accelerating the expansion of the universe, it's not unreasonable to wonder whether it's giving certain comets an extra speed boost, too. But Martin is right: we don't really understand dark energy, so invoking it to explain these weird zigzagging objects is probably a dead end—and I'm not sure dark energy operates on such a specific and tiny scale.*

I like Meissner's idea that these objects might be pushed by an electromagnetic force! One issue, though, is that of composition: highly metallic asteroids, including ones like Psyche (which is potentially an exposed iron core from a destroyed planet), don't appear to be affected by the magnetic field of the sun or Jupiter in this way. So this is probably not the explanation for dark comets.

MINING THE SEAFLOOR

I read "Deep-Sea Mining Begins," by Willem Marx, with anger. It seems the problem of deep-sea mining is not only an economic or political one; it is also an ethical and moral one. Too many people think only of their own livelihood. They care about their present lives but not about Earth's future. We must boost our society's moral standards.
HIROYUKI UCHIDA *TOKYO*

REPTILE CALIBRATION?

"Turtle Dance," by Jack Tamisiea [Advances], observes that loggerhead sea turtles dance when they find food and also form lifelong memories of Earth's magnetic field specific to such feeding grounds. If a sea turtle can navigate with our planet's magnetic field, it must have a magnetic sensor. It seems likely to me that a sea turtle's "dance" creates the lifelong memory by finding which body orientation maximizes the response in its magnetic sensor, similar to the calibration of a magnetic fluxgate compass.
JAMES R. McGEE *LAKE ELMO, MINN.*

WHALE OF A PROBLEM

"Shape Shift," by Rachel Crowell and Violet Frances, presents mathematicians' descriptions of beautiful and intriguing forms and surfaces. Among them, Sarah Hart of Birkbeck, University of London, discusses cycloids—curves traced out by a point on a circle's circumference as it rolls along a line—and describes an interesting property concerning the descent of a particle along a cycloid: under gravity, the particle "will reach the bottom in the same time no matter where on the curve it is released."

I wonder whether Hart is aware that in Herman Melville's 1851 novel *Moby-Dick,* the character of Ishmael observes and empirically solves this very problem while scrubbing a large iron pot used to render oil from whale blubber: "I was first indirectly struck by the remarkable fact, that in geometry all bodies gliding along the cycloid, my soapstone for example, will descend from any point in precisely the same time." Melville was an intuitive mathematician and an extremely acute observer of everything.
"CWITHAL" *VIA E-MAIL*

GENDER AND OPPRESSION

In "Romantic Hopes" [Advances; June], Clarissa Brincat reports on a review of past studies that suggests that men place more importance on romantic relationships than women do because they "expect to gain more."

"The problem of deep-sea mining is not only an economic or political one; it is also an ethical and moral one."

—HIROYUKI UCHIDA *TOKYO*

The article quotes psychologist Mariko Visserman as noting that the paper explains "how gendered norms and experiences early in life can set the stage for the differences between men's and women's relationship benefits and vulnerabilities later on." It is not surprising that early experiences set up adult patterns. What is surprising is that the article never mentions the form of society that produces the cultural "gendered norms" from which those early experiences and relationship patterns arise: patriarchy.

As a now retired psychotherapist with a master of social work degree, I'd say it is no wonder that female expectations of romantic relationships are not very high. Most women are still often harshly judged by men who don't believe they are entitled to enjoy the same freedoms, including sex, as men do. The gendered norms of patriarchy give rise to men who seek to dominate women and use them as sex objects. Romantic relationships should be fun and exciting for women and men in our sociable species. But from adolescence, women are "hit on" at school, in the workplace and in public with sexual innuendo, ridicule and unwanted sexual advances. This barrage of insults and pressure that approaches or, more likely, is the cultural norm naturally disheartens many women from actively seeking romance. Many women of course still seek romantic relationships, although most are understandably quite guarded and take time to trust. Feminism has been attacked for decades, if not longer.

Your article's omission of a mention of what seems to be the obvious determinative role of patriarchy in relationships is concerning and suggests the same norms may be at work covertly or overtly in your publication.

ELLIOTT LIBMAN *VIA E-MAIL*

ERRATUM

In "Dark Comets," by Robin George Andrews, an image of the asteroid Bennu was incorrectly identified as showing the asteroid Ryugu.

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the Agenda Setters

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THE NEW ERA OF HEALTHSPANS

This past May, **Scientific American Custom Media** hosted a dynamic forum exploring this topic with experts in clinical medicine, biotechnology, data science and drug discovery **(1)**.

Held in the stunning rooftop lounge of **Cure**, a New York-based health innovation accelerator, and in partnership with **Google Cloud**, **Phenome Health** and **the Buck Institute**, this round-table salon brought together unique perspectives on a timely and exciting topic. From the rise of new diagnostics to track and prevent chronic disease, to the social, economic and ethical considerations of a healthier elderly population, the conversation was far-ranging and provocative.

The evening began with opening comments from **Jeremy Abbate (3)**, VP and Publisher,

Scientific American, and **Seema Kumar (2)**, CEO of Cure.

Before the panel discussion, life science leaders **Lee Hood (8)** and **Eric Topol** set the tone on how sound science will drive the next revolution in longevity.

The lead program participants included:

Bill Fitzgerald (5), Head of Biotechnology Markets, Americas, Google Cloud

Klaus Romero (4), CEO, Critical Path Institute

Matt Kaeberlein (6), CEO, Optispan

Victoria Lee (7), Chief Medical Officer at PAVmed

This event officially launched a special issue going deep into the new science of Healthspans.

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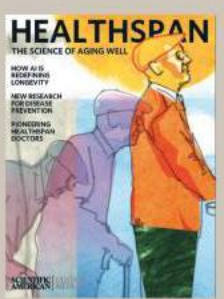


ADVANCES

ENGINEERING

Drink Deep

New tech pulls fresh water from the bottom of the sea

FROM CAPE TOWN TO TEHRAN to Lima to Phoenix, dozens of cities across the globe have recently experienced water shortages. In the next five years the world's demand for fresh water could significantly outpace supply, according to a United Nations forecast. Now several companies are turning to an unexpected source for a solution: the bottom of the ocean.

Called subsea desalination, the idea is to remove the salt from water in the deep sea. If it worked at scale, the technology could greatly alleviate the world's water-access problems.

Costs and energy requirements have kept desalination from going mainstream in most of the world. Early desalination involved boiling seawater and condensing the steam, a purely thermal method that used loads of energy. This approach was later replaced by multistage flash distillation, in which temperature and pressure "flash" salt water into steam. In the past 25 years reverse osmosis has become more common. This process uses high pressure to push seawater through a membrane with holes so small that only water molecules squeeze through, leaving salt behind.

Reverse osmosis is more efficient than distillation, but it takes a lot of energy to pressurize millions of gallons of seawater to force it through filters. What if we could let that movement happen naturally by harnessing the pressure hundreds of meters underwater?

That's the concept behind subsea desalination. Reverse osmosis pods are submerged to depths of around 500 meters (1,600 feet), where immense hydrostatic pressure does the hard work of separating water from salt. Purified water is then pumped back to shore. Far-fetched as the setup may sound, there are multiple prototypes already at work; the companies behind them aim to take cheap, large-scale desalination from pipe dream to reality.

One of these companies is Oslo-based Flocean. Its founder and CEO, Alexander Fuglesang, says there's no revolutionary new technology behind his business; it's "essentially a subsea pump cleverly coupled to existing membrane and filter technology." What's new is the energy savings—Flocean uses 40 to 50 percent less energy than conventional plants—and modular systems that can be deployed to many deep-sea locations without bespoke engineering.

The seafloor has other benefits, too. This region harbors

Companies are building desalination plants that take advantage of conditions far below the waves.

Piola666/Getty Images

fewer bacteria and other microorganisms than shallower depths do, and there is little local variation in temperature or pressure. "The deep sea is really predictable," Fuglesang says. "It's the same 365 days a year." This isn't the case at land-based plants, which have to deal with algae blooms, river runoff, storms and seasonal temperature changes. Plus, less chemical pretreatment of the water is needed at depth, and because the equipment is all underwater, there's no "not in my backyard" controversy over putting big, unsightly infrastructure near the seashore.

Despite its advantages over land-based plants, subsea desalination has several hurdles to clear before scaling to commercial levels. For starters, it's still too expensive. Land-based desalination is several times more costly than pulling water from aquifers or lakes, even at gigaplants in the Middle East that benefit from abundant solar power and large economies of scale. So even if subsea technology can undercut the cost of land-based desalination by 40 percent, it will still be a pricey way to obtain potable water.

Plus, "we need to remember that once the water is desalinated, it still needs to be pumped up from depths of up to 600 meters," says Nidal Hilal, founding director of New York University's Water Research Center in Abu Dhabi, who has studied water-treatment engineering for more than 30 years. "Early pilot tests show promise, but the technology has yet to be proven at large scale."

Affordable renewable energy will make subsea desalination more viable. Improvements in technology will also help. Hilal's research group, for example, is developing electrically conductive reverse osmosis membranes that keep themselves clean by repelling salt ions and impurities. They would extend maintenance intervals, which Fuglesang says might be two to three years with existing membranes.

Although proponents of the technology say it would have little effect on undersea life, others urge further research to gauge its impact on marine ecosystems. "Many organisms thrive at 500 meters' depth," says Adina Paytan, a professor affiliated with the Institute of Marine Sciences at the University of California, Santa Cruz. The twilight zone—which extends from 200 to 1,000 meters below the surface—is not only home to organisms such as whales, squid and jellyfish but "extremely important for many ocean processes, such as the carbon cycle and nutrient cycling," Paytan says. Companies will need to ensure that their water intake and salty brine by-product don't harm marine life or significantly alter these processes.

Given the depths required, subsea desalination won't work in just any seaside location. "Many coastal cities lie on wide continental shelves, meaning deep water is far offshore," Hilal says. Coastlines with steep drop-offs are ideal because shallow shelves would require long pipelines, adding to capital and operational costs.

Fuglesang isn't worried about technical or engineering hurdles; he says the industry's biggest challenge will be aligning customers, governments and financial partners. Flocean is working on what will be the world's first large-scale subsea desalination plant, off the coast of Norway, and is finalizing a contract to supply water to an industrial facility in 2026. Netherlands-based Waterise has also secured its first industrial customer, with plans to start building a plant in the Red Sea's Gulf of Aqaba later this year, and Bay Area–based OceanWell is testing its prototype near Los Angeles.

Long-term government contracts will most likely be needed for subsea desalination to really take off, and they may prove elusive. "The water-infrastructure industry is quite conservative," Fuglesang says, noting that because new projects are so expensive, "nobody wants to be first" to go all-in on a new technology.

Hilal says he believes subsea desalination could go mainstream and supply water to entire cities. But, he adds, "reaching true city scale will take time, conceivably a decade or more."

— Vanessa Bates Ramirez

SPACE

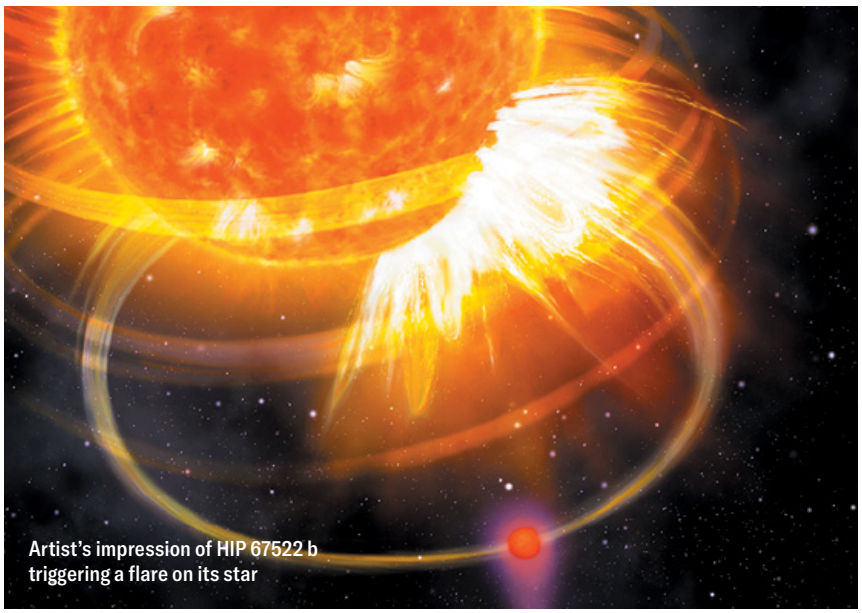
Self-Destruct

This planet triggers flares on its star—spelling its ultimate doom

STARS OFTEN WHIP their planets with stellar winds and radiation, pull them ever closer with gravity and sear them with heat. But a newfound planet exerts an unexpectedly strong—and ultimately self-destructive—influence on its star in return.

The star, HIP 67522, is slightly larger than our sun and shines roughly 408 light-years away in the Scorpius-Centaurus association. It's 17 million years old, a youngster by stellar standards, and has two orbiting planets that are even younger. The innermost of these two planets, a Jupiter-size gas giant called HIP 67522 b, orbits HIP 67522 at a distance of less than 12 times the star's radius—about one-seventh Mercury's distance in solar radii from our own sun. This in-your-face proximity, combined with HIP 67522's volatile teenage nature, creates a spectacle that astronomers have never seen before: a planet that triggers powerful flares on the surface of its host star, leading to the planet's own slow destruction.

"In a way, we got lucky," says Ekaterina Ilin, an astrophysicist at the Netherlands Institute for Radio Astronomy (ASTRON), who led a study published in *Nature* on the HIP 67522 system. "We took all the star-planet systems that we knew of and just went ahead looking for flares—sudden, intense bursts of radiation coming from the star's surface." Parsing the data gathered by two space-based telescopes, NASA's TESS (Transiting Exoplanet Survey Satellite) and the European Space Agency's CHEOPS (Characterizing Exoplanet Satellite), Ilin's team noticed that HIP 67522's flares seemed to be synchronized with its closest planet's orbital period. And those flares were gigantic—"thousands of times more energetic than anything the sun can produce," Ilin says.



Artist's impression of HIP 67522 b triggering a flare on its star

Danielle Futselaar (www.artsource.nl)

The orbiting gas giant most likely sparks these powerful flares by perturbing the star's strong magnetic field lines as it passes by. This disruption sends waves of energy downward along the lines—and when those waves meet the star's surface, a flare bursts out. The star's magnetic loops are "almost like a spring waiting to be let go," Ilin says. "The planet's just giving it this last push." Based on the team's observations, HIP 67522 b sets off a flare once every Earth day or two.

This action has severe consequences for the planet: Ilin estimates the unlucky gas giant is hit by six times more flares than it would be if it weren't triggering extras, and it's blasting away its own atmosphere. At this pace, Ilin's team says, HIP 67522 b will shrink from Jupiter's size to Neptune's or smaller in about 100 million years. "Flaring might cut the lifetime of the planet's atmosphere in half," she says.

Researchers had suspected this type of star-planet interplay might occur, but they had never previously confirmed it, says Antoine Strugarek, an astrophysicist at the Institute of Research into the Fundamental Laws of the Universe, which is part of the French Alternative Energies and Atomic Energy Commission. "This is the first time we see very convincing evidence such interaction has been actually detected," says Strugarek, who was not involved in the study.

It is too early to draw far-reaching conclusions from this first example of the phenomenon. As a next step, Ilin says, researchers can compare HIP 67522 b with the other planet in the system (orbiting a bit farther from the star) to calculate how much mass the more closely orbiting world loses through this process compared with the more distant one, which is probably hit with only random flares.

Another unanswered question is exactly how the flare triggering works. "Is it a wave [of magnetic energy] that propagates from the planet?" Ilin wonders. She suggests that what happens could be like an effect seen on the sun: smaller solar flares sometimes perturb nearby magnetic loops and tip them over the edge to snap and produce a larger flare.

But perhaps the most important question is how common the phenomenon is. For now Ilin wants to try to find more systems in which planets induce stellar flares that scientists can study. "Once we figure out how it works, we can turn it into a planet-detection technique," she says. Instead of searching for the planets themselves, researchers could find stars that flare following a certain pattern—suggesting they, too, might have planets with a self-destructive bent. —*Jacek Krywko*

ANIMAL BEHAVIOR

People Watching

Our social voyeurism may have deep evolutionary roots

Macaques

Whitworth Images/Getty Images

THE HUMAN FASCINATION with watching others—whether through reality TV, Instagram stories or overheard drama—is often dismissed as nosiness. But new research suggests this impulse may be a social survival tool dating back millions of years.

To explore the origins of social curiosity, Laura Lewis, a comparative and developmental psychologist at the University of California, Santa Barbara, and her colleagues studied how human children between four and six years old from San Francisco's Bay Area and adult chimpanzees responded to certain videos showing members of their respective species. The results, published in the *Proceedings of the Royal Society B*, show that both groups preferred watching social interactions over scenes involving solitary individuals—even forgoing small rewards to see the former.

"These findings demonstrate that social information is important, rewarding and valuable for humans and other primate species," Lewis says. "It suggests that social information was also important for our shared primate ancestors who lived around 25 million years ago and that for millions of years it has been adaptive for primates to gain social information about those around them."

PHYSICS

Unruly Beauty

Mysterious antimatter physics discovered at the Large Hadron Collider

MATTER AND ANTIMATTER are like mirror opposites: except for their electric charge, they are the same in every respect. Well, almost the same—very occasionally matter and antimatter behave differently from each other, and when they do, physicists get very excited. Now scientists at the world's largest particle collider have observed a new class of antimatter particles breaking down at a different rate than their matter counterparts. The discovery is a significant step in physicists' quest to solve one of the biggest mysteries in the universe: why there is something rather than nothing.

Everything around us is made of matter—the stars, planets, people and things that populate our cosmos are composed of atoms that contain only matter and no antimatter. But it didn't have to be this way. Our best theories suggest that when the universe was born, it had equal amounts of matter and antimatter, and when the two made contact, they annihilated each other. For some reason, a small excess of matter survived and went on to create the physical world. Why? No one knows.

So physicists have been on the hunt for any sign of a difference between matter and antimatter known in the field as a violation of charge conjugation–parity symmetry, or CP violation, that could explain why some matter escaped destruction in the early universe.

Physicists working on a machine called the LHCb experiment at the Large

Magnet for CERN's LHCb particle detector

Among the children (but not the chimps), the researchers noticed another pattern: as they grew older, boys became increasingly interested in watching scenes of social conflict, such as a tug-of-war over toys or one child crying while another yelled, whereas girls developed a stronger preference for positive interactions, such as play or hair grooming. The researchers hypothesize this result could reflect differing socialization patterns and evolutionary pressures particular to humans.

Another recent study, published in *Animal Cognition*, explored peer-watching behavior in long-tailed macaques. Both female and male macaques showed more interest in aggressive interactions than in peaceful grooming, and both paid more attention to videos of familiar individuals. The study's lead author, Liesbeth Sterck, a primatologist at Utrecht University in the Netherlands, says the latter behavior mirrors the way humans are drawn to the social lives of people they recognize—whether family, friends or movie stars. Interest in aggressive interactions, which are likely to reveal shifts in dominance or signal potential threats, echoes findings that humans are especially attuned to watching conflict in media. "Keeping track of the power balance in your own group likely has prime value for primates, including humans," Sterck says.

Gillian Forrester, who studies comparative cognition at the University of Sussex in England and was not involved in either study, says social attention is key to maintaining a good reputation. In ancient humans and other primates, reputational damage can bar access to food and mates, incite physical confrontations and, in extreme cases, lead to potentially fatal ostracism. With so much at stake, primates evolved to keep a close eye on group members. "Modern humans retain this keen attention to other people's social interactions as an evolutionary adaptation," Forrester says—so people watching might just pay off.

—Clarissa Brincat

CERN/Science Source

Hadron Collider (LHC) near Geneva announced in *Nature* that they have measured CP violation for the first time in baryons, the class of particles that includes the protons and neutrons inside atoms. Baryons are all built from triplets of even smaller particles called quarks. Previous experiments dating back to 1964 had revealed CP violation in meson particles, which, unlike baryons, are made of a quark-antiquark pair. In the new experiment, scientists observed that baryons made of three quark types—an "up" quark, a "down" quark and one of their more exotic cousins, called a "beauty" quark—decay more often than baryons made of the antimatter versions of those same three quarks.

"This is a milestone in the search for CP violation," says Xueting Yang of Peking University, a member of the LHCb team that analyzed the data behind the measurement. "Because baryons are the building blocks of the everyday things

around us, the first observation of CP violation in baryons opens a new window for us to search for hints of new physics."

The LHC is the only machine in the world that can summon sufficient energies to make baryons containing beauty quarks. It does so by accelerating protons to nearly the speed of light, then smashing them together in about 200 million collisions every second. As the protons dissolve, the energy of the crash makes new particles spring into being.

"It is an amazing measurement," says theoretical physicist Edward Witten of the Institute for Advanced Study in Princeton, N.J., who was not involved in the experiment. "Baryons containing *b* [beauty] quarks are relatively hard to produce, and CP violation is very delicate and hard to study."

The 69-foot-long, 6,000-ton LHCb experiment can track all the particles created during the collisions and the many ways they can break down into smaller particles. "The detector is like a gigantic four-dimensional camera that is able to record the passage of all the particles through it," says LHCb spokesperson and study co-author Vincenzo Vagnoni of the National Institute for Nuclear Physics in Florence. "With all this information, we can reconstruct precisely what happened in the initial collision and everything that came out and then decayed."

The matter-antimatter difference scientists observed in this case is relatively small and fits within predictions of the Standard Model of particle physics, the reigning theory of the subatomic realm. This puny amount of CP violation alone cannot account for the profound asymmetry between matter and antimatter seen throughout space.

"The measurement itself is a great achievement, but the result, to me, is not surprising," says Jessica Turner, a theoretical physicist at Durham University in England, who was not involved in the research. "The observed CP violation seems to be in line with what has been measured before in the quark sector, and we know that is not enough to produce the observed baryon asymmetry."

To understand how matter got the upper hand in the early universe, physicists must find new ways that matter and antimatter diverge, most likely via particles that have yet to be seen. "There should be a new class of particles that were present in the early universe that exhibit a much larger amount of this behavior," Vagnoni says. "We are trying to find little discrepancies between what we observe and what is predicted by the Standard Model. If we find a discrepancy, then we can pinpoint what is wrong."

The researchers hope to discover more cracks in the Standard Model as the experiment keeps running. Eventually LHCb should collect about 30 times more data than used for this analysis; that will allow physicists to search for CP violation in particle decays that are even rarer than the one observed here. So stay tuned for an answer to why anything exists at all. —*Clara Moskowitz*

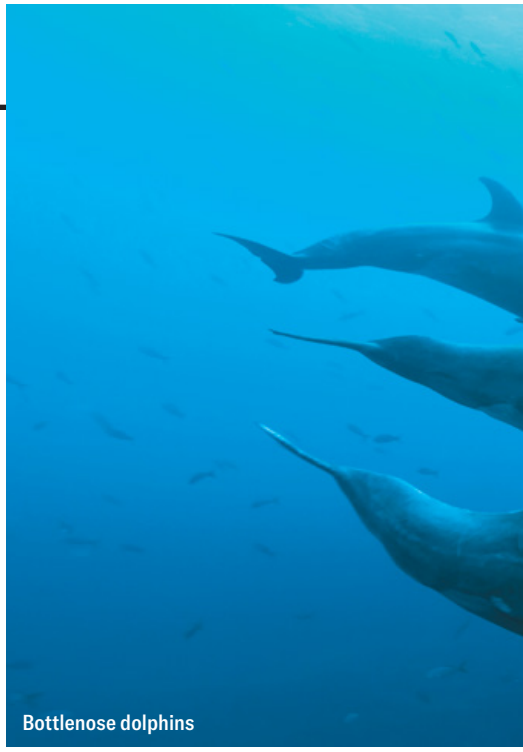
ANIMAL SENSING

Echolocation Touch

Dolphins' echolocation may be more like feeling than like seeing

IT'S MIDNIGHT in a pitch-dark parking lot. Trying to unlock your car, you fumble and drop the keys. You squat down and run your hand across the invisible pavement. To the left you feel a firm, rubbery tire. Reversing course, you pass over jagged pebbles and papery leaves. Finally your fingers discover—and instantly close around—a notched piece of metal. This kind of tactile exploration may be the closest we can get to imagining the experience of dolphin echolocation, say the authors of a study on dolphin brains that was recently published in *PLOS One*.

People often imagine echolocation as "seeing" with sound—experiencing auditory signals as a world of images like the ones our brains typically create from light perceived by our eyes. Like sonar devices, which turn sonic waves into visual representations, echolocators emit sounds and then decode spatial and textural information in the echoes that bounce back. And when Russian scientists inserted electrodes into the heads of dolphins and porpoises in the 1970s and 1980s, they reported detecting brain activity in the visual cortex while the animals heard sounds.



Bottlenose dolphins

Chris A. Crumley/Alamy Stock Photo

"It made a neat little story because you have visual and auditory [brain regions] right next to each other," says Lori Marino, a neuroscientist and president of the Whale Sanctuary Project, who was not an author of the new study but is mentioned in its acknowledgments section. She adds, however, that thanks to today's more precise technology, "the whole [research] landscape is changing." Although we still can't translate echolocation perfectly into human terms, the new findings suggest a better metaphor: "touching" with sound.

Dolphin echolocation works differently in the brain than human echolocation, which, for those who learn the skill, is processed primarily in the visual cortex. To pinpoint the neural mechanisms

behind the dolphin variety, the researchers compared preserved brains from three echolocating dolphin species with that of a sei whale, which is closely related but doesn't echolocate. They measured the diffusion of water molecules along nerve fibers—like cars driving along a highway, as Marino puts it—to better understand which parts of the brain interact in living dolphins and in sei whales. Contrary to the earlier Russian research, there seemed to be nothing exceptional occurring in the dolphins' visual cortex. Instead an entirely different stretch of neural highway caught the researchers' attention: the one linking the inferior colliculus to the cerebellum.

In dolphins, as in humans, the inferior colliculus is a relay point for auditory input after it enters the ear, and the cerebellum is where information from senses and bodily movements gets combined for rapid calculation of the body's next best move. "Anytime you need to move quickly, decisively and without consciously deliberating, your cerebellum comes alive," says Peter Cook, a comparative neuroscientist at the New College of Florida and senior author of the new study. He and his colleagues found a strong connection between these two brain structures in the dolphins but not in the sei whale. So just like touch does in humans, echolocation seems to rely heavily on the cerebellum's precise motor control and the tight feedback loop it promotes between sensation and motion—and less on the visual cortex. "Every time you move, you get different feedback," Cook says. "And every time the feedback changes, you change how you're moving. It's like this constant circle of sensory, motor, motor, sensory."

This process makes sense to lead author Sophie Flem, a master's student at the New College of Florida. If you need to constantly fine-tune your movements to home in on prey, Flem says, "it does seem intuitive that something like a cerebellum would really help." And there's another way in which echolocation seems more similar to touch than to vision: a dolphin's sonar beam is far narrower than our visual field. Whereas we take in 180 degrees at a glance, dolphins move their beam around and build spatial understanding gradually—like a human groping for dropped keys in the dark.

Still, it would be hubris to presume we know for certain what an animal's experience of echolocation is actually like. "There may be things other animals do for which there is no model in our sensory system," Marino says. "We just have to realize that."

—Cody Cottier

SCIENCE IN IMAGES

Neural Stretch

Scientists map a mouse's peripheral nervous system in unprecedented detail

YOUR PERIPHERAL NERVOUS SYSTEM (PNS) is crucial to navigating daily life. It lets you walk, controls your eye movements, and rings your brain's alarms when you step on a Lego brick. Yet researchers have never built a complete map of this essential network in any mammalian body.

Now a study published in *Cell* shows a complete, three-dimensional map of every single nerve fiber threading through a mouse. It completes the first-ever mammalian "connectome," a flowchart of an entire nervous system, beyond just the well-researched brain and spinal cord.

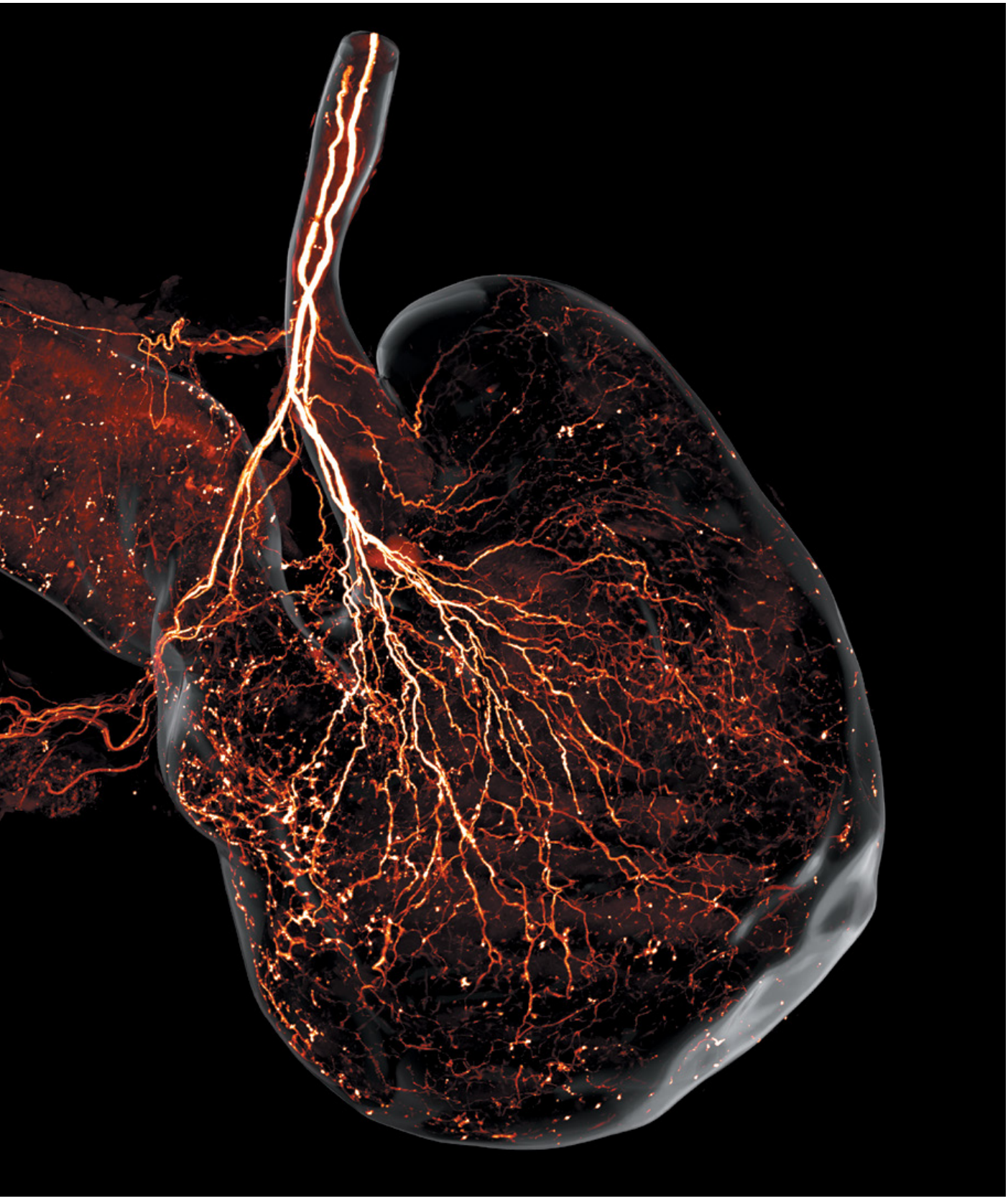
"Mapping of the PNS has been a neglected component of mapping the connectome in animal and human brain studies," says John Darrell Van Horn, a brain and data science researcher at the University of Virginia, who was not involved in the study.

The research team began by making the bodies of 16 mice as visually transparent as possible, removing fat, calcium, and other materials that block light. They then used a custom combined slicing tool and microscope to take images of each of the bodies 400 microns at a time, which took about 40 hours per mouse—providing data the researchers say would otherwise have taken months or years to collect.

The scientists genetically modified seven of the mice to have fluorescent neurons; as expected, this caused mostly the head to light up. In four of the mice, the team applied a technique called immunostaining, which uses antibodies to target and color specific proteins—in this case, those in the body's sympathetic nervous system, which controls "fight or flight" responses. In the remaining five mice, the researchers tested a method using viruses to measure the full length of nerve projections known as axons. They specifically focused on tracing the vagus nerve, which contains projections threading in from thousands of individual neurons. The team found that each vagus nerve fiber connected to only one organ in the gut, rather than branching to many different organs as some had predicted. (Its path through the stomach and part of the small intestine is visualized here.)

"By revealing the precise projection patterns and organ-specific targeting of different peripheral nerves, these maps will provide a structural framework for understanding how the PNS mediates body physiology," says co-author Guo-Qiang Bi, a biophysicist at the University of Science and Technology of China.

The researchers hope to apply this method to human tissue next to help plan precision surgeries. Van Horn says the work could also inspire therapies for nerve-related disorders such as chronic pain. "It moves us closer to the precision mapping of the entire mammalian connectome and the diseases that affect it, not just the part between the ears." —*Nora Bradford*



From "High-Speed Mapping of Whole-Mouse Peripheral Nerves at Subcellular Resolution," by Guo-Qiang Bi et al., in *Cell*, Vol. 188, No. 14; July 10, 2025

TECH

Search Broadly

The way you search the Internet can reinforce your beliefs—without you realizing it

PEOPLE'S VIEWS are becoming more and more polarized, with "echo chambers"—social bubbles that reinforce existing beliefs—exacerbating differences in opinion. This divergence doesn't just apply to political opinions; it also touches on factual topics, from climate change to vaccination.

And social media is not the sole culprit, according to a recent study published in the *Proceedings of the National Academy of Sciences USA*. It turns out that people use search engines in ways that confirm their existing beliefs, potentially amplifying polarization. A simple tweak to search algorithms, the researchers propose, could help deliver a broader range of perspectives.

Online participants were asked to rate their beliefs on six topics, including nuclear energy and caffeine's health effects. They then chose search terms to learn more about each topic. The researchers rated the terms' scope and found that between 9 and 34 percent (depending on the topic) were "narrow." For example, when researching the health effects of caffeine, one participant used "caffeine negative effects," whereas others used "benefits of caffeine."

These narrow terms tended to align with participants' existing beliefs, and generally less than 10 percent did this knowingly. "People often pick search terms that reflect what they believe, without realizing it," says Eugina Leung of Tulane University's business school, who led the study. "Search algorithms are designed to give the most relevant answers for whatever we type, which ends up reinforcing what we already thought." The same was true when participants used ChatGPT and Bing for searches aided by artificial intelligence.

When the researchers randomly assigned participants to view different results, they saw those results affect people's opinions and even behavior. For instance, participants who saw search results using "nuclear energy is good" felt better about nuclear energy afterward than those using "nuclear energy is bad." People who saw results using "caffeine health benefits," rather than "risks," were more likely to choose a caffeinated drink afterward.

Pointing out biases in the search terms had only a small effect on people's final opinions. But changing the search algorithm either to always provide broad results or to alternate between results obtained with broad and user-provided terms mitigated the effects of narrow searches.

The researchers "have thought through how these technologies could be optimized for the benefit of users," says Kathleen Hall Jamieson of the University of Pennsylvania, who studies political and science communication. For search technology to do what we need it to do, "this kind of research is very important."

Participants rated the broader results as just as useful and relevant as standard searches. "People are able to bring in different perspectives when they're exposed to them, which is encouraging," Leung says. "At least for the topics we tested." The researchers recommend implementing such strategies, possibly as "search broadly" buttons. "This would be really helpful," Leung says, but whether it will ever happen "is hard to predict." —*Simon Makin*

MEDICINE

Blocking Pill

An early human trial shows the safety of hormone-free male birth control

WHEN IT COMES TO BIRTH CONTROL, the market has long been skewed: female contraception comes in a variety of pills, implants, injections and devices, all approved by U.S. regulators, but condoms and vasectomies are the only male contraceptives available. Researchers have been chipping away at this problem for decades, and progress is finally ramping up. Now a male birth-control pill with an entirely new kind of contraceptive mechanism has been tested in humans.

In the first clinical trial of its kind, a nonhormonal oral contraceptive that reversibly stops sperm production was deemed safe for human use earlier this year. The daily pill, called YCT-529, blocks a vitamin A metabolite from binding to its receptor in the testes; this action prevents the chain of gene-expression changes that are required to start the sperm-making process. Safety results from the early phase 1 clinical trial were published in *Communications Medicine*.

The trial did not assess the pill's efficacy in reducing sperm counts, and the drug's developer, YourChoice Thera-

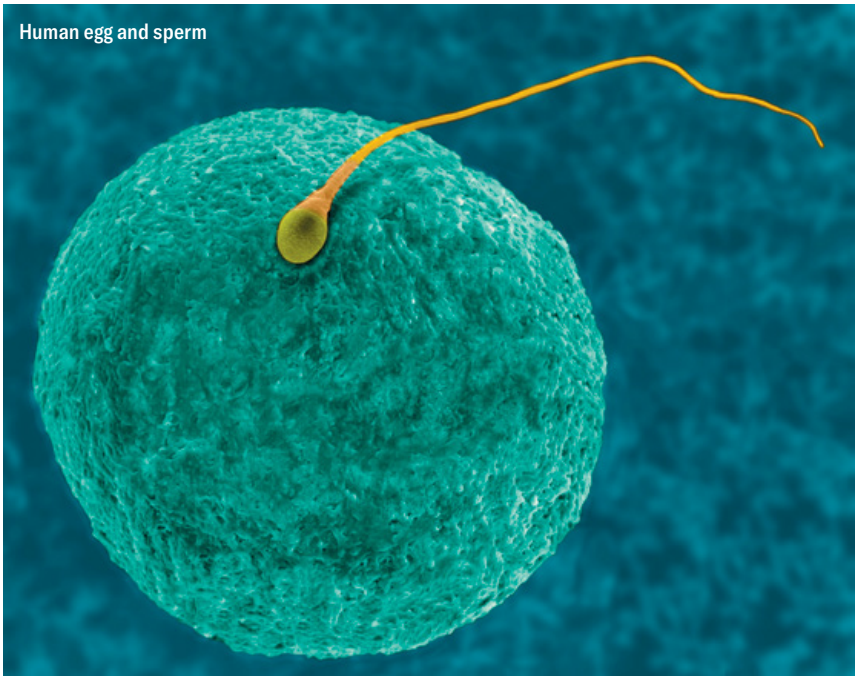
Illustration by Thomas Fuchs

peutics, is currently running trials to collect those data. But the safety finding is a crucial milestone, says Stephanie Page, an endocrinologist at the University of Washington School of Medicine, who wasn't involved in the study but has worked on other male hormonal contraceptives for more than 20 years. "We really need more reversible contraceptive methods for men," she says.

The small trial included 16 healthy men ages 32 to 59, all of whom had undergone a vasectomy—a common surgery in which the vas deferens ducts in the scrotum are snipped to prevent the release of sperm. Enrolling only such participants was an extra precaution to avoid the risk of permanently affecting fertility. No one has tested a nonhormonal male contraceptive pill in clinical trials before, says study lead author Nadja Mannowetz, co-founder and chief science officer of YourChoice Therapeutics. Using nonfertile participants worked for this trial because the team was evaluating not the drug's effectiveness but rather its tolerability and bioavailability (active levels that build up in the body), Mannowetz says.

Participants were split into two groups. In the first, people either received an initial dose of 10 milligrams (mg) of YCT-529 and then a second, 30-mg dose two weeks later or got a placebo each time. Participants in the second cohort either received a first dose of 90 mg and then a second dose of 180 mg two weeks later or always received a placebo. All participants took the pills after fasting. Four from each cohort were selected to return and take a third, 30-mg dose after a high-fat, high-calorie breakfast to see whether food might affect the drug's tolerability.

Across dosages, "we saw good and quick bioavailability," meaning the drug didn't rapidly break down in the body, Mannowetz says. On average, it took two to three days for the levels of available drug in the blood to decrease by half—a promising result that suggests the pill might be needed only once daily if it later proves effective at reducing sperm.



Human egg and sperm

Dennis Kunkel Microscopy/Science Source

Mannowetz anticipates that if the drug is eventually approved by the U.S. Food and Drug Administration, the final dosage that will hit stores will probably be closer to the higher amount tested, 180 mg, although follow-up trials will help scientists discern the exact optimal dose.

The research team didn't note any adverse side effects related to the drug. An advantage of a nonhormonal contraceptive medication is that, in theory, there's a smaller chance of certain side effects such as changes to sexual function, libido or mood, Mannowetz says.

The results are exciting and important, Page says—but she points out that this study was just one small trial. "I think it would be overstating the data to say they know much about side effects yet," she says. "*Every* medication on the market has side effects."

Several other reversible male birth-control methods are now in the clinical trial pipeline as well. The furthest along is NES/T, a combination of testosterone and the progestin medication Nestorone. Applied daily as a gel to the shoulders and upper arms, it is absorbed into the bloodstream through the skin. Like the YCT-529 pill, the gel targets sperm production, but it does so by increasing the amount of circulating testosterone and progestin—hormones that tell the brain to halt the production process. Researchers have just completed a larger, longer phase 2 clinical trial of NES/T to show effectiveness and hope to start a phase 3 trial soon, says Page, who has been involved in the gel's clinical research.

Users of a male contraceptive that targeted sperm production, such as NES/T or YCT-529, would need to take it daily for about three months before it took effect, because that's how long it takes the body to produce mature sperm cells. Sperm production would resume about three months after a user stopped taking the medication.

A couple of other candidates for hormonally acting daily male contraceptive pills are in early development. A hydrogel implant called ADAM is also being tested in early clinical trials. ADAM acts as a reversible vasectomy, physically blocking off the vas deferens to prevent sperm release until the implant is removed.

Studies show growing interest. One paper published in 2023 found that of more than 2,000 men surveyed in the U.S. and Canada, 75 percent were will-

ing to try novel contraceptives. And a report in 2019 found that among U.S. men ages 18 to 49 who had sex with women, did not have a vasectomy or beliefs that prevented the use of contraception, and did not want their partner to become pregnant, nearly 50 percent were "very interested." These stats line up with Page's experience in the field: "Men are very eager to have more reproductive agency and to participate in contraception," she says, and all these contraceptives in the pipeline could elevate individuals' and couples' decision-making about sex and reproduction.

—Hannah Seo

MATH

Why Knot

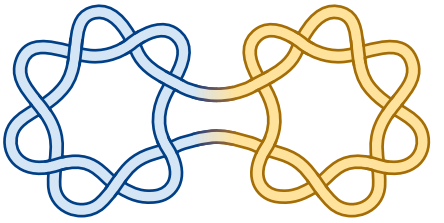
Mathematicians unravel a long-standing conjecture about knot theory

SCANNING THE CROWD at a fancy soiree may reveal a wide array of neckties, each fastened with a highly complex mathematical object masquerading as fashion. An entire field of mathematics is devoted to understanding mathematical knots, which one can obtain from any traditional knot by gluing the loose ends together. Mathematicians long believed that if you attach cut ends of two different knots to each other, the new knot will be just as complex as the sum of the individual knots' complexity. But researchers recently managed to find a knot that is *simpler* than the sum of its parts.

Knot theory is a branch of topology that has surprisingly practical applications, such as understanding how proteins coil DNA and how molecular structures remain stable. The theory's central question: How can we tell which knots are unique or which are the same as others? Mathematicians consider two knots the same if one can be manipulated to look like the other without being cut open—any knots you can produce with mere tugging and pulling are fundamentally the same. Only cutting and reconnecting to let two strands cross yields unique knots.

Using these careful manipulations, mathematicians assign each knot an unknotting number, which is the minimum number of cutting and reconnecting "moves" it would take to unravel the knot into a simple loop. This computation is often deceptively difficult. Many mathematicians assumed that if we construct a larger knot by joining together smaller ones whose unknotting numbers are known, then the quickest way to untangle the larger knot will be by simply undoing each piece independently. This idea that two conjoined knots' unknotting numbers can be added was first proposed as a conjecture by Hilmar Wendt in a 1937 paper and remained open for nearly a century. Until recently, "there was no clear way to prove this conjecture," says Mark Brittenham, a mathematician at the University of Nebraska–Lincoln, "and now we know why—because it's false."

For a preprint paper posted online at arXiv.org, Brittenham and his co-author, Susan Hermiller, a mathematician also at the University of Nebraska–Lincoln, tied



two knots that, when connected, require an unexpectedly small number of moves to undo. The mathematicians connected one knot with an unknotting number of three to its mirror image to form a larger knot. Instead of six moves, this "complicated mess of a [knot]" ultimately can be undone with only five maneuvers and possibly even fewer, Hermiller says.

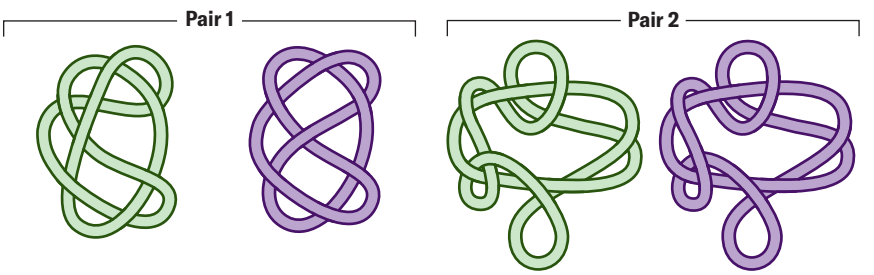
"This is quite surprising," says Rutgers University mathematician Kristen Hendricks, who was not involved in the study. "The result says that our notions of [knot] complexity could have problems."

So the next time you're battling a necktie or complicated scarf, take some comfort in knowing that even the simplest-seeming structures can conceal a world of unexpected mathematical complexity.

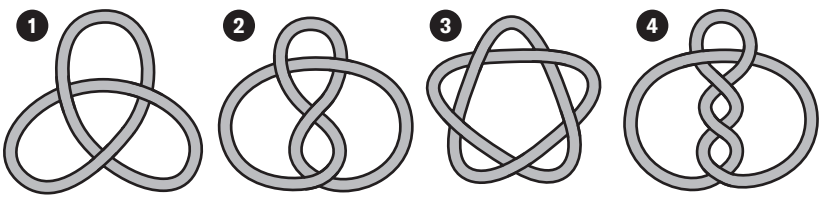
—Max Springer

Find the Unknot

MATH PUZZLE One string in each of the following pairs can be unraveled into a circle—the so-called unknot. Which one is it? *—Emma R. Hasson*



Challenge problem: The other two knots, which can't be completely unraveled, can each be made to look like one of the four most basic knots. Identify them!



Graphics by Amanda Montañez

For the solution, visit www.ScientificAmerican.com/games/math-puzzles

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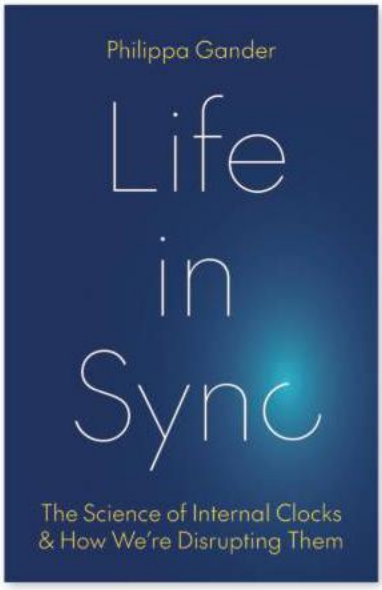
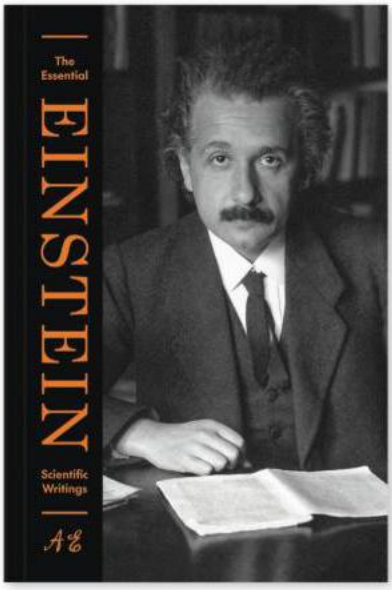
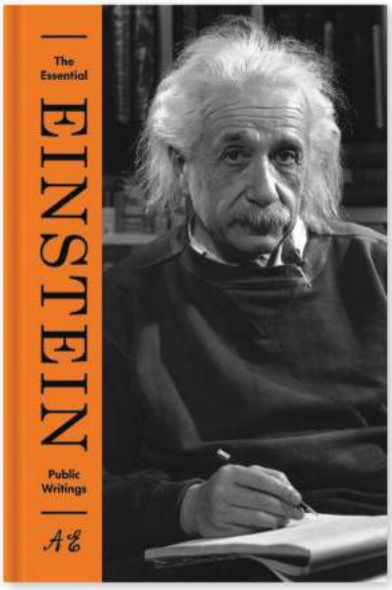
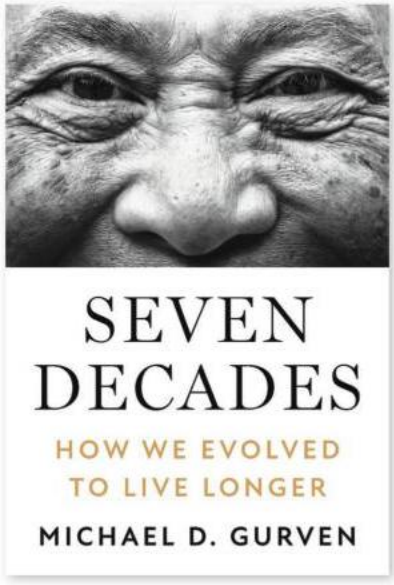


Why we need to reconnect with nature's biological rhythms—and rediscover the benefits of a good night's sleep

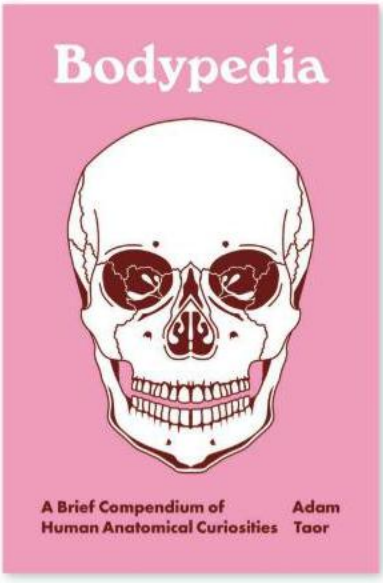




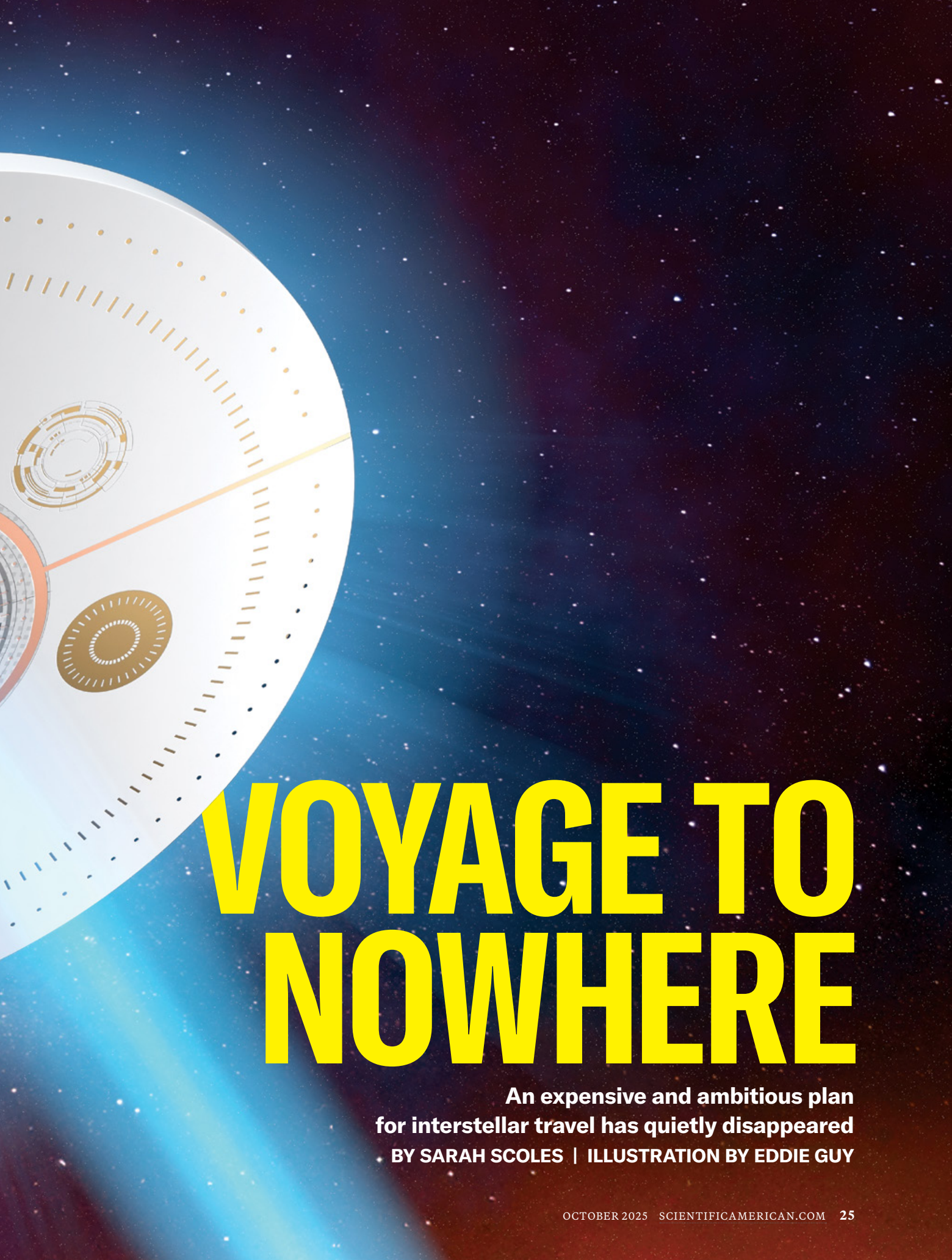
The ultimate collection of Einstein's scientific and public writings



An anthropologist uncovers new evidence for the evolutionary origins of human longevity—and explains why growing old is an opportunity, not a burden



Encyclopedic in nature and miniature in form, books in the Pedia series explore the wonders of the natural world, from A to Z



VOYAGE TO NOWHERE

An expensive and ambitious plan for interstellar travel has quietly disappeared

BY SARAH SCOLES | ILLUSTRATION BY EDDIE GUY

IN 2016 BILLIONAIRE YURI MILNER HOSTED a press conference at One World Observatory, the atrium topping the slick skyscraper at the center of the rebuilt World Trade Center complex. Milner had grown rich investing in tech start-ups, and now he wanted to spend some of that money on sending a spaceship to the stars.

He called the plan Breakthrough Starshot: a project that would eventually take human technology to another solar system. The idea was that high-powered lasers would propel tiny probes to 20 percent of the speed of light, impelling them with enough inertia to launch them toward the nearest star system, Alpha Centauri, within 20 years. Milner and his Breakthrough Initiatives, a group of space science research projects related to life in the universe, were pledging $100 million toward a proof of concept. At the event, Milner was joined by, among others, Mae Jemison, a former astronaut and head of 100 Year Starship, an interstellar research program funded by the Defense Advanced Research Projects Agency; Pete Worden, former director of NASA's Ames Research Center; and Stephen Hawking, world-famous physicist.

Zachary Manchester, currently an associate professor of robotics at Carnegie Mellon University, signed on for the project's early stages. He remembers it seeming incredible that he, then a wide-eyed 20-something, was at the top of a metropolis, hanging out with people he considered legends—people such as Freeman Dyson, a physicist best known for positing that advanced civilizations could eventually cloak their stars in megastructures that siphoned their power. Dyson was one of several scientific luminaries who were joining the project, including Nobel Prize winner Saul Perlmutter and Martin Rees, then the U.K.'s Astronomer Royal.

In short, the Starshot launch event was flashy. A video preview narrated by actor Seth MacFarlane was also flashy. The text that went along with the announcement? Flashy. "With current rocket propulsion technology, it would take tens or hundreds of millennia to reach our neighboring star system, Alpha Centauri," it read. "The stars, it seems, have set strict bounds on human destiny. Until now."

Milner's money wasn't quite an Apollo-project investment, but it was more than anyone had ever dedicated to interstellar travel, a field with a history of relatively little funding and a trail of projects that never reached the stars. In the 2010s DARPA and NASA founded the 100 Year Starship research program to figure out how to send humans light-years away in the next 100 years. Private research groups such as the Tau Zero Foundation and Project Icarus also launched initiatives. None of them have come to much. Maybe this time the goal was within reach. After all, besides the money itself, the big names attached to Breakthrough Starshot gave legitimacy to an endeavor that might otherwise have seemed fringe. The announcement made a splash in the press, including a cover story in this magazine.

But almost a decade later Breakthrough Starshot is conspicuously quiet. After the initial big bang the project seemed to whimper out. Now there are no more big announcements, no multi-institution meetings and no more funding. What remains is

Sarah Scoles
is a Colorado-based science journalist and a contributing editor at *Scientific American*.

Bryan Bedder/Getty Images for Breakthrough Prize Foundation

The Breakthrough Starshot announcement event in April 2016 included, from left, documentary writer and producer Ann Druyan, Zachary Manchester, Yuri Milner, Stephen Hawking, Freeman Dyson, Mae Jemison, Pete Worden, Avi Loeb and Philip Lubin.

confusion among even scientists working on Breakthrough Starshot about the project's status. According to an e-mail from Worden, Starshot's executive director, who declined an interview for this article, "We have put the program on hold and are working to transition portions to others."

Between 2016 and today scientists and engineers on the project did make progress toward the stars—or at least toward understanding what it would take to make progress toward the stars. But engineering an interstellar journey is almost ludicrously difficult. With today's rocket technology, it would take thousands of years to get to the nearest star. Processes and components need to be invented, iterated on and vetted, at great expense, most likely over decades. Sure, "$100 million sounds like a lot of money," says Edwin Turner, an emeritus astrophysicist at Princeton University and one of the first people to be involved in Breakthrough Starshot. "It's certainly more than pocket change for most of us, but it's not really very much for huge technological programs."

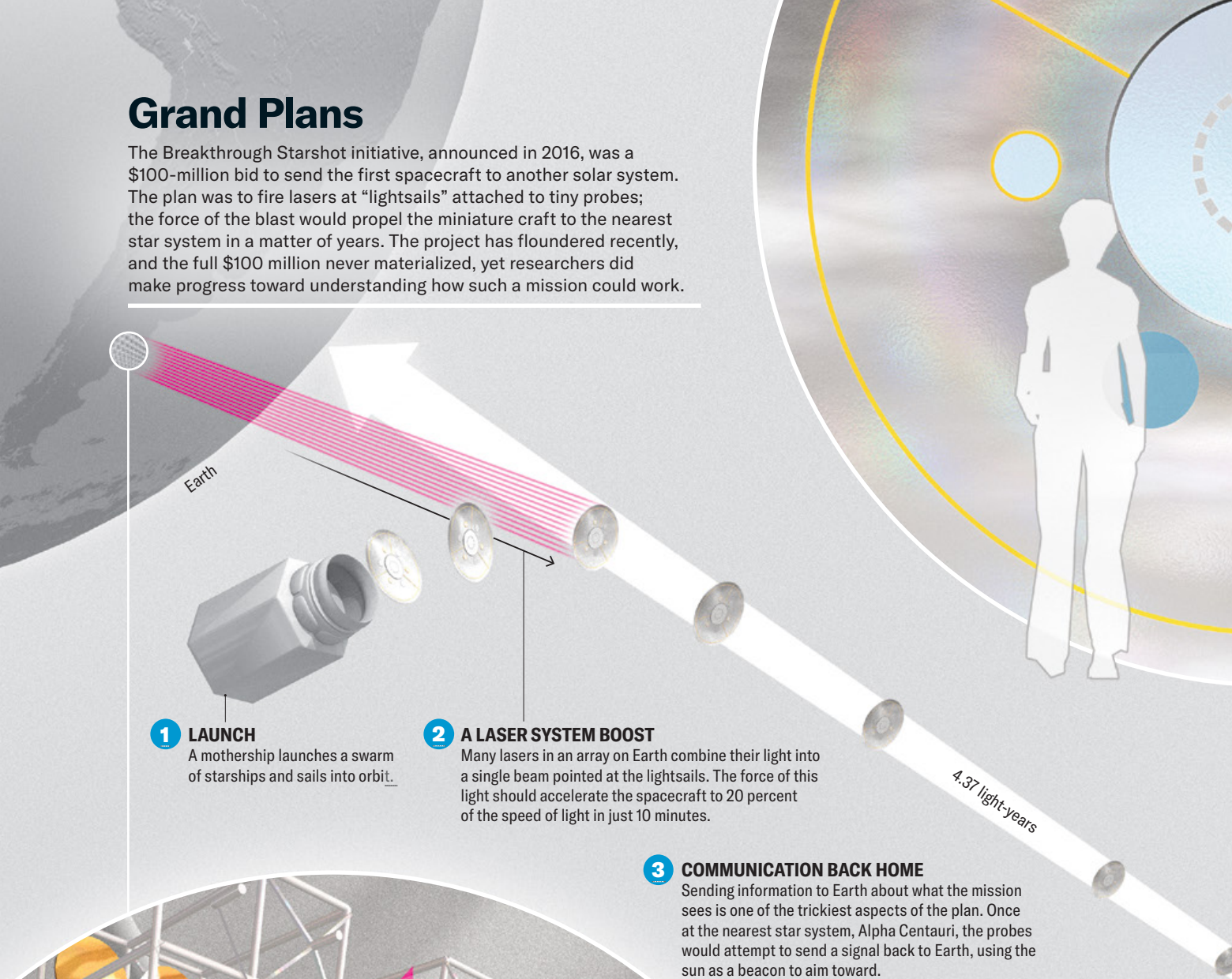
The total doled out, according to one insider, was far below $100 million anyway. The fact that most of the money never seems to have materialized means the case of Breakthrough Starshot isn't necessarily one of waste. But it's a study in the perils of relying on the ultrarich to fund science: when the guy with the billions is ready to move on, the whole project is off.

BREAKTHROUGH STARSHOT is based on a simple but technologically audacious concept: build a powerful set of lasers on Earth, and use them to propel "lightsails" on tiny spacecraft weighing about as much as a paperclip. A traditional rocket would carry the craft to space; once it was some 37,000 miles from Earth, the lasers would light up, shooting 100 gigawatts of power at the lightsails. Their combined photons would slam into the sails, powering them forward like wind on a sailboat. Ten minutes later the spacecraft would be zooming at 20 percent of light speed and already halfway to Mars—a journey that takes months with current technology. At that rate it would hit Alpha Centauri—specifically, Proxima Centauri, the closest star in the system—in a couple of decades. During its flyby Starshot would glimpse both the star and the Earth-size exoplanet known to exist in the star system. The craft would send a signal back to Earth before sailing on toward the rest of the Milky Way.

The basic idea of using light for propulsion dates to the 1920s, when Russian scientists Friedrich Zander and Konstantin Tsiolkovsky, pioneers of

Grand Plans

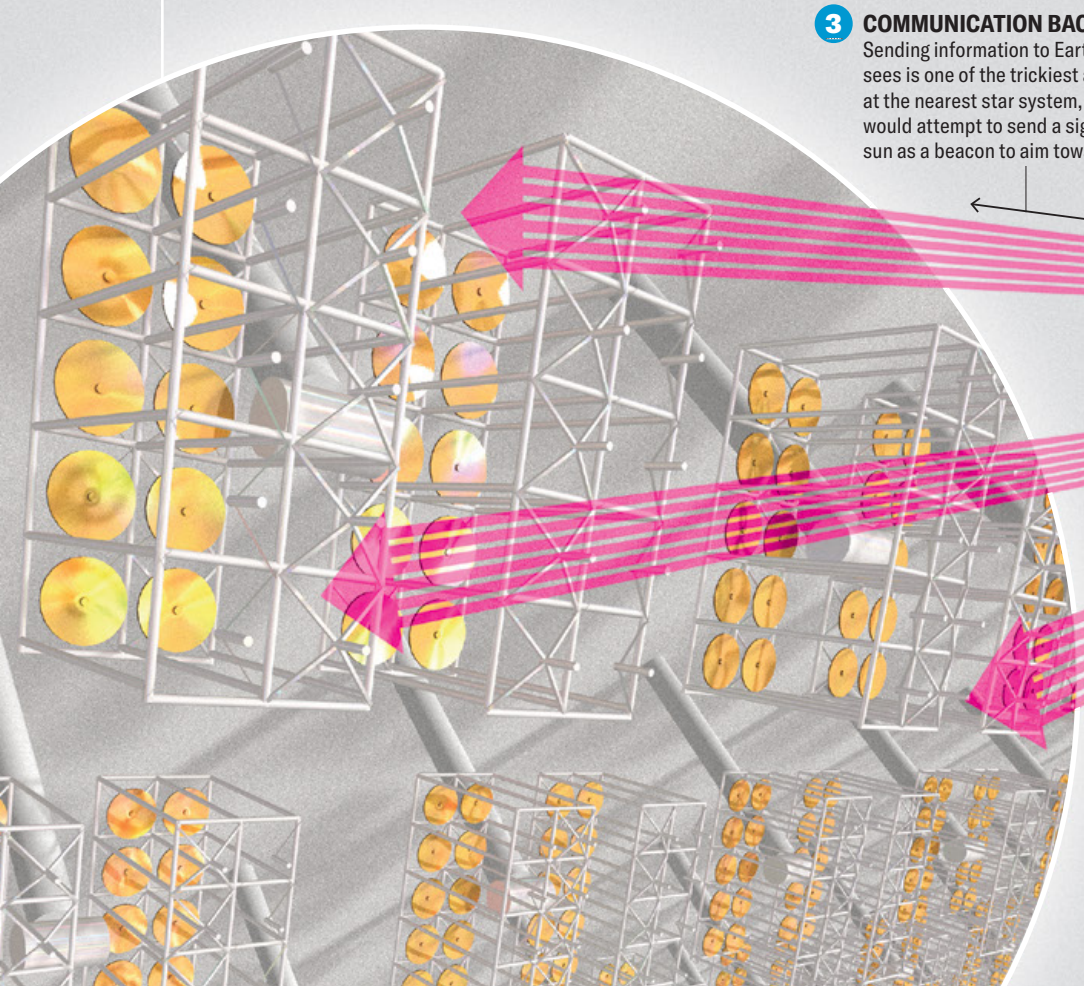
The Breakthrough Starshot initiative, announced in 2016, was a $100-million bid to send the first spacecraft to another solar system. The plan was to fire lasers at "lightsails" attached to tiny probes; the force of the blast would propel the miniature craft to the nearest star system in a matter of years. The project has floundered recently, and the full $100 million never materialized, yet researchers did make progress toward understanding how such a mission could work.



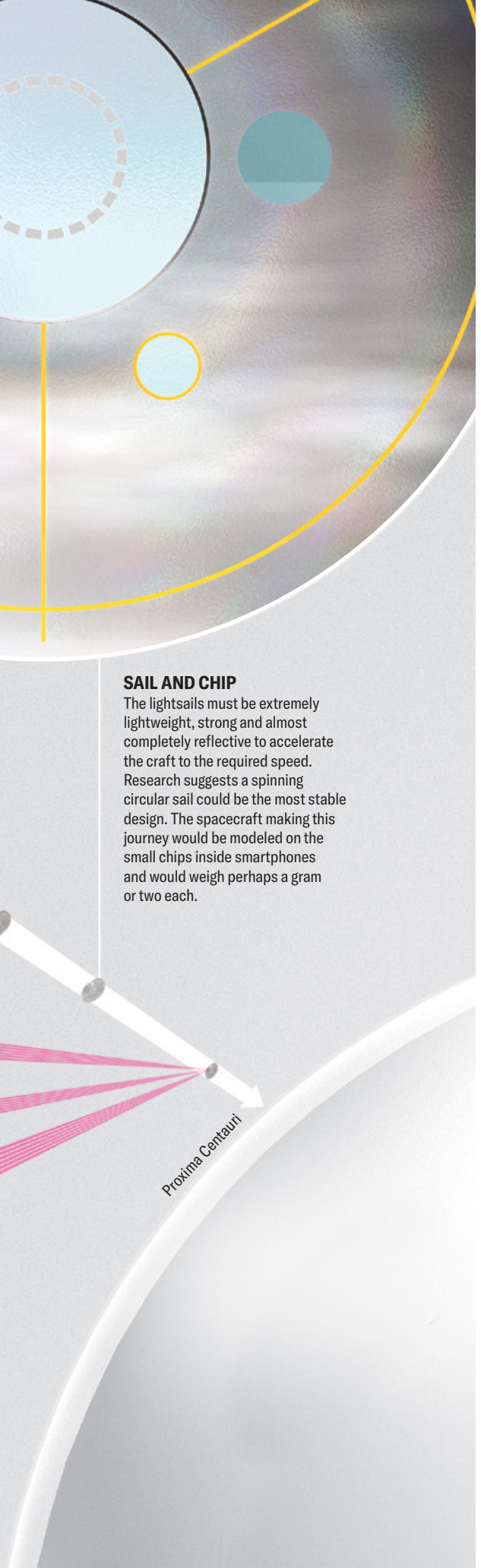
1 LAUNCH
A mothership launches a swarm of starships and sails into orbit.

2 A LASER SYSTEM BOOST
Many lasers in an array on Earth combine their light into a single beam pointed at the lightsails. The force of this light should accelerate the spacecraft to 20 percent of the speed of light in just 10 minutes.

3 COMMUNICATION BACK HOME
Sending information to Earth about what the mission sees is one of the trickiest aspects of the plan. Once at the nearest star system, Alpha Centauri, the probes would attempt to send a signal back to Earth, using the sun as a beacon to aim toward.



Graphic by Violet Frances



SAIL AND CHIP
The lightsails must be extremely lightweight, strong and almost completely reflective to accelerate the craft to the required speed. Research suggests a spinning circular sail could be the most stable design. The spacecraft making this journey would be modeled on the small chips inside smartphones and would weigh perhaps a gram or two each.

rocketry, proposed using the pressure of sunlight to push a vehicle through space. Some details of Breakthrough's specific plans, however, came from the work of a University of California, Santa Barbara, physicist named Philip Lubin. Back in 2009, seven years before Breakthrough Starshot began, Lubin attended a conference at the Naval Postgraduate School in Monterey, Calif. There researchers were discussing focused energy in the form of lasers, microwaves, particle beams, and more, known as directed energy, "mostly for purposes of taking down threats," Lubin says, meaning incoming missiles.

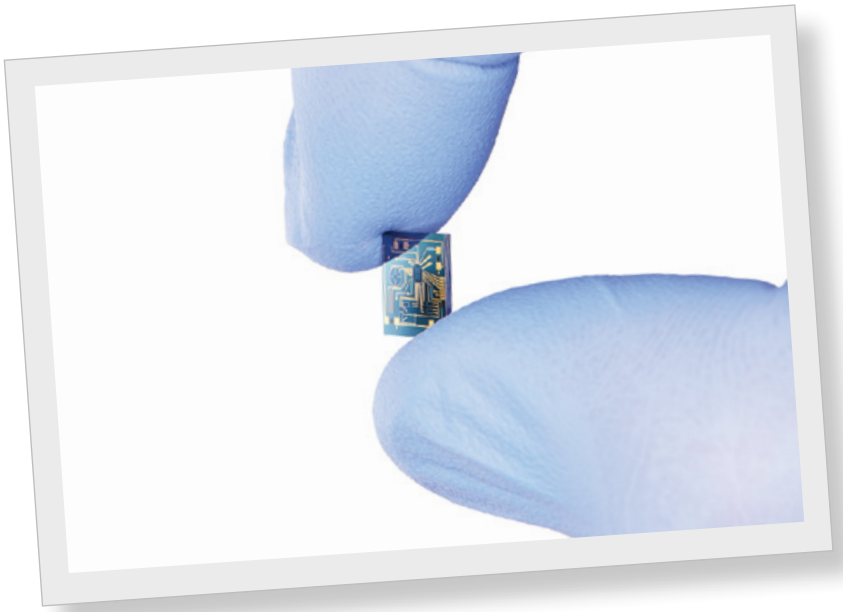
But as Lubin sat at the conference, he began to dream about other uses for the technology, especially if it were scaled up. Could it be used to protect Earth from asteroids rather than from intercontinental ballistic missiles? Or, he thought later, to propel a spacecraft far, far away? At home Lubin started crunching numbers. "I always want to figure out why it won't work, why you cannot do this," he says.

Despite his best efforts to defeat himself, it seemed the idea would work: You could direct energy at an incoming space rock, heat a portion of it up, vaporize that spot and shift the asteroid's orbit just enough to curve it away from Earth. And you could probably also send a spaceship on a significant journey. Lubin eventually applied for and received NASA funds to research both plans.

After the explosion of a meteor over the Russian city of Chelyabinsk in 2013, Lubin's planetary-protection work—under the project name DE-STAR—got more attention. Lubin, perhaps a future savior of the planet, was invited to give talks to other scientists, including one at the SETI (Search for Extraterrestrial Intelligence) Institute. There he mentioned that this same technology could also enable interstellar flight. A colleague told him he should talk to a guy named Pete Worden.

Lubin didn't, but he did keep working on his interstellar laser ideas with continued money from NASA. In 2015 he spoke at a conference hosted by the 100 Year Starship project. There he finally met Worden, who suggested Lubin send over a written version of his ideas. Lubin responded with a roadmap for interstellar flight, later published in the *Journal of the British Interplanetary Society*.

Worden wrote Lubin back quickly. "I have a friend," Lubin recalls him saying. "You mind if I send it to my friend?" Lubin told him sure, send it to whomever you want. The friend, of course, was Milner, and by January 2016 Lubin was meeting with Milner at his Bay Area mansion. In front of Milner was Lubin's interstellar roadmap, bedazzled with yellow Post-it notes. "Yuri says to me, 'You know, I've always dreamed, since I was a child, of



The Breakthrough Starshot spacecraft would probably be a small computer chip called a nanocraft. The prototype shown here is about 15 millimeters wide.

Stan Musilek

going to the stars,'" Lubin recalls. "'And now you've shown me the path.'"

Milner wanted to send the paper to experts who could evaluate its strengths and flaws. "If the reviews come back positive, then I'm willing to put in a fair amount of money," Lubin recalls Milner saying. He mentioned $100 million. "Unfortunately that, by the way, never came true," Lubin says. "There was no $100 million." The top two scientists affiliated with the project declined to be interviewed for this story.

BEFORE OFFICIALLY ANNOUNCING Starshot, Breakthrough officials had quietly recruited other thinkers in the field. In addition to Turner, who already knew Milner through a separate project called Breakthrough Listen, which searches for signals from alien civilizations, there was Mason Peck, an engineering professor at Cornell University and previously NASA's chief technologist. "That kind of opportunity does not come along every day, and I was all in from the very beginning," Peck says. Kelvin Long, a physicist and aerospace engineer who co-founded Project Icarus, also hopped onboard early. He sent Worden a design study, which he had written in three days while stuck in travel, for a hypothetical space probe that could move at 10 percent of the speed of light.

At Starshot's founding, the group identified around 30 problems to be solved before anyone could send an interstellar probe anywhere. Worden and James Schalkwyk of the Breakthrough Prize Foundation, working with three researchers from the Australian National University, wrote a chapter providing an overview of the project's initial phases for physicist and editor Claude Phipps's 2024 book *Laser Propulsion in Space: Fundamentals, Technology, and Future Missions.* Thirty-seven research groups, according to that summary, convened to understand and reduce the technology risks in those major areas. "Then the whole project came down to trying to figure out how to spend $100 million productively," Turner says.

Sometimes members of the crew got a bit of money to support their research, sometimes not. Starshot did bring people together, though—in person and virtually—to talk about their personal research on those problems. "Breakthrough is essentially a set of meetings," Lubin says. Other sources also cited meetings as a primary way scientists participated in the project.

Beginning in 2016, the Breakthrough Initiatives sponsored Breakthrough Discuss meetings "focused on life in the Universe and novel ideas for space exploration." The meetings, which were never specific to Starshot but did frequently cover topics related to the interstellar mission, have continued through 2025, with a gap in 2020 and a virtual meeting in 2021. Smaller satellite meetings also convened over the years to discuss specific technological and scientific aspects of the problem.

While they lasted, the meetings brought scientists and engineers together to investigate where the technology stood, what problems they didn't have solutions to, how feasible it was to overcome those problems and build something launchable, and what timelines and costs doing so would entail. There was palpable excitement in the early years—scientists felt they were part of a team embarking on an ambitious but tractable undertaking. They knew their biggest challenges were in certain areas: the design of the sail, the functionality of the laser system, the makeup of the spacecraft, and the construction of a communications apparatus that could signal back to Earth from light-years away. So, essentially, the whole system.

IT'S HARDLY WORTH SENDING a ship to another star if you won't be able to prove you've done it. Starshot would need to not just reach Proxima Centauri but also find a way to send back a signal strong enough to be detectable on Earth. It's a considerable challenge, however, to point a signal in the right direction from light-years away when both the probe and Earth are moving. Plus, both those feats must be accomplished with diminutive instruments on a spacecraft the mass of a pen cap or two.

According to Peck, Milner might have had unrealistic ideas—or at least ideas that conflicted with some of the scientists' suggestions—about what those signals should be like. "I do think Yuri Milner is very intelligent," Peck says. "I do think he has an adequate technical background" for the project. But he wanted things like video or 4K images from Alpha Centauri. And that, in Peck's view, was putting the

cart before the horse, to make an ancient analogy for a 21st-century endeavor.

To Peck, getting just one computer bit of information from another solar system would be valuable. Perhaps the probe could send a yes-or-no answer to a single question—is there a certain percentage of oxygen in the planet's atmosphere, for instance, or does the radiation environment seem suitable for life? "It's only incrementally better to get a gigabit from Proxima Centauri," he says.

According to the 2024 book chapter, the team found several ways to make comms somewhat feasible. The scientists could build a huge array of smaller receivers on the Earth end to catch weak transmissions. They also could enlarge the spacecrafts' transmitting antenna and send communications in optical instead of radio wavelengths, which can transfer more data faster. The team decided to use the sun as a beacon to point the homebound transmission toward, helping the information reach the right part of the vast universe. Still, Long calls the communications problem the "elephant in the room" in that it didn't get as much attention in initial research as other topics did—an assessment Carnegie Mellon's Manchester agrees with.

PROPELLING THE PROBES far enough and fast enough that they have something to communicate requires solving another problem: the lasers. Or, as the Starshot team called them, "the photon engine."

The first issue, the team found, was that a single laser would need to be impractically powerful— incomparable to anything that exists today. The researchers could create an array of smaller lasers whose beams would combine into one with 100 gigawatts of power, but then they'd need to ensure the light waves lined up with one another, like sound waves that are in tune. "People made serious progress on that," Manchester says. "They were able to do it with tens of lasers in the lab, which is a breakthrough."

But not quite enough of a breakthrough for Breakthrough. The project would need even more lasers, and those lasers would have to work outside the lab to reach deep into space—which poses another problem. "How do you get that out of the atmosphere without getting messed up?" Manchester asks. Turbulence in the upper air will cause the beam to twinkle.

They would need to adjust for that twinkling in real time. One laser, called a guide star, could shoot through the atmosphere constantly, and the scientists could use data about how it got distorted to correct the other lasers. But that correction would require millions of adjustments every second. In the 2024 book chapter, Worden and his co-authors pegged it as potentially the largest technical hurdle for the entire program.

There was palpable excitement in the early years—scientists felt they were part of a team.

The lasers pose a financial hurdle, too. To make Starshot feasible, the cost of powering them must come down from the current price of $100 per watt to around $0.01 to $0.05 per watt, according to Long. Peck is optimistic because, theoretically, the cost of laser power should decrease over time, similar to how Moore's law predicted that transistors in computer chips should get steadily smaller as the years passed. Still, that discount isn't instantaneous. "We were likely looking at a launch date not in the next 20 years, as the sponsor had hoped, but perhaps in 30 or 40 years," Long says.

Regardless of how much the laser costs, what form it takes or when any of this finally happens, policy is an issue. A laser that blasts out the equivalent of four power stations' worth of energy is, as the conference that spurred Lubin's original research interest demonstrates, a weapon. The only solutions for that problem are international cooperation and trust, which aren't at all-time highs right now.

ONCE THE PHOTON ENGINE is up and working, that laser energy has to hit the lightsail of a given spacecraft and propel it forward with a power of about 100 gigawatts. The sail must hold up to the onslaught while withstanding acceleration at a g-force of 40,000—that is, 40,000 times the pull of gravity you would feel if you fell off a cliff.

Substances that can withstand both the rigors of warp speed and the shock of a laser-cannon blast and remain reflective tend to be heavy. Starshot envisioned a lightsail material that can stretch four meters wide but weigh only a gram. The initial Breakthrough phase aimed to identify potential materials and designs, a process led by Harry Atwater of the California Institute of Technology, who did not respond to a request for an interview. The leading candidate substance his team found, according to the 2024 summary, is silicon nitride. Atwater and his colleagues published that result in 2022. Engineers have been able to fabricate it at submicron thicknesses—less than one-tenth the thickness of Saran Wrap.

Ultrathin wafers of the material can be puzzle-pieced together into a larger structure that is mostly reflective and doesn't absorb much light. Breakthrough engineers have done this assembly on the

When the project started, people thought interstellar travel was crazy.

millimeter scale but not the meter scale. Atwater and his team also coded a computer simulation that could figure out how various lightsail designs would perform during interstellar flight.

Another group, based at the University of Sydney, worked on ways to keep the hypothetical lightsail stable. The researchers joined meetings in 2021 and 2022 and shared their findings, but they never received any money from Breakthrough. "The whole thing always was outrageous," University of Sydney physicist Michael Wheatland says of the project's ambition. "I never believed it. But I think my perspective on things like this is that if you do fundamental research to try to solve a problem in the context of some outrageous scheme like that, then you can do really useful research."

And that's what the Sydney team did. They knew the sail would constantly be pushed around by the laser beam as it accelerated, so the team had to find some way to push it back to center. "But that then gives you oscillations," Wheatland says. Moving the laser could account for that, but like with the correction to untwinkle the lasers, the movement may be too much to ask of a bunch of lasers.

The sails are a separate problem from the spacecraft itself, which must be as small and lightweight as possible. Breakthrough calls the tiny spaceships "nanocraft." The leading candidate is the brainchild of Manchester, that wide-eyed graduate student when the program began. Manchester's early creations weren't meant for voyaging beyond the solar system—or even beyond Earth's orbit. As a graduate student at Cornell, working under Peck, he started designing postage-stamp-sized satellites around 2009. He called them, variously, Sprites and ChipSats. In 2011 he crowdfunded the project, and in 2014 he launched around 100 ChipSats to space. A glitch prevented them from deploying, though, and they burned up on the way back through the atmosphere.

After that disappointment, Manchester became involved with Breakthrough. His tiny satellites seemed like just what the team was looking for. "The notional idea was that some version of my ChipSat would end up being attached to that lightsail," he says. Manchester went on to do his postdoc at Harvard University, working officially on non-ChipSat projects. But with Breakthrough's help he was able to keep the ChipSat project on life support. "They were super nice to me during all of that," he says. "They would help me out, and they gave me little bits of funding."

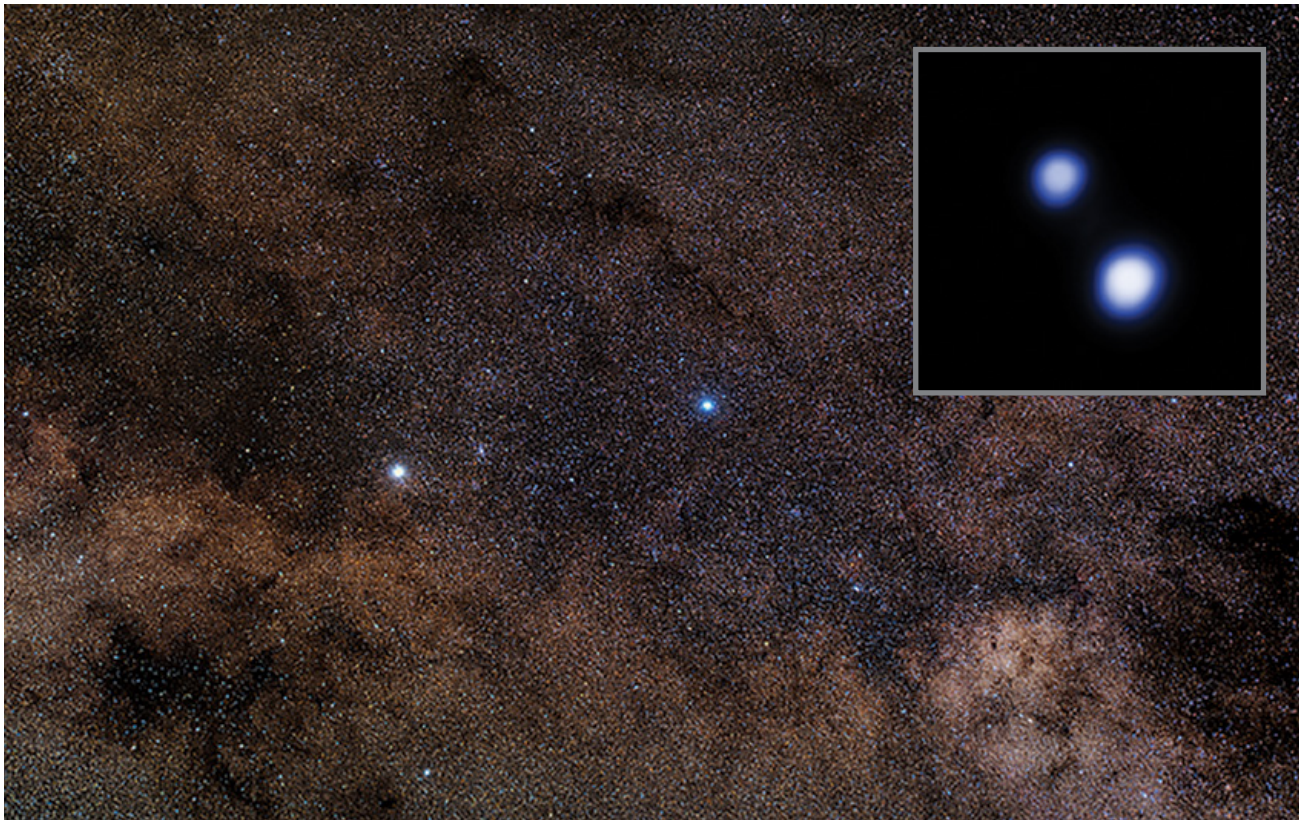
In 2019 Manchester was able to go for launch again, successfully deploying 105 ChipSats at once. He showed they could communicate with one another in space, acting as a swarm. The federal government let him fly them only once. "Then the [Federal Communications Commission] decided that we were going to destroy the world with space debris," he says—which wouldn't be a problem if they were headed way beyond low Earth orbit, to infinity and beyond.

BREAKTHROUGH hasn't gone beyond anywhere, of course. Still, in all four problem areas, the teams found that nothing was technically wrong with the basic plan. They also did enough research to find out what they didn't know and what kinds of technical development (and money) would be required to make the concept reality.

Progress was almost certainly slowed by the fact that the $100 million never materialized. Although the Starshot grants weren't made public, Lubin's experience might illustrate the scale of the spending. His group got two grants, one for $116,000 and another for about $80,000. Some of his colleagues in Australia also got $80,000. "We got less than $200,000 spread out over eight years," Lubin says. That was much less than NASA put toward Lubin's directed-energy interstellar work, although Breakthrough's press-centric approach meant its name was better associated with the project. "Breakthrough contributed less than 5 percent of the funding in our program in the end," Lubin says. "So it was always a little blip along the way. But in the public mind the entire program was a Breakthrough program, and that is simply not true at all."

Lubin calculates that overall, Breakthrough spent roughly $4.5 million on about 30 contracts. In late January 2025, after I contacted Worden and Avi Loeb of Harvard, also a Breakthrough scientist, a spokesperson for the Breakthrough Prize Foundation reached out. Worden and Loeb had declined interviews, but the spokesperson said, "I have a potential way to move your story significantly forward." She later referred to a report on the project that would be finished around spring 2025 and made available to SCIENTIFIC AMERICAN, but that report had not appeared by the time this issue went to print.

At this stage the future of the program is murky. Starshot appears to be on indefinite hold, if not over, although there was no final announcement and no fanfare to match its beginnings. Peck is not sure where things stand. "As far as I can tell, they've put it on pause, at least," he says. "And I think it's probably not going to continue for the near future."



Zdenek Bardon (*optical*); NASA/CXC/University of Colorado/T. Ayres et al. (*x-ray*)

The closest star system to the sun, Alpha Centauri, includes three stars. Two of them are a binary pair, seen in this close-up from NASA's Chandra X-ray Observatory (*inset*). A third star, Proxima Centauri, orbits the central two.

Physicist Martijn de Sterke, part of the Sydney group, and his colleague Boris Kuhlmey, a Sydney physicist who's helping with Starship-related research, heard only informally that Breakthrough Starshot was done. "It appears that this project has kind of disappeared," de Sterke says. "We have not heard from them for probably two years."

Some sources have interpreted the program's end as a realization that an actual starship, though technically possible, is still distant. "I think it's going to take 30 to 50 years of very hard work by a large number of very dedicated people, much like a Manhattan Project on steroids," Lubin says. Maybe that timeline wasn't appealing to Milner, some sources speculate, and neither was spending a Manhattan Project amount of money. Turner has a different perspective on how things turned out. To explain, he turns to the familiar example of medieval cathedrals, which took centuries to build—a length of time that humans rarely dedicate to any single project these days. "That [comparison] is often made as a kind of snide criticism of the short-sightedness of modern civilizations or people or profit motives," Turner says. "But I think it's actually a result of how fast technology is moving."

The innovations behind a cathedral's arches and finials didn't change much over the 200-year course of its construction. But the technology undergirding our world is unrecognizable compared with that of just a couple of decades ago. "It's very hard trying to imagine a major technological thing we're working on now for which they could have done anything at all useful 200 years ago," Turner says. "Nothing they could have done would make the slightest difference to us." Maybe that's what Breakthrough leadership decided about Starshot: it's best left to the people of tomorrow.

Despite the project's nebulous end and uncertain future, many participants spoke about Breakthrough positively. Manchester, for instance, sees it as at least a psychological success. When the project started, people thought interstellar travel was crazy—or they didn't think about it at all. "Breakthrough changed society's conception of this kind of stuff as a legitimate area of scientific inquiry," he says.

Serious people worked on the project, did serious things, made serious progress—even if not directly on a path toward Alpha Centauri. "It's still a long way off, but it's a lot closer than it was five or six years ago," Manchester concludes. The program also inspired people such as de Sterke and Kuhlmey to work on fundamental physics and engineering problems that might not have gotten attention otherwise. And maybe, at the end of the day, that will be Starshot's legacy. "If there was a one-sentence summary of what Breakthrough was and did," Lubin says, "it was to bring attention to the dream."●

FROM OUR ARCHIVES

Near-Light-Speed Mission to Alpha Centauri. Ann Finkbeiner; March 2017. ScientificAmerican.com/archive

The Landslide in Your Backyard

As climate change brings more intense rain to the mountains, dangerous debris flows are on the rise

BY JEN SCHWARTZ | ILLUSTRATION BY MARK ROSS

THE LANDSLIDE BEHIND MY NEIGHBOR'S BACKYARD doesn't exist—not according to the New York State landslide map or Greene County's hazard-mitigation plan or the federal inventory managed by the U.S. Geological Survey. But when you're standing in the middle of the debris field, the violence of the event is still evident 14 years after it occurred. The fan of the landslide, where a surge of boulders and mud blasted the forest open after rushing down the steeper slopes of Arizona Mountain in the Catskills, is about 100 feet wide—an undulating plane of rocks, mangled tree trunks, and invasive plants such as Japanese stiltgrass that thrive in disturbed areas.

Jen Schwartz is a senior features editor at *Scientific American* who writes about how we're adapting, or not, to a rapidly changing world.

On a hot July day the seasonal stream that runs through this ravine, named the Shingle Kill, is small enough to step over. When Tropical Storm Irene hovered over these mountains on August 28, 2011, the Shingle Kill swelled like all the otherwise unremarkable streams in the area, frothing downhill in a torrent the color of chocolate milk. This storm was a particularly bad one, dropping up to 18 inches of rain on the northeastern escarpment of the Catskills. Throughout the region explosive rivers eroded their banks, flooding towns and ripping away buildings.

The first house the Shingle Kill passes as it emerges into our community belonged at the time to Diane and Ken Herchenroder, who had lived there for nearly three decades. In the past, when the Shingle Kill occasionally raged, they could hear rocks colliding in the streambed. But this time it was louder—and faster.

From the screened-in front porch of their 1880s colonial, they saw the stream crest its banks. First it took out a 32-foot-long footbridge that connected one side of the property to the other. Then trees started coming downriver, crashing into a culvert at the bottom of the yard. The culvert clogged, washing out the road. Water got diverted across their lawn on one side of the stream, and in the other direction it blew out the garage side door, then the front doors. (Their lawn tractor was found downstream days later.) Diane watched her row of beloved lilac bushes, probably more than 100 years old and 15 feet tall, get ripped from their roots. "They just floated away. And we thought, that's going to be it," she recalls. "Then we heard a rumble like a train barreling down the mountain."

Less than 2,000 feet above, in a hollow high on Arizona Mountain, oversaturated soils released themselves into the headwaters of the Shingle Kill, picking up speed and whatever materials the flow encountered as it carved downhill.

As the slope flattened out, the landslide blew open the channel and spread out, depositing a wall of uprooted trees just upstream of the house. A slurry of rocks and mud continued flowing, plugging the Shingle Kill streambed all the way to the road, where it was stopped by the debris dam at the culvert.

Robert Titus, a retired geology professor, and his wife, Johanna Titus, explored the slide about a month later for their Kaatskill Geologist column in a local newspaper. "We don't use the words 'awe,'

Cassie Marion

In July 2025, days of heavy rain triggered multiple mudslides and rockslides in New York State's Adirondacks, including this one on Mount Colden. It blocked access to hiking trails in a popular recreation spot in the High Peaks Wilderness area.

'awesome' or 'awed' very often; we save them for when they are truly appropriate," they wrote. "This was one of those times." They described scenes that were evidence of boulders "floating on the moving muds," as well as hundreds of "twisted and broken trees" that had been thrown high above the stream bank and were now stranded on top of the ravine. The Tituses recently told me it was unlike anything they had seen before or since.

To this day, the scar where the landslide began is unmissable from miles away.

That this landslide didn't get recorded is somewhat a quirk of disaster recovery. Debris from the slide itself wasn't the singular cause of damage to any buildings or roads, so there was no financial fingerprint. The slide didn't injure or kill anyone. Landslides aren't mapped in the same way that the Federal Emergency Management Agency, for instance, tracks flood zones and inundation risk, and a rate of occurrence can't be modeled like a flood. Because landslide insurance practically doesn't exist in most of the country, no one needs the data to assess actuarial risk for homeowners. According to the New York Geological Survey, the vast majority of landslides in the state go unreported.

But the Shingle Kill landslide did change the mountainside that day. Joel DuBois, director of the Greene County Department of Soil and Water, visited the site in the days after Irene and reviewed some recent photos of the stream corridor that was affected by the debris flow. "There appear to be a number of cycles of incision and aggradation," DuBois wrote. "That is to say that channel incision, or down-cutting, results in steeper bank angles and higher bank heights, leaving the adjacent hillsides susceptible to landslide" both during and after flood events. The sediment then flows downstream and accumulates at existing debris dams, which tends to cause channels to migrate laterally, he explained. That too can trigger landslide activity.

The area remains vulnerable at a time when landslide risk is expected to increase across much of the northeastern U.S.—as well as a lot of the world. That's because climate change is causing concentrated bursts of rain that fall over a short period to occur more frequently. Such intense rainfall events are known to be the biggest trigger of landslides.

It's not quite right to say landslides aren't common in the Catskills, because this superold plateau has been eroding for perhaps a few hundred mil-

lion years. On a nongeological timescale, though, landslide susceptibility isn't something many people think about in New York State, and the state geological survey can estimate only that between 100 and 400 occur every year.

As warmer temperatures lead to more moisture in the air, climate change is quickly warping that math. In the Northeast, the heaviest rainstorms are now 60 percent heavier than they were in the 1950s, according to the Fifth National Climate Assessment. In a 2023 study, researchers at Dartmouth College found that extreme precipitation in the region will increase by 52 percent by the end of this century, mostly because of a higher number of such events each year. "Our landscape has pretty much been in equilibrium, for the most part, since the glaciers left," Andrew Kozlowski, a New York State geologist, explained during a 2022 USGS presentation. "With climate change, we may be shifting that equilibrium and throwing all of this completely off balance, and there's going to be a natural readjustment."

"LANDSLIDE" IS THE BROAD TERM for the movement of soils, rocks, and other debris down a slope. There are several different classifications for landslides. Some, like the Shingle Kill debris flow, move far too fast to be outrun. More than any other factor, they are set off by an intense storm. Others, such as rotational slides—backward-curving masses of material that can be hundreds of feet deep—are more sensitive to rainfall over the course of a season. They can move very slowly when a destabilized slope takes months to fail.

Landslides can happen pretty much anywhere certain conditions exist but are most common in very steep mountain terrain where plenty of rain falls. In 2024 the U.S. Landslide Susceptibility Index was released and stated that 44 percent of the land in the U.S. could potentially experience landslide activity. Susceptibility is based partly on where landslides have occurred previously, and it wasn't until the past decade that high-resolution lidar made it possible for states to survey vast swaths of land for evidence and clues. The extent to which states have done so is uneven.

Benjamin DeJong, director of the Vermont Geological Survey, says you can think of landslide susceptibility as an inexact recipe. You're going to need steeper slopes to achieve some kind of baseload that puts weight on the slope. Next, add loose, unconsolidated materials that can become saturated with water. If those saturated materials are overlying or underlying another kind of material that has very different permeability, meaning its ability to take in water, that contrast is a big factor.

Then you look at what's on the base and on the top of the slope. If the base, or toe, is undercut—by a road, for instance, or a meandering stream—that's going to make the slope more susceptible. Overloading the top, or head, of a slope with weight also drives it toward failure.

The fourth ingredient is the loss of vegetation that helps to hold soils together. In California, for example, this loss happens on a regular cycle with wildfires. Vermont, DeJong says, went through an experiment in the 1800s where "the state tried to turn itself into Scotland by cutting down all the trees and bringing in sheep." It was a bad idea that caused erosion and mass slope failure everywhere. The state gave up on that plan and allowed the forests to regrow. The last variable is how the slope handles stormwater. With more extreme precipitation events, it doesn't take much mismanagement of a slope for the heavy weight of rain to concentrate in ways that cause the slope to fail.

Geologist David Petley, who writes the Landslide Blog for the American Geophysical Union, has been maintaining a database of deadly landslides worldwide since 2004. He's seen a clear long-term trend. "But by far—by far—the year that had the greatest total landslides that I've recorded was 2024," he says. "Last year was completely off the scale." Why? "The most simple hypothesis is that it was the year with the highest-ever global temperature. I do genuinely think it's that simple." There's solid evidence that high atmospheric temperature, and possibly high sea-surface temperatures as well, drove high-precipitation events globally. "Last year I saw an extraordinary frequency of big storms that were triggering hundreds of thousands of landslides," Petley says. They occurred at different locations all over the world.

In the U.S., the remnants of Hurricane Helene, which came ashore in Florida in September 2024, dumped between 20 and 30 inches of water over the mountains of North Carolina. The storm ended up triggering more than 2,000 landslides across the Southeast. According to the USGS, in some cases several smaller mudslides converged into a single channel, burying entire communities in debris. The total number of people killed by landslides specifically, versus by flooding or a combination of the two, is hard to parse. But one storm-triggered mudslide in Craigtown, N.C., swept through a house, killing 11 members of the Craig family for whom the town is named. During the storm, four successive landslides in that valley wiped out the town.

In the Appalachian Mountains of North Carolina, very old landslides might have been "brought back into activity" during Helene, Petley explains,

reactivated by staggeringly intense rain. Scientists at World Weather Attribution pinned that extra intensity on climate change, reporting that it had made the storm's rainfall throughout the Southeast about 10 percent heavier and the "unprecedented" rainfall totals over three days about 70 percent more likely than they would have been otherwise.

In California, where dramatic debris flows have long been a concern, climate change is making matters worse in two ways. Bigger, more destructive wildfires wipe out more of the vegetation that was stabilizing the landscape. And then atmospheric rivers—a newer phenomenon consisting of long, narrow conveyer belts of moisture—arrive, bringing a series of intense rainfall events. Between December 2022 and January 2023 nine back-to-back atmospheric rivers struck California, leading to more than 600 landslides.

Climate change is increasing landslide risk globally in other ways. In high mountain regions such as the European Alps and the Himalayas, melting permafrost and retreating glaciers are destabilizing steep slopes. A catastrophic glacier collapse in Switzerland this past summer destroyed an entire village; thankfully officials evacuated people just before it happened, but one person was killed.

Petley says the thing that's surprised him most recently is the speed of change, especially during this past El Niño cycle. Strong rainfall events have always happened occasionally, but suddenly they are happening a lot. "I don't think I fully understand why we're seeing such a rapid shift to these events where a heavy rainfall will trigger 2,000 or 3,000 landslides in a relatively small area," Petley says. In New Zealand in 2023, Cyclone Gabrielle triggered at least 100,000 landslides. Even in regions such as the Himalayas, where the monsoon season is becoming drier overall, the number of landslides is going up because the rainstorms that do arrive are more intense. "I worry a bit," Petley says, "that the shift is happening so fast and becoming so extreme that in some places the risk is essentially unmanageable."

VERMONT, LIKE NEW YORK STATE, got clobbered by Tropical Storm Irene in 2011. DeJong, the Vermont state geologist, describes Irene as a wake-up call. "The mountains," he says with a degree of irony, "are now where hurricanes come to die."

But it wasn't until two freak July rainstorms—spaced exactly a year apart, one in 2023 and one in 2024—that the state's geological survey became alarmed that landslides were going to be a much bigger problem than in the past. Given his experiences with Irene, DeJong expected the July 2023 storm to lead to maybe a handful of slides. Within a month of the storm his team had received more than 70 requests for landslide evaluations. Working on the ground in the aftermath of these two storms made DeJong realize that rainfall events at that scale "are fundamentally altering the landscape in ways that are not immediately recognizable," he says.

> "By far the year that had the greatest total landslides that I've recorded was 2024. It was completely off the scale."
>
> —DAVID PETLEY *UNIVERSITY OF HULL*

Now the four-person Vermont Geological Survey team is working on putting together a landslide-susceptibility map. The goal is to start with a more technical tool for scientists that can be overlaid with forecasts from the National Weather Service, which would create debris-flow forecasts like the ones already produced by the Los Angeles Department of Public Works. If that's successful, the next step, DeJong says, would be creating a map that's more accessible to the public, something that a person who's looking to buy a parcel of land could reference to do some due diligence on landslide risk.

But that gets tricky. The city of Juneau, Alaska, carried out a mapping project to evaluate levels of risk, with the aim of incorporating that risk into its land-use planning in 2024. The maps also would have highlighted concerns with existing buildings, though, meaning homeowners identified as living in high-risk areas might see their property values decline. Juneau's susceptibility map was vehemently rejected by the community last year and was not adopted. In Vermont, as in many places, evidence of slope instability—and even past failures—hardly factors into development or the issuing of building permits.

Rising landslide risk in mountainous places also creates a difficult tension about how to adapt to the effects of climate change. Recent disasters have made clear that mountain valleys in certain regions may not be great places to live. In Vermont "we're losing a lot of housing in our flood corridors—which is a good thing," DeJong says. "We're getting people out of harm's way." But the state, like many others around the country, has a housing crunch with the need to build more. "When we've lost options down in the valleys, that puts a lot of building pressure up onto our slopes," he explains. "And it's really hard to make the argument not to do that." Successfully adapting to one climate effect means running headlong into another.

Recent intense rain events "are fundamentally altering the landscape in ways that are not immediately recognizable."

—BENJAMIN DEJONG
VERMONT GEOLOGICAL SURVEY

THERE ARE MANY CLIMATE-related problems to worry about in my Catskills community: the surging numbers of disease-carrying ticks, the choking out of native plants by invasive species, the hurricane-remnant floods, the decrease in winter snowfall that would replenish the aquifers, the summertime whiplash between deluge and drought. The Shingle Kill landslide wasn't on my radar as a potential climate problem until a massive, ultraluxury resort and "branded residences" development was proposed for the hillside next to it. The plan calls for building more than 85 new structures totaling 275,593 square feet on a 102-acre site, 45 percent of which is classified as having steep slopes. To do so, developers will have to cut down about 11 acres of trees. The site, like the rest of our hamlet, has no access to municipal water or sewage. In addition to lining ponds for water storage and building a wastewater-treatment plant, a road network will be cut into the mountainside.

The public documents for the project do not appear to show that a geologist evaluated whether the weight of all that development, plus the deforestation and excavation during construction, might further destabilize the slopes of the Shingle Kill. Our town planning board approved the project in May 2025 without requiring an environmental impact statement that would have identified and attempted to mitigate the biggest hazards. (I am a member of a community group that is suing our town planning board, arguing it didn't take a hard look at potentially significant adverse effects to the environment from this project, including on groundwater availability, erosion, flooding and landslide risk.)

Diane and Ken Herchenroder's house wasn't damaged by the 2011 landslide, but the event did plenty of harm. Much of their property was rearranged by the acute displacement of raging water. The solid plug of rocks and mud, some 10 feet tall, had to be excavated from the streambed. Even once things were fixed, they didn't want to stay. "We used to listen to the rain and the stream with the windows open, and it was very comforting," Diane says from their house in New Hampshire, where they moved two years after the storm. "Honestly, after that slide occurred, Ken and I, I would have to say, have a little bit of post-traumatic stress from that." Diane says her photographs of the landslide are on a CD somewhere; she hasn't looked at them since. "I don't really ever even talk about that day," she says. "It was pretty devastating."

In 2018 Joe Merlino bought the Herchenroders' former property, where he now lives with his daughter and his mother. A few years ago they had members of the U.S. Army Corps of Engineers come assess ongoing erosion along the Shingle Kill. The streambed continues to widen, and a sharp curve just upstream of Merlino's house means floodwaters could rush right at it. He recalls that in 2021, when Tropical Storm Henri came through the Catskills, boulders smashed against the bridge that provides access between his house and his mom's trailer. "[The Army Corps] basically told us the erosion is not going to stop," Merlino says.

Merlino often walks along the edges of the fan with his dog, observing the changes to the old debris piles with each storm. The possibility of more landslide activity is never far from his mind, he says, especially with a major development approved for the hillside above his home.

I asked him whether he gets scared every time there's heavy rain. "I come home from work early," he says, to keep an eye on things and intervene if necessary. A few years ago he moved his daughter's bedroom to the front of the house, away from the steep pitch of his backyard. "My fear is about my living room, which is in the back and has a lot of glass," he says. "I watch the water rip around that curve, and one day something is going to come through and take the side of my house right out."

Greene County, where the Merlino family and I both live, is one of the four counties identified by New York State as the most vulnerable to expected annual building loss from landslides in the future. The county has steep escarpments that slope into the Hudson River Valley, which is rich in clays and silts from Glacial Lake Albany, a prehistoric waterbody that drained some 10,500 years ago. "I think we're going to see a lot more slope failures in some of these populated areas in the Hudson Valley," Kozlowski, the New York State geologist, said in 2022.

Greene County considered landslides a threat back in 2016. In 2023 the county revisited its hazard-mitigation plan; our town, Cairo, was the only municipality out of 19 that did not participate. In the updated plan, the county removed landslides as a hazard, reasoning that they are "unlikely to lead to a disaster."

CJ Matherne

A section of the Shingle Kill streambed 14 years after a debris flow occurred on Arizona Mountain in New York State's Catskills during intense rain. The southern slope, shown on the left, continues to erode.

IT'S TRUE THAT LANDSLIDES don't do the same economic harm to our county as flooding and ice storms. But when they do occur, rebuilding is rarely an option. When a family lost their house in the town of Catskill to a landslide after a heavy rain event in May 2024, there wasn't much anyone could do but condemn the structure.

With funding for emergency response and climate resilience endangered at the federal level, is it worth investing in susceptibility maps for landslides that may never occur? Should people hesitate to build on potentially unstable slopes when that's perhaps less risky than living directly in a flood path?

DeJong says these are valid questions, but after his experiences over the past few years, he sees things differently. "We in Vermont have, so far, been incredibly fortunate to not see any fatalities," he says. He remembers an older couple who were sitting in their house in July 2023 when the slope behind it failed. The structure warped outward, bending absurdly into something "that looked like a fun house falling over on them," he recalls. Emergency services extracted them relatively unharmed, but DeJong knows it could have been worse. It turned out a lot worse in western North Carolina during Helene, where for years many building codes dismissed the risk of construction on steep slopes.

It might take only one bad slide to change people's minds about the risk. Before 2014, DeJong says, Washington State, much like New England, did not pay much attention to landslides and had no landslide program in its state geological survey. But then a slope in Oso, about an hour outside Seattle, experienced a catastrophic failure, taking out a neighborhood and killing 43 people. The state now takes landslides very seriously.

"The Oso slide of New England could be right around the corner," DeJong says. "People will say, 'Why didn't we know about this hazard? X number of people just died.'" He hopes his team can get its landslide-susceptibility maps finished so that when big rainfall events are forecast for the Green Mountains, officials can warn people in especially risky areas. "We're really trying to switch to being more proactive so that X never becomes a number." ●

FROM OUR ARCHIVES
The Disasters Science Neglects. Naomi Oreskes; April 2023. Scientific American.com/archive

EVOLUTION

The Dawn of Polar

Fossils hint at when birds began making their mind-blowing journey to the Arctic to breed

BY LAUREN N. WILSON AND DANIEL T. KSEPKA

ILLUSTRATION BY CHASE STONE

Bird Migration

A breeding pair of ornithurine birds and their hatchlings survey the coastal floodplain. These birds shared this landscape with dinosaurs, such as the *Pachyrhinosaurus* herd in the background, 73 million years ago in what is now northern Alaska.

GOLDEN AUTUMN SUNLIGHT GLINTS through the sedges and shrubs of the tundra in northern Alaska. Winter is approaching, and soon the region will be buried under snow and ice. For the past three months the chatter of the Arctic Tern colony has served as the soundtrack of the summer breeding season. But now, with daylight waning, the terns need to head south. In an instant, the usually noisy birds will fall silent, a behavior known as "dread." Moments later the entire colony will take to the skies to begin its 25,000-mile journey to Antarctica—the longest known migration of any animal on Earth.

Lauren N. Wilson is a Ph.D. student at Princeton University, where she studies the evolution and paleobiology of birds and reptiles. Her fieldwork has taken her to the badlands of Montana and the polar wilderness of Alaska's North Slope, among other locales.

Daniel T. Ksepka is a paleontologist and science curator at the Bruce Museum in Greenwich, Conn. His research focuses on the evolution of birds and reptiles.

The Arctic Tern is not the only bird that spends its breeding season in the Arctic. Billions of birds belonging to nearly 200 species—from small sparrows such as the Smith's Longspur to large waterfowl such as the Greater White-fronted Goose—make their way to the far north every spring to reproduce and then make the return flight south for the winter. It's no easy feat. Migration is costly. Even under ideal conditions, such an epic journey requires huge amounts of energy and exposes the travelers to dangerous weather. The mortality risk is high.

But undertaking these trips allows the birds to take advantage of the seasonal conditions in these environments. The endless summer sun supports lush plant growth, flourishing insect swarms, and plentiful fish populations nourished by zooplankton blooms. With 24 hours of light a day, the birds can more easily catch food such as slippery fish and tiny insects. The round-the-clock daylight also means many of the animals that prey on birds are less likely to sneak up on a nest unnoticed.

Scientists have long wondered when birds began making these extraordinary journeys. New fossils that we and our colleagues have discovered and analyzed are finally providing some clues. A decade of expeditions to the Arctic Circle in Alaska has yielded a trove of bird fossils—including several hatchlings. The remains, which date to approximately 73 million years ago during the Late Cretaceous period, constitute the earliest known record of birds reproducing at polar latitude. The fossils hint that early birds may have already been traveling to the top of the world to raise the next generation of winged wonders.

THE POLAR MIGRATION of birds is one of nature's great spectacles. To make the marathon journey to the Arctic, birds need physical stamina. They typically have various anatomical and behavioral adaptations to long-distance travel. The Arctic Tern, for example, is a marvel of efficiency. Its skeleton is lightweight and partially filled with air, allowing it to glide for long distances without expending any energy to flap its wings. It can eat on the move, plucking fish from the surface of the ocean as it flies. And, like many migratory birds, it can sleep while gliding.

Migrants also need to be skilled navigators to reach their breeding ground. The precise methods by which birds find their way remain mysterious, but biologists generally agree that they use some combination of visual landmarks; the position of the sun, moon and stars; Earth's magnetic field; and scent-based clues. A degree of learning also seems

Pat Druckenmiller

Scientists have recovered dozens of three-dimensionally preserved teeth and bones from hatchling birds, including this tip of a beak, from the Arctic Circle in Alaska, showing that birds were reproducing at polar latitude by 73 million years ago.

to be involved—in many species, first-time migrants appear to simply fly in the correct general direction, whereas experienced birds may use landmarks to take a more efficient route.

As impressive as the trip itself is, the Arctic migration is part of a much grander scheme: the birds are literally changing their ecosystems at their destinations. Although most Arctic birds are only physically in the Arctic for the breeding season, they spur the success of plants by pollinating flowers and dispersing seeds. They also help to manage insect and rodent populations and, by extension, help to control the spread of disease. In fact, birds are so critical to the success of their habitats that they are hypothesized to have played a key role in structuring remote ecosystems over deep time. Birds carry small organisms, such as plants and insects, over long distances to colonize remote polar regions. Were it not for the evolution of migratory birds, today's tundra would be much more barren.

Despite the importance of migration for the birds themselves and for the wider landscape they inhabit, we actually know very little about the origins of this phenomenon. To answer such a fundamental question, we have to look backward in time to the fossil record. Unfortunately, the polar fossil record is sparse, and most of the fossil-bearing sediments there are covered in ice or water. In spots where these sediments are exposed, fieldwork is often challenging, dangerous and expensive. Furthermore, bird bones are some of the rarest fossils in the world because they are small and fragile, making them less likely to survive long enough to fossilize, let alone to be discovered by paleontologists.

In the rare cases when we do manage to find a fossil bird in the Arctic, it can be difficult to determine whether that bird was a visiting migrant or a permanent resident. Let's say we find exactly the same species, in rocks from exactly the same time period, at both temperate and polar latitudes. Even then, we can't say the extinct species migrated. There's always the possibility that it merely inhabited a broad area year-round. The range of the modern-day Common Raven, for instance, encompasses practically the entire Northern Hemisphere.

There is a clever way to home in on whether a fossil deposit contains migratory birds, however. The vast majority of living birds that inhabit polar regions migrate to lower latitudes after the breeding season ends. So, if we find fossil evidence of birds not just present but breeding at polar latitudes, we are headed in the right direction. This is where our work on fos-

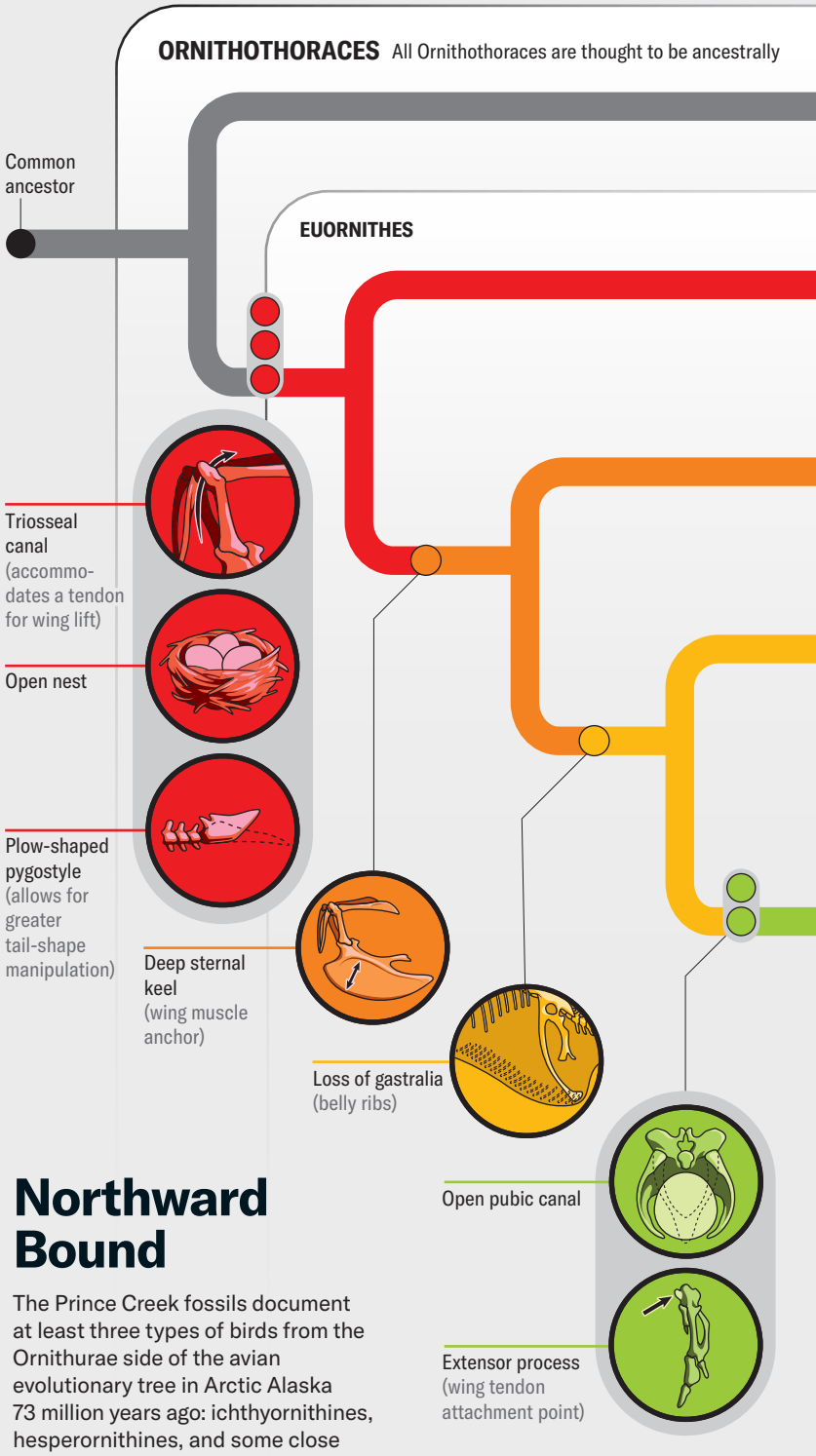
sils from a Late Cretaceous body of rock in northern Alaska called the Prince Creek Formation comes in.

AT THE BEGINNING of the 1993 movie *Jurassic Park,* a team of paleontologists gently brushes away sand to reveal an intact dinosaur skeleton in the badlands of Montana. Although fossil fieldwork is never as simple as removing loose sediment with a paintbrush (sorry, Steven Spielberg), Arctic fieldwork is in a league of its own. Winter brings temperatures as low as –50 degrees Fahrenheit, tons of snow and limited hours of daylight. The summer isn't a walk in the park, either: giant mosquitoes are out in force, it's almost always rainy and cold, and there is So. Much. Mud. Moreover, large mammals are out and about, making potentially dangerous wildlife encounters a concern.

In August of 2022 one of us (Wilson) was on her second trip to the Arctic. It was about five in the morning when she awoke in her tent along the Colville River near the Prince Creek Formation. The sun had already been up for hours. With a couple more hours before she needed to be up, she was frustrated that she had to climb out of her warm sleeping bag to pee. She begrudgingly put on a hat and coat and unzipped her tent, still half asleep. Then her heart stopped. About 20 yards away, right near one of her crewmates' tents, was a giant, fuzzy brown blob. She tried frantically to remember her bear training: Should she call out and try to wake everyone else up? Grab her bear spray? Try to scare it out of the camp? Only after putting herself through this roller coaster of emotions did she finally realize that the "bear" had a large set of horns on its head. Thankfully, the camp visitor was just a musk ox.

One may wonder why we bother with such extreme fieldwork. Wilson has often found herself wondering the same thing while working in –30-degree-F weather. But for the same reason the fieldwork is challenging, the fossil discoveries in the Arctic are some of the most exciting in the world. The Prince Creek Formation is located at a modern-day latitude of 70 degrees north and preserves fossils of animals that lived an estimated 72.8 million years ago. Plate tectonic activity has shifted Alaska south since that time. During the Late Cretaceous, these species would have been living at an even higher paleolatitude of 80 to 85 degrees north, practically at the North Pole. Summers would have brought plentiful light and warmth, but year-round occupants of the ecosystem had to endure winters with freezing temperatures, snowfall and about four months of continuous darkness.

Paleontologists have known about dinosaurs from the Prince Creek Formation since 1983, but it's only



Northward Bound

The Prince Creek fossils document at least three types of birds from the Ornithurae side of the avian evolutionary tree in Arctic Alaska 73 million years ago: ichthyornithines, hesperornithines, and some close relatives of living birds possibly in the Galloanserae clade. There are no enantiornithines—the dominant birds of this time period elsewhere in the world—in the assemblage. This pattern suggests that the evolution of key traits related to reproduction and development in the ornithurine lineage might have allowed these birds to exploit the sun and insects that abound in the Arctic in summer.

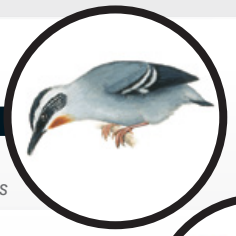
Graphic by Brittany Cheung (feature icons) and Rebecca Gelernter (bird illustrations)

capable of long-distance flight

Enantiornithes

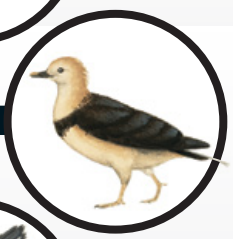
Enantiornithines were the dominant birds for most of the Cretaceous period

Longipteryx chaoyangensis



Archaeorhynchus

Archaeorhynchus spathula



Songlingornithidae

Yixianornis grabaui

Gansus

Gansus yumenensis

Apsaravis

Apsaravis ukhaana

ORNITHURAE

Ichthyornithes

Ichthyornis dispar

Fossils from the ornithurine clades Ichthyornithes, Hesperornithes, and possibly Galloanserae (*hatched, below*) are the only bird fossils found in the Cretaceous Prince Creek Formation.

Hesperornithes

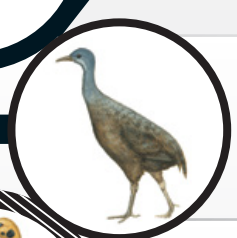
Hesperornis regalis

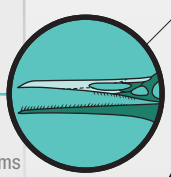
Faster growth

NEORNITHES

Palaeognathae

Lithornis celetius





Expanded premaxilla (bone that forms the tip of the upper beak)

NEOGNATHAE

Galloanserae

Asteriornis maastrichtensis

Loss of teeth

Biparental care

Neoaves

Tsidiiyazhi abini

Altricial development (chicks hatch underdeveloped and require parental care)

Researchers excavate a fossil site along the Colville River in northern Alaska (*left*). To recover small bones and teeth, the team washes fossil-bearing sediments through screens (*right*) and takes the resulting concentrate back to the laboratory for examination under a microscope.

in the past couple of decades that work led by Patrick Druckenmiller of the University of Alaska Museum of the North and Gregory Erickson of Florida State University has begun to change our perception of Arctic life in the Cretaceous. Their team's discovery of baby dinosaur fossils helped to demonstrate that dinosaurs were year-round inhabitants of the ecosystem because the baby dinosaurs would have been too young to migrate before the onset of winter. More recently, smaller bones found alongside the dinosaur fossils have led to another exciting discovery: the oldest evidence of polar bird reproduction.

To date, we have identified more than 50 three-dimensionally preserved bird bones, along with dozens of teeth, from the site. The fossils are so tiny that they could all fit together in a single jam jar. Nevertheless, they represent one of the best collections of Late Cretaceous North American bird fossils and document the presence of at least three types of birds that lived alongside nonbird dinosaurs in Arctic Alaska. Not only that, but many of the fossils belong to baby birds and represent the earliest known growth stages of these groups of birds. Together these fossils demonstrate that birds have been nesting in the Arctic for at least 73 million years, nearly half the time they have existed on Earth.

Close study of these delicate fossils has allowed us to reconstruct the birds of the Prince Creek Formation and their role in the ecosystem. Picture the Arctic in early summer 73 million years ago. The coastal floodplain that was desolate throughout the long winter is now lush with plant life and buzzing with insects. It's the perfect setting for a newly hatched chick to grow up in. A head pops up from a bowl-shaped nest. It belongs to a baby ornithurine, a close relative of modern birds. He is still covered in downy feathers and scrambles about on skinny legs, not yet ready to take flight. While learning his way around the world, he takes special care to stick close to his parents. Unlike many other Late Cretaceous birds, he and his relatives have a toothless beak that serves as a precise tool for picking off creeping insects under their watchful eyes. This chick hatched a month ago and is already off to a strong start thanks to a new evolutionary innovation: the larger egg laid by advanced ornithurine birds.

The coastal floodplain offers premium real estate for nesting. Dinosaurs of all kinds are preparing for the arrival of their young, and last year's young are still recovering from their first Arctic

Kevin May

Kevin May

winter. The ornithurine chick and his family aren't the only types of birds here to call this landscape home. Kick-diving hesperornithines are hunting in the river waters, and ternlike ichthyornithines are wheeling overhead. And they're all here for the same reason birds still nest in the Arctic today: lots and lots of sunshine.

THE PRINCE CREEK BIRDS provide definitive evidence that birds bred in the Arctic during the Cretaceous. Whether they migrated there from elsewhere to reproduce is tougher to establish. We can get at this question from a few angles, however. Let's start by considering whether these birds had the ability to make such a journey in the first place. We know that any birds from the preceding Jurassic period are unlikely to have flown very far. Such early birds had not yet evolved many of the features that help modern birds fly skillfully and efficiently. For example, the iconic *Archaeopteryx* was capable of flight, but it appears to have had relatively low endurance and couldn't perform complex maneuvers. The keeled sternum, or breastbone, that anchors the pectoral muscles in modern birds was either absent or at most a flat cartilaginous plate in *Archaeopteryx*. Clawed fingers interrupted the leading edge of its wing, and compared with birds of today, its feathers appear to have been less flexible and thus less adept at forming a coherent airfoil. Even its tail seems like an archaic reminder of *Archaeopteryx*'s grounded ancestry. Whereas modern birds have a short tail with a special plough-shaped bone called the pygostyle that lets them spread their tail feathers into a fan, *Archaeopteryx* retained a long and aerodynamically unwieldy tail similar to that of its theropod dinosaur ancestors.

Over time birds evolved a panoply of skeletal and soft-tissue features that improved their flight capabilities. The bony tail became shorter, and the fingertips diminished from large claws to tiny bones hidden under the feathers. Advanced Cretaceous birds in the group Ornithothoraces, which includes the Prince Creek specimens, are in many ways the first birds with an unquestionably proficient flight apparatus. In these birds, the sternum bears a keel that provides additional attachment for the muscles that power the flight stroke. The shoulder joint is oriented higher on the back, allowing for better positioning of the wings. The first finger also anchors an alula, a cluster of small feathers that acts as a mini airfoil, helping in

fine maneuvers. Thanks to these anatomical innovations, the Prince Creek birds (apart from the flightless hesperornithines) would have been capable of flying great distances to the Arctic to breed.

A closer look at where these birds fit in the avian family tree provides more clues to how they came to reproduce in the far north. Ornithothoraces is divided into two groups: the enantiornithines and the ornithurines. Enantiornithines were the dominant birds for most of the Cretaceous period. These toothed birds ranged from sparrow- to turkey-size and showed a great diversity of forms, from *Longirostravis,* with its slender bill, to the blunt-toothed *Bohaiornis,* to the toucan-beaked *Falcatakely.* They lived almost everywhere.

Ornithurines, which include modern birds and their close relatives, were rarer in Cretaceous ecosystems. Like enantiornithines, most Cretaceous ornithurines still had teeth. But advanced members of the group differed from enantiornithines in having fewer teeth; no gastralia, or belly ribs; and separated pubis bones, which allowed them to lay larger eggs. In contrast to the enantiornithines, which seem to have thrived in forested environments, ornithurines appear to have stuck largely to aquatic habitats during the Cretaceous.

Intriguingly, the Prince Creek bird fossils all come from ornithurine birds. We have identified bones and teeth of three types so far: ternlike ichthyornithines; hesperornithines, which used their feet to propel themselves through water; and some nearly modern close relatives of living birds. Notably absent from our assemblage are any enantiornithines. If all Ornithothoraces were capable of long-distance flight, why are the otherwise ubiquitous enantiornithines missing from Alaska?

WE SUSPECT ONE ANSWER lies in the egg. Anyone who regularly cooks eggs has probably noticed a little white blob, which for many people spoils the otherwise appetizing appearance of the yolk. This blob is the chalazae, a pair of protein-rich "tethers" that attach the yolk to the shell. Chalazae protect the embryo when birds rotate their eggs in the nest to ensure that the embryos get thoroughly bathed in nutrients during incubation. Reptiles, which lack chalazae, do not practice egg rotation. In fact, rotating a crocodile egg can disrupt development of and kill the embryo.

So far paleontologists haven't found any fossil chalazae that might allow them to trace the origin of this structure. But we have a hunch that it evolved in ornithurines because crocodilians, nonavian dinosaurs and enantiornithines all buried their eggs at least partially in the ground. Fossil clutches of enantiornithines demonstrate that they placed their eggs vertically in sediment or soil, leaving only the tops exposed. This arrangement would have stabilized the eggs, keeping the embryo safely attached to the yolk, but it was much less efficient for incubation. At best, brooding enantiornithines would have been able to make only partial contact with their eggs, resulting in poorer heat transfer and slower development of the embryo. In fact, some paleontologists speculate that they could not incubate via body contact at all, because the eggs were too small to support that parent's weight.

Perhaps the lack of this tiny embryo "seat belt" explains the absence of enantiornithines in the Arctic. Most modern birds that breed in northern Alaska nest from late May through June. For birds that can nest in vegetation, this is a lovely time of year. Yet even at the start of June, snow may still persist in patches, and the soil may remain chilly or even frozen. Temperatures were warmer in the Cretaceous, but the Arctic winter was still dark and cold, and spring would have taken longer to arrive than at more southern latitudes. For ground-nesting enantiornithines, cold soil would have been highly unwelcoming for nests.

Why not just wait until later in the summer to nest? There may simply not have been enough time. Because enantiornithines could not provide full-contact incubation, their eggs probably took substantially longer to hatch than those of birds that can sit on their eggs in nests built in vegetation. The inexorable march of the seasons would have left almost no time for fledging for birds that hatched in late summer.

Still, although enantiornithines took several years to grow to full size, they appear to have been highly precocial as hatchlings. In fact, there is some evidence they could fly within a day of hatching. That might seem to make up for the longer incubation time in the race against winter. But another aspect of enantiornithine biology might have thrown up a roadblock to Arctic breeding.

Recently discovered fossils preserved in amber reveal that enantiornithines molted their body feathers all at once. This style of molting allowed them to trade their juvenile plumage for adult plumage rapidly when the time came. Yet it would have been a big liability in colder climates. If an early cold snap occurred during a molting interval, being caught half naked could have been deadly to small-bodied birds that had to generate their own body heat, as opposed to obtaining it from external sources such as the sun. By eliminating the possibility of nesting in the summer and overwintering, this molting pattern might have served as a barrier to those birds inhabiting Arctic environments year-round.

FROM OUR ARCHIVES
The Quantum Nature of Bird Migration.
Peter J. Hore and Henrik Mouritsen; April 2022. Scientific American.com/archive

Mark Boulton/Science Source

The Arctic Tern migrates tens of thousands of miles every year between its breeding grounds in the Arctic and its wintering grounds in Antarctica.

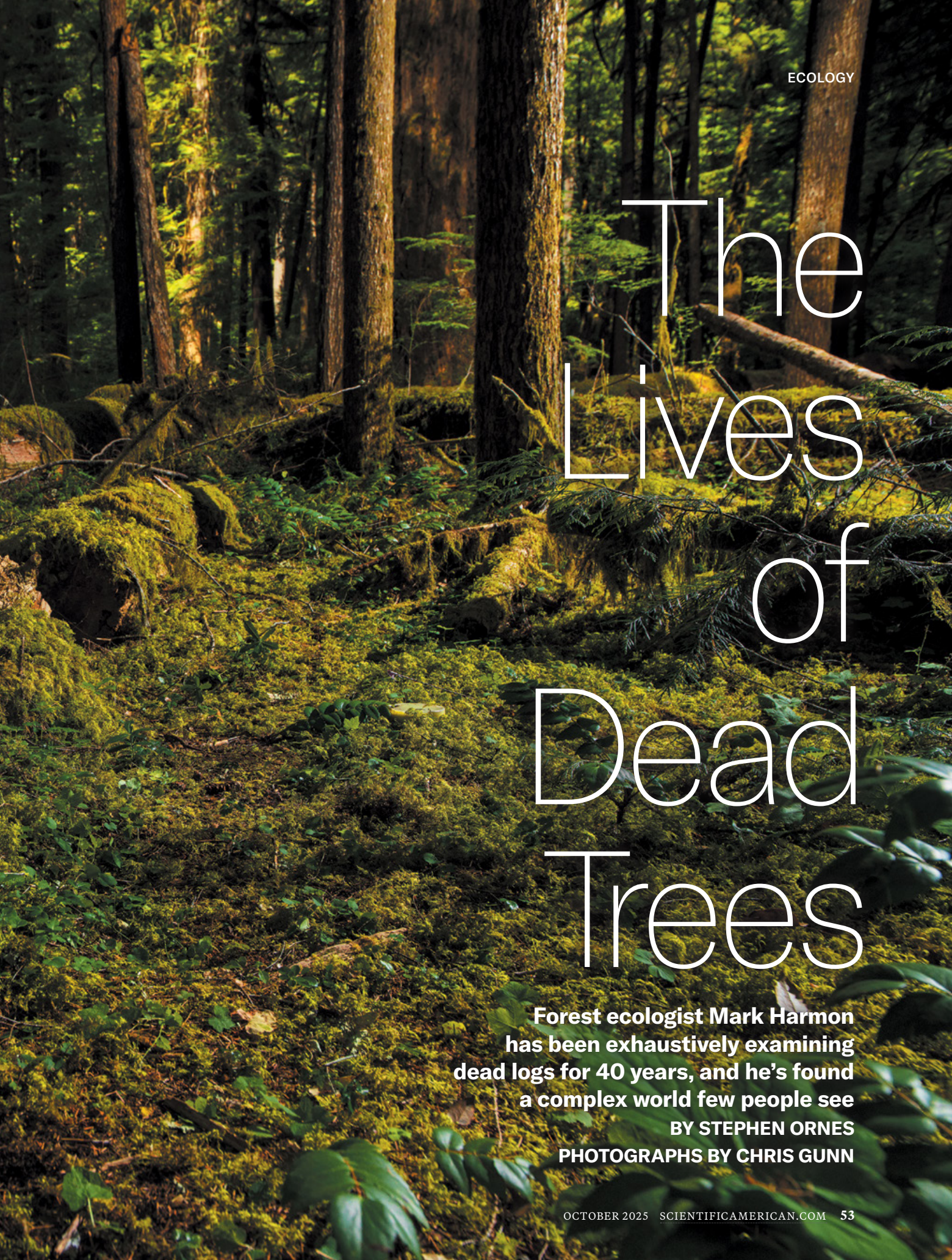
Needing a longer runway to make it from the egg to migration-ready seems to have left enantiornithines unable to establish themselves in the Arctic. Ornithurines, in contrast, were able to exploit the Arctic at least seasonally thanks to evolutionary innovations in reproduction and development that occurred in their lineage.

OUR WORK ON THE PRINCE CREEK birds is not over yet. We currently have only circumstantial evidence that they were migrating to the Arctic to breed rather than living there year-round. But we may be able to build our case with a technique called stable isotope analysis, which lets us use comparisons of the ratios of different forms, or isotopes, of the same element in an animal's teeth or bones to infer its diet, reconstruct its environmental conditions, and even trace its movements over its lifetime.

We know that dinosaurs were overwintering in the Arctic because their young would not have been ready to migrate anywhere the first winter after hatching. Perhaps comparisons of the isotopic compositions of bird and dinosaur teeth could inform us about the habits of the Prince Creek birds. Many biological factors, such as diet and metabolism, influence isotopic compositions, though. We still have a lot of groundwork to do to understand these factors before we apply stable isotope techniques to our fossil birds.

Meanwhile let's check in on our hatchling. The Late Cretaceous world is harsh for an ornithurine chick still learning the ropes. At just a month old, he is still very vulnerable and depends on his parents for comfort and safety. If he strays too far, he risks becoming dinner for one of the many dromaeosaurs who are also trying to provide for their young. Because of these predators, many of his siblings won't survive to the end of the summer, and some just might end up as fossils in the long run. If he can make it a few months, perhaps he will fly south with his kin to somewhere sunny for the winter. He'd be one of the lucky ones. This scenario is the harsh reality of life at the top of the world. But in the remarkable adaptations and behaviors of birds lies hope for survival. ●

Green moss encases dead, downed logs at site 3 in Oregon's H. J. Andrews Experimental Forest, part of a remarkable 200-year study of tree decay that is 40 years underway.



The Lives of Dead Trees

Forest ecologist Mark Harmon has been exhaustively examining dead logs for 40 years, and he's found a complex world few people see

BY STEPHEN ORNES

PHOTOGRAPHS BY CHRIS GUNN

MARK HARMON CROUCHES LOW next to log number 219: a moss-covered western hemlock tree trunk, five meters long, lying dead on the ground in the lush green woods. It's marked by a thin aluminum tag. The forest ecologist leans in close, his unruly white beard nearly brushing against the decomposing cylinder. Dark, flaky patches on the dull, reddish-brown wood closer to the ground show where fungi have infiltrated the cellulose within. Farther down the trunk, multicolored fungal conks protrude like hard shelves barely big enough for a mouse. A shiny black beetle scurries along the ground, then out of sight under the log. Harmon presses gently on 219 with three fingertips. It's so spongy that he is reluctant to roll back a chunk of it to reveal what lies underneath. "Oh, I don't want to destroy it," he says slowly. "It's all falling apart."

Harmon, a longtime faculty member at Oregon State University, has been watching number 219, and more than 500 other logs nearby, decay for 40 years. He has trekked to this site in the H. J. Andrews Experimental Forest, a watershed nestled in Oregon's western Cascade Mountains, at least 100 times. He drives more than two hours on paved and gravel roads from his home in Corvallis, Ore., then hikes in half a mile through the undergrowth, carrying tape measures, scales, saws and a computer to chronicle the relentless changes. His goal: establish an exhaustive baseline dataset that any scientist could use to test hypotheses about tree decomposition or to compare patterns of decomposition in the Pacific Northwest with those in other regions.

Decomposition can explain how and how fast carbon, captured by plants during photosynthesis, returns to the atmosphere. That process, which plays out at dizzying scales of both space and time, influences the long-term productivity and biodiversity of a forest. Harmon's findings could influence when, or even whether, forest planners decide to remove dead logs to improve the health of the woods. Decay shapes how wildfire spreads through a timberland, too. Snags (dead but standing trunks) and downed trees also provide habitat for animals.

Before Harmon and his colleagues launched this log-decomposition experiment, scientists studying the impact of dead wood on the environment primarily looked only at what had already rotted, without understanding the variety of long-term factors that affected the decay. But by the early 1980s Harmon and other researchers realized patterns of decomposition emerged only from detailed tracking of actual logs sustained over decades, like snapshots stitched together into a multidimensional movie. Even after 40 years, Harmon says, ecologists are unearthing new questions: How does temperature affect the activity of decomposers such as brown rot fungi on various wood species? How do changing ecosystems promote or hinder interactions among invertebrates, microbes and wood? At what rate is carbon released from downed wood? This last

Stephen Ornes is a freelance writer who has written about whale falls, extrasolar planets and the mathematics of cake cutting. His book, *Breakdown: A Brief History of the End of the Universe and Everything in It,* will be published next year.

Mark Harmon gently pulls up a section of a tree carcass to reveal how deeply it has decomposed. For four decades he has been gathering detailed measurements of more than 500 logs at six forest sites, looking at how bugs, fungi and microbes affect decomposition rates, tissue density and sugar concentration. Wood breakdown affects a forest's resiliency, wildfire spread and carbon dioxide emissions. Harmon's successors are to collect data for another 160 years.

one is of particular importance because it affects nutrient cycling through soils and roots, as well as climate change.

Harmon is leading the way to answers, but he may never know what they are. He designed the grand project to run for at least 200 years—well beyond his lifespan and those of his immediate successors. Ecologist Jennifer Powers of the University of Minnesota says that Harmon "really thought about long-term processes that shape forests in setting up a study he knew he would never see the end of."

MOST PEOPLE REGARD dead trees as a nuisance, a wasted resource or something to trip over. Harmon sees revelation. When he was 21, during a run in the hilly forests of central Massachusetts, he encountered a green log that seemed to glow against the dark wooded backdrop. He had a vision that he would one day run a research effort on log decay. Granted, he wasn't entirely clearheaded at the time. "It was helped by some substances," he admits. "But I can still see that log." For his first major research project, Harmon compared decomposition rates of 10 species of trees killed by fires in the Smoky Mountains. Conifer species, he found, decayed more slowly than deciduous trees, and *Quercus prinus,* the chestnut oak, decayed the fastest, losing 11 percent of its wood density every year.

In 1979 Jerry Franklin, at the time a forest ecologist at Oregon, visited the Smokies where Harmon, then a graduate student at the University of Tennessee, happened to be his tour guide. Forestry school had taught Franklin that a dead tree was valuable only if it were loaded onto a truck and sent to a mill. "But I came to realize that this wood I had been taught was a waste, a fire hazard and an impediment to travel had a lot of value," Franklin says. It was still a part of the forest, still boosting biodiversity by providing habitat and returning carbon and nitrogen to the soil, he explains.

Franklin recognized a likeminded soul in Harmon. In 1980 Harmon moved to Oregon to start his doctorate, and a few years later Franklin recruited him to run an experiment simply called

the Log Decomposition Project. Harmon described it as a "'They must be crazy, but maybe not' kind of project." He launched the endeavor with specific but far-reaching scientific questions. He wanted to know how widely the decay rate of hardwood differed among tree species, how colonies of microbial decomposers affected that rate, and whether bugs and other invertebrates sped up the process by bringing in the microbes.

The idea of cutting down a bunch of healthy trees in one location and hauling them elsewhere was a hard sell. During one dinner at Oregon State, Harmon listened to the dean of the School of Forestry call the emerging experiment the "most stupid f–ing thing I've ever heard of in my life." People regularly reminded him that he would die before it was finished or that "only an idiot" would wait 200 years for results. Over time, however, funding continued to come through.

In September 1985, under Harmon's direction, crews used chainsaws, loaders, grappling hooks, cables and shovels to cut down and drag nearly 100 trees from an area kilometers across to rot at a clearing in the Andrews Experimental Forest. Each fresh log was 5.5 meters long and 45 to 65 centimeters in diameter. Since then, Harmon, nicknamed Dr. Death by the National Science Foundation, has come to this place, known as site 3, again and again to photograph the logs' physical appearance and to catalog the succession of bugs and other invertebrates that munch on and live within the rotting wood. He uses electronic calipers and scales attached to a laptop to measure lengths, widths, weights and tissue densities, and he carries digital instruments to record the temperature, humidity and air pressure of the forest. He's also carried thousands of "cookies"—disks several centimeters thick cut from a log's end—back to the Andrews laboratory to tease out concentrations of sugars and track changes in carbon and nitrogen content. Every sample has been barcoded and stored. After the first collection, considered time zero, Harmon's team filled an entire walk-in cooler with more than 1,000 cross-

An aluminum tag identifies log number 219, a western hemlock. Researchers have discovered that logs of different species might take anywhere from three to 750 years to fully decompose (*left*). Under some dead trees, the action of decomposers might create new soil, but under others it may leave crumbled clay or sand (*right*).

sectional samples. His wife, Janice Harmon, a plant ecologist, scanned the barcodes of more than 35,000 entries over time. Plastic bags filled with rotten wood samples—some powdery, some chunky—still inhabit their garage in Corvallis, waiting to be documented.

As the crew set up site 3 in 1985, it also dragged numerous other trees, similarly cut, to five additional sites scattered throughout this forest. One worker navigating a loader at the site, Harmon says, unleashed a string of expletives describing exactly what he thought about the project. In total, the crew placed 530 logs representing four species that dominate this ecosystem: Douglas firs, western hemlocks, western red cedars and Pacific silver firs. Since the beginning of the second Reagan administration, Harmon has been leading scientists, volunteers and students to these hidden glades to measure in close detail the decay of the woody carcasses.

Although weathering, solar radiation and bugs all contribute to breakdown, wood-decomposing fungi do most of the damage. These nearly invisible microorganisms often hitchhike on invertebrates' backs to get inside the rotting wood. To catalog these populations, Harmon and his colleagues delicately scrape them off the inner walls of log cavities and into sample bags, along with whatever other tiny creatures are hanging around in there.

Today the forest is co-managed by the U.S. Forest Service, Oregon State University and the Willamette National Forest and hosts a variety of silviculture studies. It has become a long-term lab where scientists investigate the effects of disturbances such as floods and fires on a forest ecosystem. "Because we have this [70-year] baseline," says Mark Schulze, an assistant professor at Oregon State and the forest's director, "we can really understand these processes." The experimental forest is the perfect place for Harmon's work because decay plays out over scales that researchers don't usually measure. The ongoing project is still revealing new mysteries and has created a small

People reminded Harmon he would die before the research was done or that "only an idiot" would wait 200 years for results.

but energetic subfield. Scientists are now measuring tree decay in dozens of similar undertakings on six continents. Researchers in China, Germany, and other countries are probing how the climate, environment and decomposer populations in different regions interact to shape decay. They're looking at how decay rates vary by species and location, which can shape policies around forest management and habitat protection. They're feeding data to climate scientists, who can more precisely model the rates at which different kinds of forests may hold or release carbon.

Harmon is widely regarded as the de facto pioneer of the field, having published dozens of relevant papers that have garnered thousands of citations. Hans Cornelissen, a systems ecologist at Vrije Universiteit Amsterdam who in 2012 launched Loglife, a log-decomposition project in the Netherlands that mimics the Oregon study on a smaller scale, calls Harmon the "founding father" of modern wood-decomposition science. Harmon is exacting, even obsessive, about tree decay. His focus is so deeply hardwired that he can't ignore it. "It's quite aggravating, actually," he says as we move away from log 219. He seems resigned, almost exasperated. No matter where he goes or what he's doing, even on vacation, he says with a sigh, "I'm always seeing dead trees."

IT'S QUICKLY GETTING WARMER on this May morning as we make our way through the ongoing experiment. Other tagged specimens in the distance look like random waves frozen on a green pond. The woods are quiet except for the occasional knock of a downy woodpecker or the distant whistle of a varied thrush. Harmon, now 72 and technically retired, easily straddles thigh-high berms and bobs under fallen conifers. He removes his hard hat and wipes his brow as we come up on a fallen western red cedar, not part of the experiment. In the study's first couple of decades, Harmon says, the researchers found that the outer sapwood of a western red cedar decayed faster than any part of any other tree they examined. The interior heartwood, however, is the most decay-resistant, which is why it's often used to build decks and raised-bed gardens for homes. Two extremes in one species. As a result, cedars tend to stand intact until their roots give out, and they crash down all at once—unlike, say, Douglas firs, which tend to splinter in big chunks, leaving standing snags. Trees decay differently when they've fallen and are within easier reach of decomposing microbes than when they remain standing.

Harmon puts his hard hat back on over his thinning tangle of dark hair. I'm wearing a hard hat, too, because big trees drop big limbs, although I question whether this plastic shell could protect me against a falling widow-maker. A few meters away Harmon points out log number 218, a Douglas fir. Whereas the crumbling hemlock heartwood of log 219 seemed about to implode, this prone Douglas fir was firm enough for us to stand on. The advantage of the Andrews log-decomposition study, Harmon says, is that he and his team know exactly when decay started—not the case for trees downed naturally—which helps them and other scientists more clearly understand the timeline and drivers of decay within and among different species. "We knew that was our opportunity," he says. "Those were our initial [experimental] conditions."

Over the past 40 years the mounting measurements have yielded unexpected insights. Deadwood might remain on a forest floor or stand upright as a snag for anywhere from three to 750 years. In a 2020 analysis, Harmon and his colleagues estimated that decay rates can vary by a whopping 244-fold across species and climates. *Heliocarpus appendiculatus,* a tropical tree better known as a jonote, loses nearly 98 percent of its mass a year, whereas *Eucalyptus camaldulensis,* the river red gum tree, endemic to Australia, loses only about 0.4 percent a year. Rates can vary within species, too. "You could have parts of trees that could last less than a decade and others up to 1,000 years," Harmon says.

Another surprise is how drastically deadwood can alter the forest floor. Fallen trees don't simply rot. Harmon rolls sturdy log 218 away from us to reveal a patch of mineral soil the color of the darkest chocolate. It's made up mostly of crumbled clay, rocks and sand, as opposed to organic soil, which contains decaying organic matter such as that from trees and leaves. Fungal tendrils twist through the dark brown mat.

"This forest floor has kind of melted away," Harmon says. Organic soil digested by fungi or nematodes or bacteria under the log hadn't been replenished. Yet leaves and branches falling on the log had accumulated and decayed over decades, producing a fertile organic soil on top of the

Wildfire in August 2023 almost ruined the 40-year decomposition experiment, consuming three of the six log sites. Bright-orange fire moss has quickly colonized some of the burned landscape.

log, where moss and other plants were now growing. "The log has basically elevated the forest floor 50 centimeters off the ground," Harmon says. Fallen trees shift the chemistry of the soil below and above and, with that, the population of microbes in the environment.

Harmon's group found that the soil changes the tree, too, as ants and other insects ferry dirt and microbes into the decaying log. Whether a dead tree touches mineral soil, stands as a snag or remains suspended over the forest floor after falling against a living tree can dramatically influence the concentration of carbon it stores. And the mix of the many decay factors influences the likelihood that new trees will take hold in that ground, reshaping the habitat of a forest, which in turn affects the overall health of the region.

A forest dominated by slow-rotting species can hold enormous stores of carbon for decades or centuries, whereas quickly decaying species can release lots of carbon into the air. Extrapolated to a global level, sequestration and emission can significantly affect amounts of atmospheric carbon dioxide and therefore influence climate change.

Knowing these rates is particularly important to climate change modelers, says Jonathan Schilling, whose lab at the University of Minnesota focuses on decomposition and fungi. He has run wood-decomposition experiments in Alaska and New Zealand, among other places. "We've got logs rotting all over the place," he says. In 2024 he and his colleagues compared the decomposition preferences of white rot—fungi that break apart the tough lignin in trees and thus release the carbon dioxide—with those of brown rot, which head for the cellulose, leaving the lignin behind.

"There's a lot more carbon left behind in the soil for the brown rot mechanism," Schilling says. That matters because white rot fungi, which prefer warmer forests, are encroaching on northern regions because of changing temperatures and rainfall. The result? More carbon dioxide gets pumped into the air. "There's a lot of carbon at stake," Schilling says, "and enough uncertainty that we need to know how that process works."

The Andrews experiment has inspired many

others around the world. In the 2000s Powers launched the first tropical decomposition study, which involved 14 countries, with Harmon's work as a model. In 2012 Cornelissen and his crew in the Netherlands arranged logs of 25 species in two "tree cemeteries" for his Loglife experiment. Cornelissen has also collaborated on decomposition projects in Romania, Germany and China. In 2024 he worked with Amy Zanne, an ecologist at the Cary Institute of Ecosystem Studies in Millbrook, N.Y., on a review of wood-decomposition studies that explains varying decomposition patterns around the world. Zanne sees a hidden wonderland in decomposition, populated by overlooked, disregarded players that nonetheless have critical roles in an evolving ecosystem. "I love thinking about the underdogs, the underseen things, and how hidden things make the world go round," she says.

HARMON ALMOST LOST the entire Andrews project on August 5, 2023, when lightning struck a tree on Lookout Ridge. Fire spread quickly, and within a few weeks it had incinerated 70 percent of the watershed, nearly 10,500 hectares. It burned through three of the six log-decomposition sites, stripping living trees of leaves and incinerating much of the deadwood, which was a blow. After Harmon and I hike back to the gravel road near site 3, we drive farther up the ridge, get out and walk through the ghostly remains of site 6.

Nearly two years on, this site retains a faint, mephitic whiff of smoke and char. The fire felled giant firs and sculpted cedars, and the burned boles still stand in scorched, abstract shapes. Remaining branches, leafless and thin, glow silver against the dark snags in the sharp afternoon sun. They'll fall eventually. Harmon squats and cradles a singed aluminum tag, barely readable, identifying a round, blackened wood skeleton as a western hemlock. There are no scurrying beetles in sight.

Fire changes the game, Harmon says. It can be tragic. Yet it is also an opportunity to see a forest in a new light. "It's changed, but, you know, it's going to come back. It's going to be another manifestation of the same thing," Harmon says. Unlike in site 3, the organic soil here has all but vanished under ash and charcoal. It's unclear which of the four species in the log-decomposition project may proliferate most in regions devastated by fire. And even though fire kills trees, it doesn't remove them. Harmon points to a snag, maybe 30 feet tall, with tiny mushrooms protruding from cracks. He notes a little patch of uncovered mineral soil where seedlings have emerged. New trees will grow with the legacy of snags and downed trees around them, and the new forest may be even more structurally interesting.

The future, of course, is uncertain. The log-decomposition project is one of 27 in the Long-Term Ecological Research network, a collection of large-scale experiments funded by the National Science Foundation probing everything from how expanding cities affect tree-growth rates to how disturbances such as extreme wind, fire and flood shake up an ecosystem. Funding has always been an issue, and Harmon, Franklin, and others worry that recent widespread cuts to federal grants may reach the Andrews experiment. Its timescale might save it. The project requires little maintenance, and the logs will rot whether anyone is watching them or not. For now, someone is.

Harmon retired in 2016 but can't stay away; he is still churning through enormous datasets to publish papers. He has turned the reins over to two younger researchers at Oregon State: Georgia Seyfried, a soil scientist who studies biogeochemical processes, and Jacob Bukoski, an ecologist who focuses on carbon cycling and climate change mitigation. "I think there's a real opportunity here," says Bukoski, who looks forward to working fire into the increasingly complex, emerging view of decomposition.

On our way back to the forest headquarters, Harmon and I cross a narrow stream flanked with hemlocks and pull over. We walk about 45 meters into the forest—over dead logs, under dead logs—and arrive at one of Harmon's sacred spots, a grove of giant Douglas firs. Their trunks stretch at least two meters across. Their lowest branches are higher than most of the other surrounding treetops. These behemoths are older than the *Mona Lisa*. We stand silently in the shadow of the living giants for a few moments. With their thick, deep furrows and invisible crowns, they seem invincible and infinite. But that's an illusion, Harmon says. This scene is a snapshot. After the trees fall—unless they burn—they'll probably remain intact on the forest floor for another few hundred years, housing bugs, remodeling the forest and eventually sinking softly into the contours of the new woodland, in the shadow of new giants.

Scientists used to assume that decomposition was instantaneous, Harmon says—that when a tree dies, it essentially disappears. "But that's not true anywhere on Earth, and it's never been true," he says. A dead tree is "just a transition to something else." ●

FROM OUR ARCHIVES
A New Generation of American Chestnut Trees May Redefine America's Forests. Ferris Jabr; Scientific American.com, March 1, 2014. ScientificAmerican.com/archive

The 2023 Lookout Fire left hundreds of snags—dead trunks that remain standing. Dead Douglas firs tend to splinter and fall in pieces; cedars tend to stand until their roots give out and then crash down in an instant. Ongoing study of scorched logs should uncover how fire ash and charcoal alter decomposing wood.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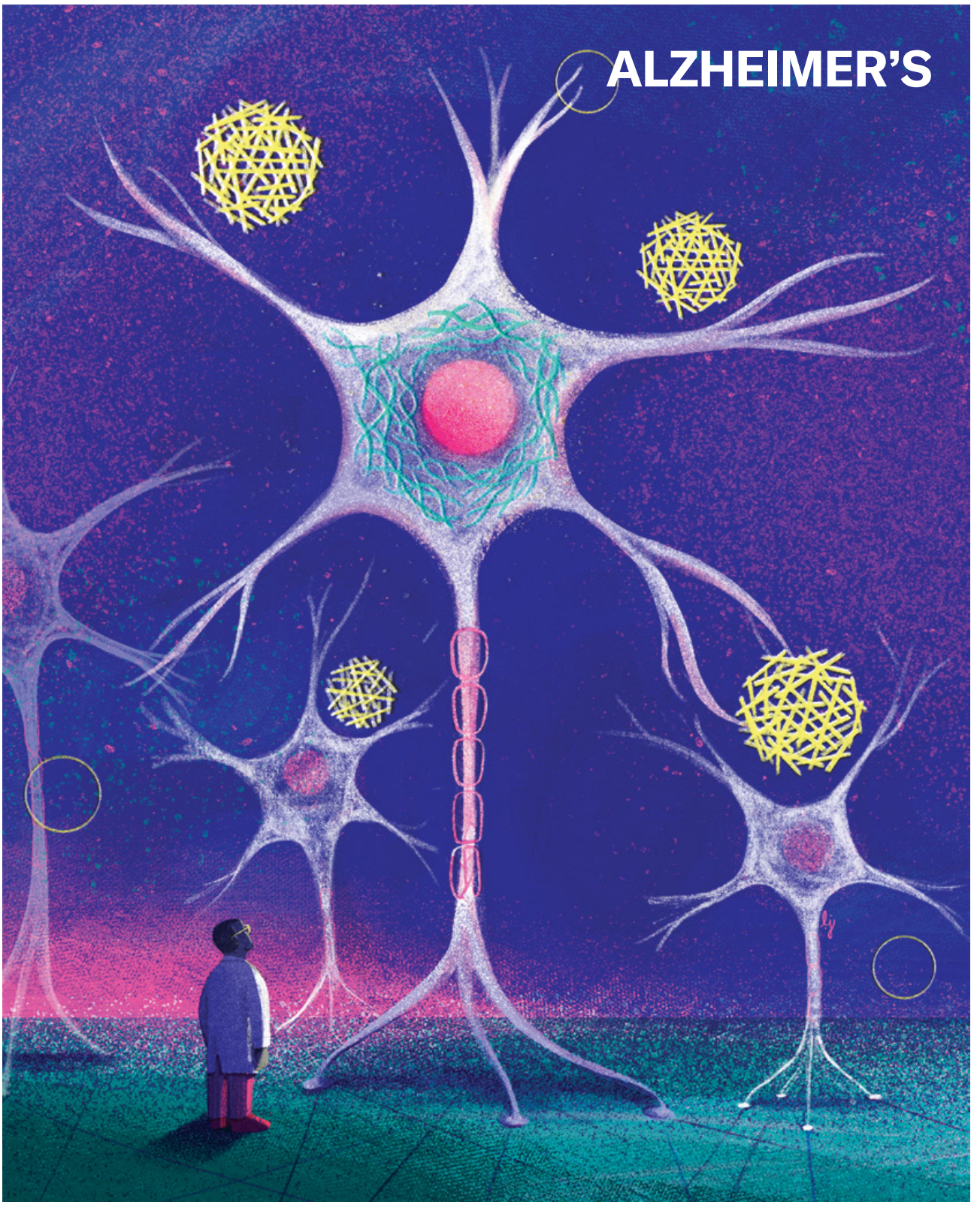

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SPECIAL REPORT

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Hope Swells in Alzheimer's Research

A DIAGNOSIS of Alzheimer's disease is typically followed by years of uncertainty, grief and a painful decline into oblivion. But although there is so much researchers still don't understand about the disease and what drives it, scientists are making progress faster than ever before and providing patients and their families with options for both diagnosis and treatment.

Over the past few decades researchers have begun to realize that Alzheimer's is more than the tangles of tau proteins and clusters of amyloid plaque that are the defining biological signs of the disease. Today, as **Esther Landhuis** describes, with the help of detailed graphics, there are more than 100 ongoing trials aimed at slowing or even stopping disease progression, and they target a variety of underlying mechanisms. The first therapies that specifically home in on and break up amyloid plaques have already been approved by the U.S. Food and Drug Administration. In clinical trials, they slowed decline for some people with early Alzheimer's, but, as **Liz Seegert** reports, the drugs also come with substantial risk and are not a one-size-fits-all solution.

Changes to daily habits, such as increased exercise and social interaction, better nutrition, and supplements, are another option to consider. **Sara Harrison** notes that although the results from studies are mixed, researchers hope that focusing on someone's day-to-day health can delay onset of the worst symptoms of dementia. Such improvements aren't available to everyone, however. Black Americans are twice as likely as white Americans to be diagnosed with Alzheimer's or other dementias. **Jyoti Madhusoodanan** analyzes the substantial evidence that this higher rate is a direct result of systemic racism, environmental pollution, and other experiences related to discrimination.

The earlier someone is diagnosed with Alzheimer's, the sooner they can begin interventions and start to plan for the future. Blood tests can finally make this early detection easier. They're not infallible, however. **Cassandra Willyard** explains that the currently available blood tests are less a screening tool and more part of a confirmatory approach, best for people already experiencing dementia symptoms.

The global incidence of Alzheimer's is increasing at a rapid rate. In the U.S., more people than ever are being diagnosed even as the number of care options dwindles. **Tara Haelle** explores the reasons for that and profiles one program aiming to help states coordinate and improve care for dementia patients and their caregivers.

Alzheimer's is a devastating diagnosis. But for the first time since the condition's initial description in 1906, scientists and clinicians are providing both dementia patients and their family members with glimmers of hope.

—**LAUREN GRAVITZ,** CONTRIBUTING EDITOR

Cultivating Resilience

Early research suggests that Alzheimer's risk can be mitigated through diet, exercise and social stimulation. But definitive studies remain elusive **By Sara Harrison**

WHEN JULI COMES HOME after work, her husband doesn't regale her with stories about his photography business the way he once did. Instead he proudly shows her a pill container emptied of the 20 supplements and medications he takes every day. Rather than griping about traffic, he tells her about his walk. When they go out to a favorite Mexican restaurant, he might opt for a side salad instead of tortilla chips with his quesadilla. "He's actually consuming green food, which is new," says Juli, who asked to be identified by only her first name to protect her husband's privacy.

Over the past year Juli's husband has agreed to change his daily habits in hopes of halting the steady progression of Alzheimer's disease, which he was diagnosed with in December 2023 at age 62. Juli and her husband are both self-employed, and their insurance plans didn't cover the positron-emission tomography scans for disease tracking that a neurologist prescribed, which would have cost thousands of dollars. So they decided to spend that money on a doctor who promises that diet and lifestyle changes can treat Alzheimer's. He recommended a keto diet, along with light cardio exercise and strength training. He also prescribed a bevy of supplements, such as creatine, which Juli's husband takes alongside the

Sara Harrison is a freelance journalist who writes about science, technology and health.

memantine and donepezil prescribed by his neurologist. Juli doesn't expect the diet and daily walks to cure her husband, but she hopes the healthy lifestyle will help manage and even improve his condition. It feels like common sense. "You stop eating fried food, you move your butt, and you feel better," she says.

Increasingly, evidence suggests that addressing health problems such as vision and hearing loss, stress, poor diet, diabetes, obesity, high cholesterol and high blood pressure can help slow or even prevent Alzheimer's symptoms. It's a tantalizingly simple solution to a complicated condition that has proved difficult to treat. For families like Juli's that have been left with a grim diagnosis and few options, lifestyle changes bring a much needed sense of hope and agency. But researchers worry about overpromising on the efficacy of these changes, especially for people already experiencing dementia symptoms. Evidence around the importance of different diets, exercises and activities—when to start them and which to prioritize—is mixed, and only in a few high-quality studies have researchers examined large, diverse groups of people. It's a promising but nascent field of research, one that scientists worry gives patients dangerous and heartbreaking hope for a cure that doesn't exist.

It's not clear whether these interventions prevent disease onset or simply delay it.

"There are a lot of claims," says Miia Kivipelto, a dementia researcher at the Karolinska Institute in Sweden. She worries about expensive but unproven regimens that promise to reverse cognitive decline, restore and protect the brain, or significantly improve cognition for people with early-stage Alzheimer's or other dementias. "Of course, people want to have hope," she says. But she cautions against making promises that can't be upheld. "It's risk reduction," she says. "That's maybe what we can promise."

Kivipelto led the Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER), a trial that enrolled more than 1,200 residents of Finland between the ages of 60 and 77. Results were published in 2017. They showed that after two years, participants who were given nutritional advice, exercise regimens and brain-training games had improved their executive function, processing speeds and complex memory by about 83, 150 and 40 percent, respectively, compared with those who didn't take those measures. Kivipelto has continued to follow that initial FINGER cohort and found that several years after the initial trial, their health in general continues to be better than that of their counterparts. The participants had a lower risk of stroke, had fewer medical emergency room visits and needed less inpatient care. Now Kivipelto is running World Wide FINGERS, a global network of studies investigating the same interventions in different countries and populations.

Similarly encouraging data have come from the Systematic Multi-Domain Alzheimer Risk Reduction Trial (SMARRT), a two-year randomized, controlled study. Researchers tested the effect of treating modifiable risk factors such as uncontrolled hypertension, social isolation and physical inactivity with more than 170 septuagenarians and octogenarians at high risk for dementia. Participants chose a few interventions to prioritize out of eight options, such as improved physical fitness or social connection. After two years, no matter which intervention people opted for, those who received individualized treatments had reduced risk factors for dementia and a 74 percent greater increase in cognition compared with their counterparts in the control group.

It's not clear whether these interventions prevent disease onset or simply delay it. At a certain point, prevention and treatment become almost the same thing: if people can postpone the onset of symptoms until they're 85 or 90 years old, Kivipelto says, "they might die of something else." A report from a commission on dementia from the Lancet Group—which comprises experts who make recommendations on health policy and practice—suggests that addressing a range of these lifestyle-based risk factors could help reduce the global incidence of Alzheimer's and dementia by 45 percent population-wide. For people with a genetic predisposition to dementia, introducing diet, exercise, and other modifications before symptoms appear might be particularly important for fending off illness.

The idea that diet and exercise could curb a disease that currently affects more than 55 million people globally is an exciting prospect. But scientists say the field is simply too young for anyone to make bold assertions that lifestyle interventions could act as treatments or cures. "We don't have mature information," says Howard Feldman, a neurologist at the University of California, San Diego.

One big caveat is that studies such as SMARRT and FINGER were conducted with people who had mild cognitive decline, not full-blown dementia. "There are people who are really exaggerating some of these claims," says Kristine Yaffe, a neurologist at the University of California, San Francisco, and the lead author on the SMARRT study. "There's very little evidence that these [interventions] work when people have the disease."

Also, the list of possible risk factors gets longer as more data emerge. When Kivipelto started FINGER, she didn't look at elements such as poor sleep and stress. But more evidence suggests that these factors could increase risk for Alzheimer's. Meanwhile interventions that had shown initial promise, such as the MIND diet—a diet geared toward brain health that combines elements of Mediterranean and hypertension-focused diets—weren't backed by further research.

Answering questions about lifestyle changes—what works, what doesn't and why—is particularly challenging because these interventions are not as easy to quantify as medications are. When researchers test pharmaceuticals, they're often investigating how a molecule interacts with a specific receptor. "We're gonna look at making sure that we've got tar-

get engagement, that we've got the right amount of medicine for the target and that we're getting the right effects," Feldman says. Nonmedical interventions don't work in that way. Take exercise: There's no particular receptor to examine. Instead exercise might lead to better blood flow in the brain. It might affect cerebral metabolism. It could affect insulin levels or increase oxygen flow. All these factors have been linked to the development of Alzheimer's in some way.

Then there's the matter of dosage: What is the right amount of exercise? How much should people exert themselves and for how long? And how can researchers assess compliance? When researchers test pills, they can easily dispense medication and count how many pills are left at the end of a trial. It's much harder to know whether someone in a lifestyle study has done the assigned exercises or whether all participants worked out at the same intensity.

Another big unknown is when these interventions should begin. Some research suggests that to reduce risk factors, middle age might be the most impactful time. Kivipelto says that it's never too late to start but that the most effective interventions may vary with age. Stress and sleep might be bigger risk factors in middle age, whereas social isolation might become more important as people grow older. "You should have a kind of check wherever you are in your life," she says.

Perhaps the biggest limitation, however, is that scientists can't measure all the biological and environmental systems at play, nor can they follow enough people for a long enough period to understand which systems are most important. One theory suggests that health interventions—such as diet, exercise and social stimulation—work because they boost cognitive reserve, or the ability of a person's brain to resist dementia. People with more cognitive reserve might not show symptoms even if they have the same pathology as someone else who is symptomatic. Researchers think being active, eating right and socializing might help build up that cognitive-reserve buffer. But they can't measure it. There is no known biomarker for cognitive reserve and no way to measure its effects over time. "It's an evolving concept," Kivipelto says.

Even while scientists work on more high-quality studies of lifestyle changes for Alzheimer's—with large, diverse patient populations, control groups, and careful measurements for the intensity of the intervention—numerous commercial companies claim to offer scientifically backed cures. These products, including the approach Juli and her husband are trying, are often based on research in predatory journals, which charge authors high fees to publish papers that look scientific but have none of the oversight of peer-reviewed publications. Others lack rigorous trials and rely only on case reports that don't describe study methods and can't be replicated. Still others haven't been tested in large groups or in humans at all. For example, small studies have suggested ketosis could help improve cognition, but no large-scale clinical trials have tested the hypothesis. Similarly, creatine supplements have shown promise in mice but have not been tested extensively in humans. No large, high-quality clinical trials have shown that supplements can improve human cognition or brain health, but companies selling these products now represent an industry valued at more than $6 billion globally.

Some people spend their life savings to follow a protocol that requires them to remediate mold in their homes, even though the evidence linking mold and dementia is debated. Other families report that sticking to a restrictive diet ultimately feels cruel when a parent or spouse has few pleasures left. Neurologist Joanna Hellmuth, then at the University of California, San Francisco, wrote an article in 2020 in the *Lancet Neurology* about pseudoscience and dementia, warning that fraudulent solutions can be financially and emotionally harmful for families. "Hope is important in the face of incurable diseases and intuitive interventions can be compelling," she wrote. "However, unsupported interventions are not medically, ethically, or financially benign, particularly when other parties might stand to gain."

Even under the best of circumstances, changes to diet and exercise cannot ward off Alzheimer's for everyone. Yaffe has seen patients who play bridge, go running and practice über-healthy lifestyles only to be astonished to learn they also have Alzheimer's. "There's something called bad luck, and there's something called genetics," she says. Scientists measure the impact of lifestyle modifications in population-wide estimates that don't translate to individual risk. Diet, exercise, hearing aids, and other interventions might reduce the global incidence of dementia by 45 percent, but that doesn't mean they will reduce your specific risk by the same amount. Yaffe estimates that roughly half of a person's Alzheimer's risk is based on genetics, and half probably depends on their activity level, diet and luck. But the biggest risk factor is age.

Even as Juli is gently prodding her husband to eat more broccoli, she's also preparing for his inevitable decline. The couple is in the process of moving from their two-story home in a Dallas suburb to a single-story house they are having built in a nearby gated community. Her husband will trade in his car for a golf cart, and Juli will work almost entirely from home to make sure he stays safe. She knows they are incredibly lucky to be able to afford to build their new home from the ground up. She's already designed it with a shower and doors wide enough to accommodate a wheelchair.

Juli acknowledges that it's impossible to know whether the changes to their health routines are working. There's no control group, no way to assess how her husband's disease might have progressed if they'd stuck to only medications. Right now they can afford the supplements ($150 per month), extra visits to doctors ($900 per hour twice a year), blood draws ($500 every six months), and memberships to their doctor's practice and to a platform that promotes the protocol they are following ($3,000 per year).

For Juli, the costs are justified by the change she sees in her husband. Their daily regimen gives him a sense of agency, which has alleviated some of the anxiety and depression that plagued him after his diagnosis. "It's given him work to do—and hope," she says. "If that's all we take away from it, it's worth it."

A Multipronged Assault

A new understanding of Alzheimer's is leading to a variety of new treatment approaches

By Esther Landhuis | Graphics by Now Medical Studios and Jen Christiansen

ALZHEIMER'S DISEASE HAS PROVED to be a tricky target, and researchers and drug developers have been pursuing effective treatments for decades. Debates rage over the disorder's underlying causes, and various approaches have faced one hurdle after another. But the field has reached a turning point. Over the past four years the U.S. Food and Drug Administration has approved several therapies that address some of the condition's potential biological roots rather than merely mitigating symptoms—a key scientific milestone. Despite the advances, however, there is still a long list of open questions and so much work to be done.

The brains of people who die with Alzheimer's show a distinct biology: clumps or "plaques" of amyloid beta proteins in spaces between neurons and tangles of tau proteins that accumulate primarily within the nerve cells. One prevailing theory holds that amyloid builds up early, and tau tangles develop when nerve cell damage is underway but cognitive symptoms are not yet apparent. Over time these pathogenic, or disease-causing, proteins disrupt nerve cell communication. The newest treatments—lecanemab and donanemab—bind to amyloid beta proteins, clear them from the brain and modestly slow cognitive decline.

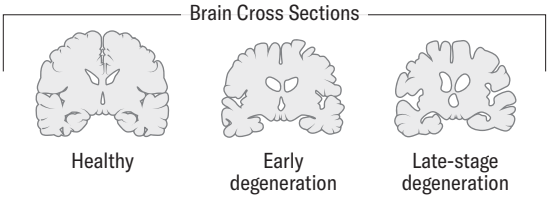
But the progression from disease-linked proteins to actual dementia is long and inexact, and amyloid and tau proteins accumulate in people with other neurodegenerative disorders, too. With Alzheimer's there is often a 20- to 30-year lag between the initial detection of amyloid and obvious cognitive decline. According to one study that predicted disease risk based on demographic data, death rates and amyloid status, fewer than one quarter of cognitively healthy 75-year-old women who test positive for amyloid in a spinal fluid analysis or positron-emission tomography (PET) brain scan will develop Alzheimer's dementia during their lifetime. Such findings suggest that amyloid alone is not driving disease progression and have spurred scientists to investigate other strategies.

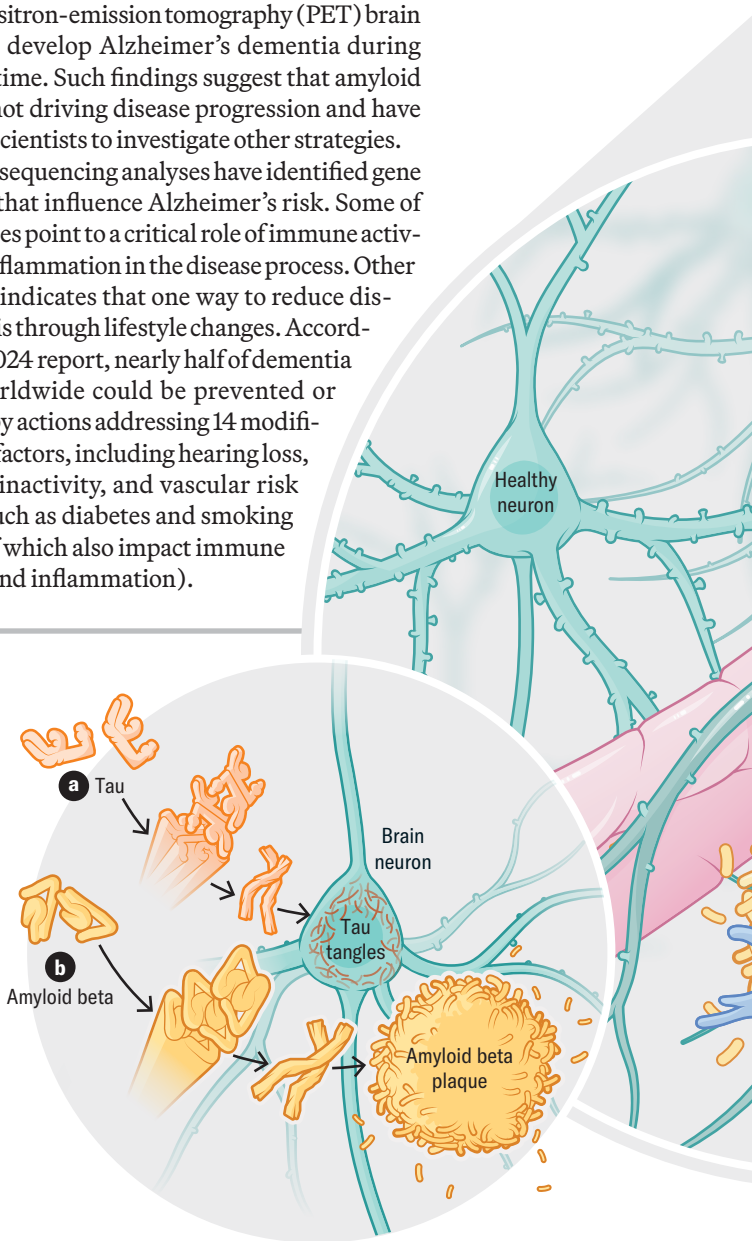
DNA-sequencing analyses have identified gene variants that influence Alzheimer's risk. Some of these genes point to a critical role of immune activity and inflammation in the disease process. Other research indicates that one way to reduce disease risk is through lifestyle changes. According to a 2024 report, nearly half of dementia cases worldwide could be prevented or delayed by actions addressing 14 modifiable risk factors, including hearing loss, physical inactivity, and vascular risk factors such as diabetes and smoking (many of which also impact immune activity and inflammation).

Esther Landhuis is a journalist in the San Francisco Bay Area. She holds a Ph.D. in immunology and covers biomedicine in all dimensions, from bench discoveries to biotech to health care.

The Basics

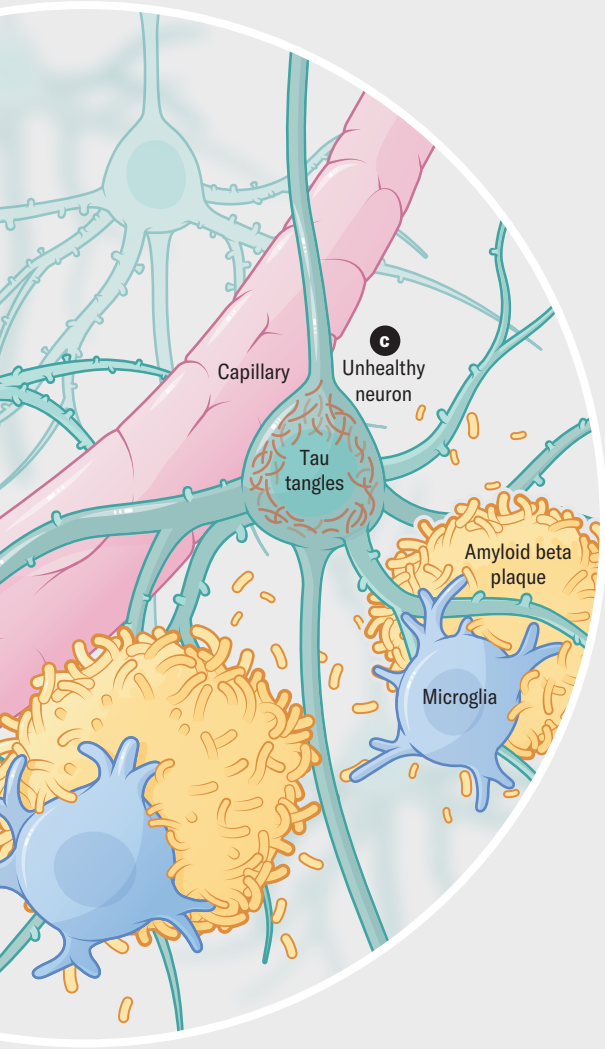
A well-known hallmark of Alzheimer's disease is the buildup of tau (a) and amyloid beta (b) proteins in the brain. Over time plaques and tangles cause neuron damage (c) and cell death. But most Alzheimer's patients have accumulated other proteins, too, such as alpha-synuclein, as well as blood vessel damage that can appear before amyloid plaques. Recent evidence suggests that inflammation, immune processes and vascular risk factors also play a key role in the disease.





Treatment Targets

There are more than 100 ongoing clinical trials testing a variety of interventions, each of which targets one or more potential contributors to dementia. "We will get there in stages," says Sudha Seshadri, a neurologist and founding director of the Glenn Biggs Institute for Alzheimer's and Neurodegenerative Diseases at UT Health San Antonio in Texas. "The amyloid-lowering treatments are a piece of it. Immune-modulating drugs are probably going to be a piece of it," she says. It will also be important to control for vascular risk, she adds, which "is important regardless of what else is happening."



The mechanisms listed here are considered key elements of Alzheimer's risk:

Neurotransmitter receptors
Proteins on nerve cell surfaces that receive signals and play a critical role in memory and learning. Some drugs for Alzheimer's block harmful activity at these receptors, and others boost activity by preventing the breakdown of neurotransmitters.

Amyloid
A protein that, when misfolded, can build up outside of nerve cells in the brain and form plaques that disrupt neural function. Several therapies aim to dissolve these deposits.

Inflammation and immune processes
These can be activated by the buildup of abnormal forms of amyloid and tau in the brain, leading to nerve cell damage and cognitive decline.

Tau
A protein that typically helps to maintain a cell's structure and shape. It can build up within neurons and form tangles, which are a hallmark of Alzheimer's, along with amyloid plaques. Some new therapies attempt to target pathological forms of tau, preventing these proteins' aggregation and enhancing their clearance.

Synaptic plasticity and neuroprotection
Ways in which the brain modifies connections between neurons during learning and memory and protects neurons from damage. Both processes can be disrupted in people with Alzheimer's.

Metabolism
The process by which the brain breaks down nutrients. In Alzheimer's patients, PET scans show abnormally low glucose metabolism in brain areas important for memory and cognition. Some therapies aim to increase the metabolism of glucose, which the brain needs for energy.

Vasculature
Specialized blood vessels in the brain form a dynamic interface known as the blood-brain barrier, which helps to supply nutrients to nerve cells and protect the brain from pathogens. Breakdown of this barrier has been associated with multiple neurodegenerative diseases, including Alzheimer's.

Growth factors and hormones
Proteins that can protect brain cells from damage and stimulate the production of new cells. Some therapies aim to replenish them and provide an extra layer of protection.

Epigenetic regulators
Proteins and other molecules that turn a gene on or off without changing the underlying DNA sequence. In doing so, they can influence the accumulation of misfolded amyloid, misfolded tau, or other pathogenic proteins.

Neurogenesis
Neural growth. Damaged nerve cells can contribute to memory impairment and cognitive decline in people with Alzheimer's. Promoting the growth of new nerve cells could help slow that decline.

Oxidative stress
An unhealthy overabundance of free radicals relative to antioxidants in the body, which can harm nerve cells and disrupt their function. Some therapies aim to relieve oxidative damage by enhancing the activity of specific enzymes that neutralize free radicals.

Proteostasis
A process that maintains proper protein folding, synthesis and degradation in cells and can become impaired in people with Alzheimer's. Some experimental interventions promote cellular pathways that prevent misfolded proteins or that facilitate their removal.

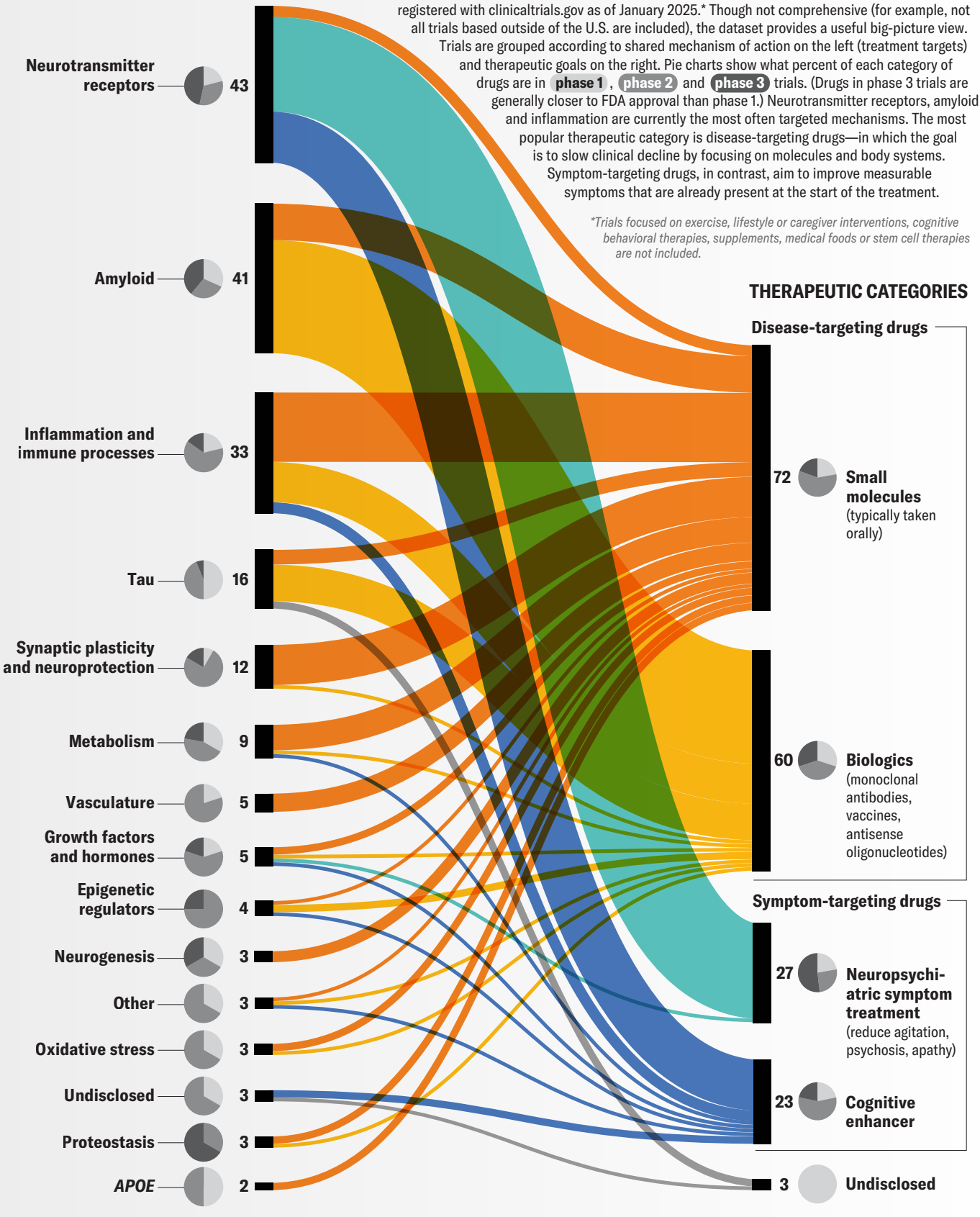
APOE
Abbreviation for apolipoprotein E, a protein-encoding gene that has three major forms: ε2, ε3 and ε4. *APOE* ε4 is the strongest genetic risk factor for Alzheimer's. People have two copies, or alleles, of *APOE*. Having one *APOE* ε4 allele can increase someone's lifetime risk of disease up to fourfold over that of people with two copies of the most common variant, *APOE* ε3; two *APOE* ε4 alleles can raise the risk up to 25-fold. People with two *APOE* ε3 alleles are 40 percent more likely to develop Alzheimer's than those with one ε3 and one ε2.

TREATMENT TARGETS

A VARIETY OF DRUG APPROACHES

This chart includes 138 drugs across 182 clinical trials—pharmacological Alzheimer's trials registered with clinicaltrials.gov as of January 2025.* Though not comprehensive (for example, not all trials based outside of the U.S. are included), the dataset provides a useful big-picture view. Trials are grouped according to shared mechanism of action on the left (treatment targets) and therapeutic goals on the right. Pie charts show what percent of each category of drugs are in **phase 1**, **phase 2** and **phase 3** trials. (Drugs in phase 3 trials are generally closer to FDA approval than phase 1.) Neurotransmitter receptors, amyloid and inflammation are currently the most often targeted mechanisms. The most popular therapeutic category is disease-targeting drugs—in which the goal is to slow clinical decline by focusing on molecules and body systems. Symptom-targeting drugs, in contrast, aim to improve measurable symptoms that are already present at the start of the treatment.

**Trials focused on exercise, lifestyle or caregiver interventions, cognitive behavioral therapies, supplements, medical foods or stem cell therapies are not included.*



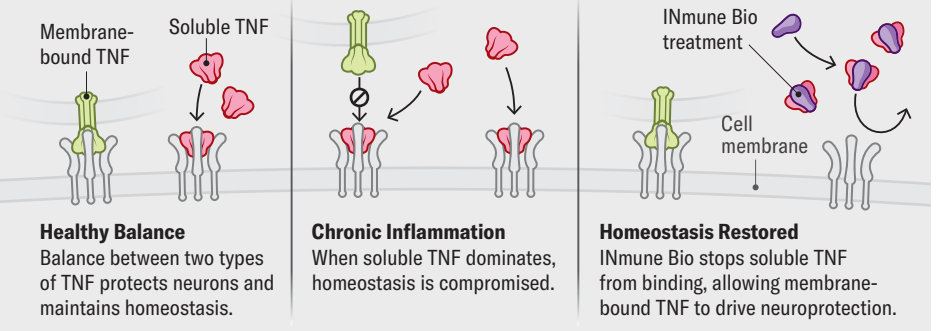
Source: "Alzheimer's Disease Drug Development Pipeline: 2025," by Jeffrey L. Cummings et al., in *Alzheimer's & Dementia: Translational Research & Clinical Interventions*, Vol. 11: April–June 2025 (*data*)

Intervention Case Studies

While drugs that target the proteins amyloid and tau have garnered heavy attention and funding, other therapeutic approaches have focused on other biological mechanisms in Alzheimer's disease. Examples of the latter group are described below.

Inflammation

Keeping nerve cells healthy and protecting them from damage requires balance between soluble and membrane-bound forms of a protein called TNF-α. In conditions of immune dysfunction, a rise in soluble TNF-α disrupts the healthy balance, or homeostasis, and drives up inflammation. One company (INmune Bio) is developing a drug that binds and deactivates soluble TNF-α, restoring health-promoting effects of the transmembrane form.



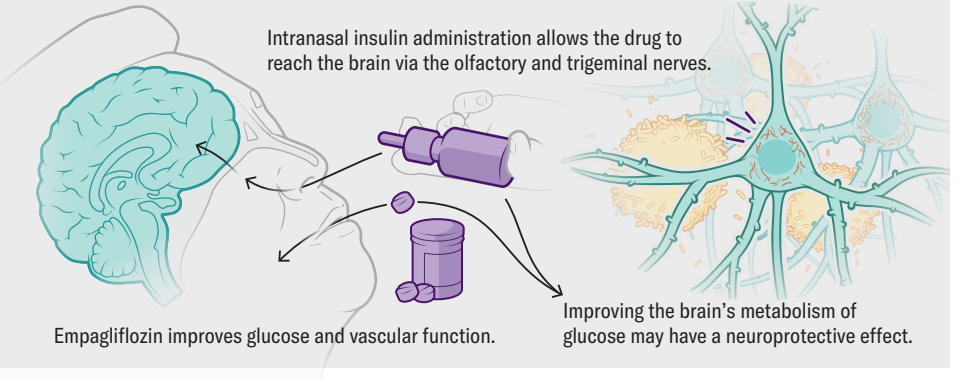
Healthy Balance
Balance between two types of TNF protects neurons and maintains homeostasis.

Chronic Inflammation
When soluble TNF dominates, homeostasis is compromised.

Homeostasis Restored
INmune Bio stops soluble TNF from binding, allowing membrane-bound TNF to drive neuroprotection.

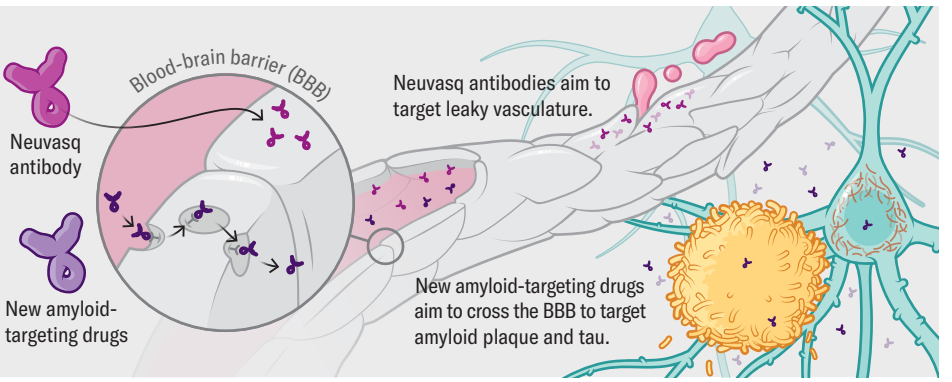
Metabolism

Insulin, a hormone that regulates energy availability and brain cell communication, also helps to maintain immune and vascular health. A clinical trial led by Suzanne Craft of the Wake Forest University School of Medicine is testing intranasal delivery of insulin and the diabetes drug empagliflozin in people with early Alzheimer's or mild cognitive impairment.



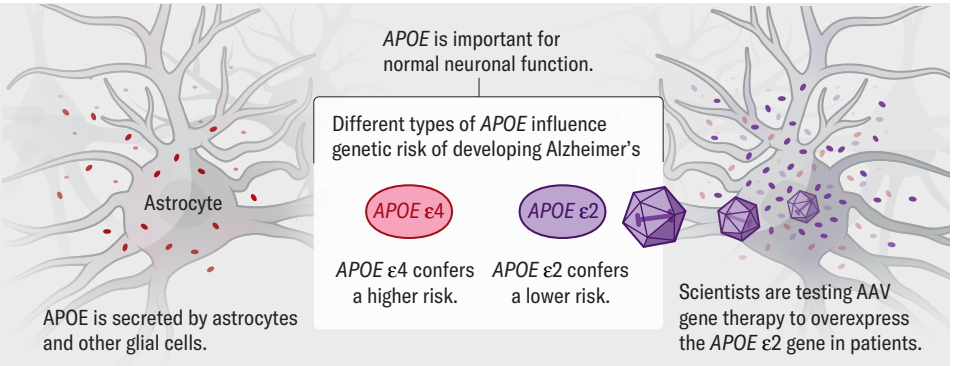
Vasculature

Several companies are developing new versions of amyloid antibodies that can penetrate the brain more effectively—for instance, Roche's trontinemab. At least one company, Neuvasq, is developing an earlier-stage intervention that targets the vasculature itself, to test whether a less leaky blood-brain barrier could slow disease progression.



APOE

Some therapeutic strategies aim to reduce or get rid of the *APOE* ε4 risk variant, increase protein production by the protective gene form, *APOE* ε2, or edit the *APOE* ε4 gene to the *APOE* ε2 version. In a small gene therapy study, Lexeo Therapeutics used modified viruses to deliver the *APOE* ε2 gene into the spinal fluid of 15 Alzheimer's patients who had the more dangerous *APOE* ε4 variant.



A Dangerous Silver Bullet

Drugs that hit an Alzheimer's target are gaining traction. Some neurologists remain dubious

By Liz Seegert

ONE OF NEUROLOGIST Anelyssa D'Abreu's least favorite tasks is giving her patients a dreaded diagnosis: early-stage Alzheimer's disease. But it's not quite as bad as it used to be. Today when they ask, "Is there anything we can do?" D'Abreu has a new answer: "Perhaps."

Unlike a decade ago, when D'Abreu had little to offer her patients with Alzheimer's, there are now drugs that may impede the disease's progression. The difficulty with this approach, however, is that it comes with a trade-off. The new medications carry the risk of serious side effects, including brain bleeds, stroke-like symptoms and even death. Yet they also come with hope, something new for Alzheimer's patients and their families.

Drugs in this class, known as anti-amyloid therapies, have not gained much traction. In limited studies, they have been shown to slow or even decrease one of the biological symptoms of Alzheimer's: the accumulation of amyloid beta in the brain. Nearly four dozen studies on these drugs have been conducted since 2018, and collectively they indicate that anti-amyloid therapies may marginally reduce the rate of cognitive decline. Some experts say that could offer perhaps an additional year of independence. But the clinical trials completed to date rely on only 18 months' worth of published data, and their success has been tempered by the drugs' significant downsides. Additionally, the framing of these drugs' success has come under criticism.

D'Abreu, who heads the University of Virginia neurology department's cognitive and behavioral neurology division, was initially apprehensive about offering antiamyloid treatments to her patients with early-stage Alzheimer's. A relatively high percentage of participants in the anti-amyloid studies experienced brain swelling and microbleeds, events known as amyloid-related imaging abnormalities (ARIA), which can lead to disability or even death. Up to 40 percent showed brain swelling, and up to 28 percent had brain bleeds. D'Abreu wasn't the only physician who hesitated over such potentially severe side effects.

In general, researchers and clinicians were highly skeptical of these drugs when they were introduced. They had shown promise in clinical studies but are only now yielding enough data in real-world scenarios for scientists to gain a better understanding of their efficacy. After much thought, D'Abreu decided it was important to offer her patients the option. When people are functionally independent, she says, delaying progression toward full-blown Alzheimer's is a big deal. "If it really slows down a person in the mild-cognitive-impairment stage, that makes a huge difference," she says. Among the 50 or so people at her hospital who have received the therapy so far, none have experienced any serious adverse effects.

Alzheimer's affects about 7.2 million people over age 65 in the U.S., according to the Alzheimer's Association, and about 74 percent of them are 75 or older. Scientists have been seeking treatments for decades; because amyloid beta plaques can begin accumulating long before noticeable symptoms appear, most efforts aimed to clear them from the brain and prevent the formation of new ones. In 2021, when the U.S. Food and Drug Administration fast-tracked the first anti-amyloid therapy, some hoped it would be what patients and providers had been waiting for: a drug that could stop Alzheimer's in its tracks.

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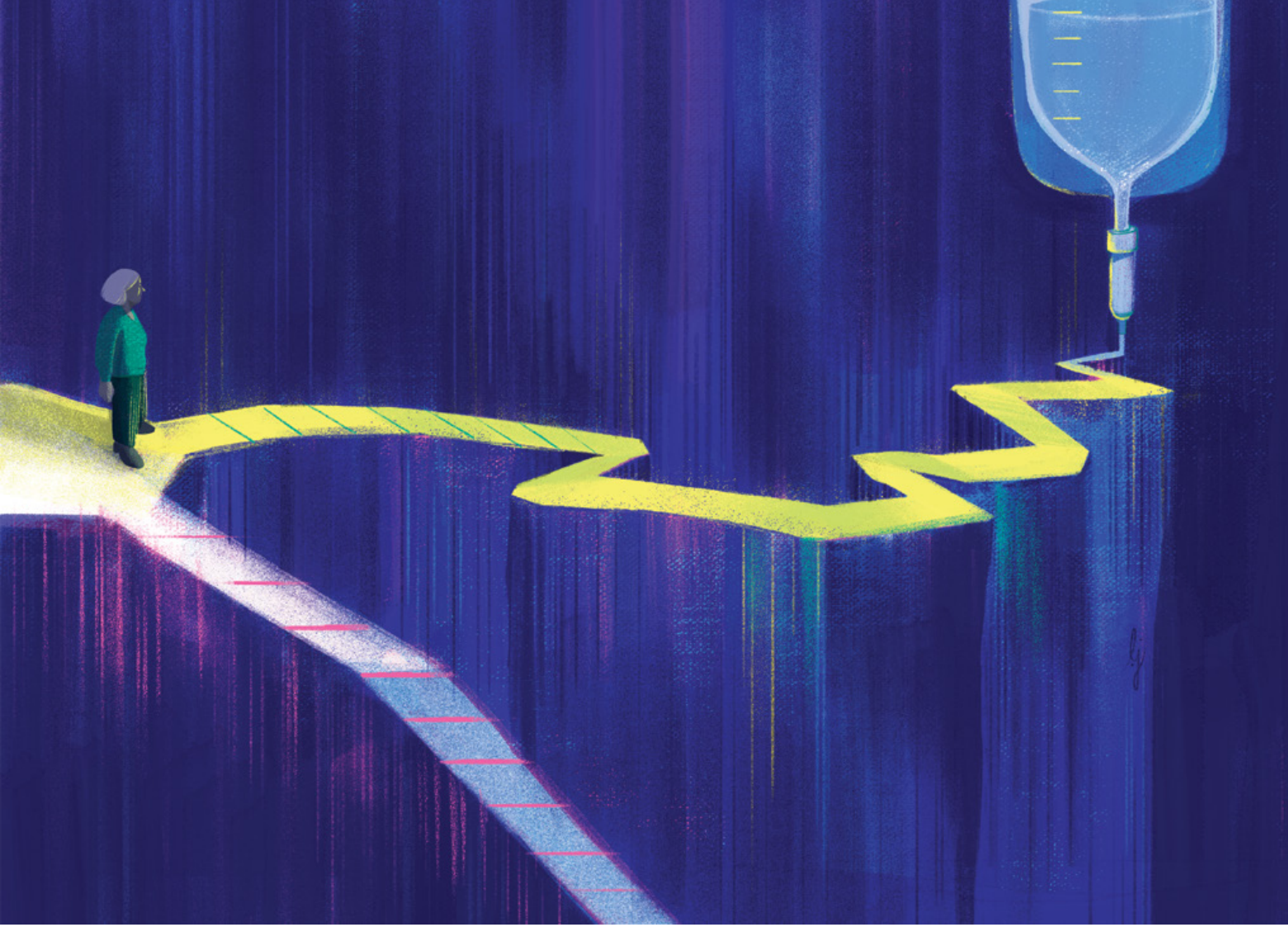
Aducanumab, marketed by manufacturer Biogen as Aduhelm, got the green light from the FDA under the agency's accelerated-approval pathway. It was the first medication to target, reduce and remove amyloid beta plaques. There was little evidence, however, that amyloid beta clearance correlated with slowed cognitive or functional decline. And the drug introduced the risk of ARIA, in addition to being riddled with other problems: controversial clinical-trial results, skepticism from the FDA's own advisory committee, an initial average annual price of $56,000, and refusal by the Centers for Medicare and Medicaid Services to cover the cost without additional clinical evidence of efficacy. Just 31 months after its approval, Biogen announced it was removing aducanumab from the market.

Since then, the FDA has approved two more anti-amyloid treatments: lecanemab (Leqembi), made by Eisai in partnership with Biogen, and donanemab (Kisunla) from Eli Lilly. Both slowed cognitive decline better than aducanumab or placebo in clinical studies. But both also come with a risk of ARIA. In the phase 3 clinical trial for lecanemab, which assessed efficacy and safety in large groups of people, about 9 percent of participants taking a placebo had brain swelling or hemorrhages, compared with 17.3 percent of those in the lecanemab group. In four separate donanemab trials, up to 30.5 percent of the participants showed brain abnormalities, compared with 0.8 to 7.2 percent in the placebo groups, and three deaths related to ARIA were attributed to the drug. Both therapies are also expensive—an average annual price of $26,500 for lecanemab or $32,000 for donanemab, plus hundreds to thousands more for required brain scans and other monitoring.

These therapies are not an option for everyone with Alzheimer's. They are recommended only for patients at early disease stages, and people most at risk for ARIA should avoid them. To identify the best candidates, D'Abreu and other neurologists put their patients through extensive cognitive assessments, costly positron-emission tomography scans to look for amyloid in the brain that would



help them diagnose the condition, and tests to determine whether they carry the gene variant *APOE* ε4, which increases dementia risk and the likelihood of brain swelling or bleeding in people taking anti-amyloid medications.

Despite the improved ability to assess risk, some neurologists remain highly dubious of available anti-amyloid therapies, as well as of the hypothesis they're based on: that amyloid is the root cause of the disease. James Burke, a neurologist at the Ohio State University Wexner Medical Center, was skeptical when lecanemab was approved in 2023 and says there still isn't enough clinical evidence to change his mind. Researchers have been collecting data beyond the 18-month time frame but currently have no good understanding of the drugs' longer-term effects.

Burke thinks it's important to draw a line between statistically significant changes, such as cognitive decline slowing by a reported 27 percent with a drug compared with a placebo, and those that are clinically meaningful, such as whether patients can drive safely or care for themselves with minimal assistance. "It's not obvious that people are even going to know the benefit is there," he says, but "the harms are very substantial and almost certainly badly underestimated." He notes that those harms, which include strokes and deaths that some attribute to the drugs, have occurred in rigorously controlled settings that do not necessarily reflect real-world conditions. Trial participants often are healthier and younger, on average, than typical dementia patients.

Burke is resigned, however, to the inevitability of prescribing anti-amyloid therapy for patients who meet the criteria. "If that's what they want, there's no point, for a provider who has access to treatment, in putting up a wall. They'll just get the treatment someplace else." But he also focuses on other approaches, such as helping people reduce vascular risk factors, eat a healthier diet and exercise more.

For now these drugs are the best pharmaceutical interventions on offer, says Judith Heidebrink, a neurologist and cognitive-disorder specialist at the University of Michigan Medical School. She was involved in the lecanemab phase 2 trial and its open-label extension. "Even given these risks," she says, those taking the drug are, on average, "more likely to maintain a higher level of independence and have slower disease progression."

That was what 80-year-old Bob Merriman was hoping for. He had seen both his parents and a brother ravaged by Alzheimer's. He knew his odds of developing it were high, and he desperately wanted to avoid the same fate. His wife, Mary, says he had signs of confusion and was getting easily frustrated with simple tasks.

Merriman reached out to his physician after hearing about anti-amyloid therapies and was referred to Heidebrink for evaluation. After extensive cognitive

testing, magnetic resonance imaging and blood work to determine whether he had cognitive impairment or a genetic predisposition to Alzheimer's (he did), Merriman began receiving biweekly infusions of lecanemab last November. He was willing to accept the potential risks and is checked regularly for signs of ARIA.

"He was determined," Mary says. "He was like, 'No, I know what the alternative is.'" She adds that he seems more focused than before and plans to continue taking the treatment for as long as possible. As anti-amyloid drugs edge into the mainstream, they are enabling additional research that can better predict who might be most susceptible to brain swelling and microbleeds, along with improved ways to find and manage potential risks. The result is increased confidence in these therapies among neurologists who might prescribe them.

Many patients who take lecanemab seem to share this confidence and, like Merriman, feel the drug helps them on some level. (Lecanemab has been on the market longer than donanemab.) D'Abreu and other neurologists say most of their patients choose to complete the initial 18-month course and often continue with maintenance therapy. That's helped sway D'Abreu's thinking on the medication, but she is not yet convinced of its efficacy. Because it's possible for patients with mild cognitive impairment to remain stable for months without treatment, she says she can't be certain how large a role anti-amyloid therapy plays.

Burke remains highly skeptical that the benefits of these therapies outweigh the risks. "This medicine can cause bleeding in the brain in one in 200 people," he says. "It's not a safe or benign medicine."

Fewer neurologists are sitting on Burke's side of the fence these days, however. More than two years after lecanemab was approved, overall hesitancy among practitioners in the field has shifted. In February 2024 market research firm Spherix surveyed 75 neurologists and found that fewer than half of them recommended lecanemab to their patients. They cited low satisfaction with the data and frustration with issues such as insurance coverage, logistics surrounding infusion access, and burdensome follow-up testing. A year later, however, 80 percent of those surveyed said they were now discussing anti-amyloid therapies with their patients. The average number of patients on lecanemab per surveyed neurologist has increased about fivefold. There are not enough data yet to gauge the acceptance of donanemab, which received full FDA approval in July 2024.

As the use of anti-amyloid medication becomes more widespread, there's also a need to better understand what happens when people on these therapies come into the emergency room experiencing a stroke or a blood clot, conditions that would usually be treated with drugs to induce thrombolysis, breaking up the clot. "Right now our data are incredibly limited, but there's a bunch of case reports of truly catastrophic bleeding when people are on amyloid-lowering agents and then get thrombolysis," Burke says. These concerns have become common enough that a report was recently published in *JAMA,* the most widely circulated medical journal, to help clinicians weed through the details.

> For now these anti-amyloid therapies are the best pharmaceutical interventions we have on offer.

The hypothesis that amyloid beta is a root cause of cognitive decline is popular, and it's where the major drug companies have placed much of their focus. But it's not the only one, and controversy has plagued it for decades. It's been the subject of allegedly manipulated studies, and some assert academic institutions and government agencies have funneled research dollars to support this approach. The first positive results from aducanumab were preceded by a long line of failures.

Even ardent proponents of the anti-amyloid theory agree that additional methods for treating Alzheimer's are necessary. One idea is to use combination therapy, similar to how HIV or cancer drugs are administered, according to geriatrician Howard Fillit, co-founder and chief science officer of the Alzheimer's Drug Discovery Foundation. He says trials are underway for other therapies that target tau proteins in the brain, as well as inflammation and various metabolic pathways, all of which contribute to disease progression [see "A Multipronged Assault" on page S6].

There also are ongoing trials to determine whether anti-amyloid drugs administered before symptoms emerge can delay or even prevent the onset of Alzheimer's. The AHEAD 3-45 study, which comprises two trials, is testing whether the approach is effective against preclinical Alzheimer's—when amyloid plaque builds slowly and silently in the brain. If the amyloid hypothesis is correct and these clumps of protein are the primary cause of Alzheimer's, presymptomatic therapy could remove or prevent the formation of these plaques early on, thereby halting disease altogether. If the trials are successful, researchers may find that "we've actually delayed the inevitable clinical course for some of these patients," says Lon Schneider, a neurologist and gerontologist at the University of Southern California's Keck School of Medicine. The study should be completed in 2031.

D'Abreu's center at the University of Virginia is participating in a longer-term trial of donanemab, comparing the daily function of patients who are taking the drug versus those who are not. She still has concerns about the risks of anti-amyloid therapy, but as more data become available, she is increasingly comfortable about its safety and efficacy. More research could provide a more nuanced understanding of whether these drugs make a difference for patients and their care partners or whether the marginal improvement is not worth the untenable—and potentially lethal—burdens. D'Abreu remains cautiously optimistic.

Decoding Blood

New biomarkers promise easier and earlier detection of Alzheimer's, but the results aren't always clear

By Cassandra Willyard

THE FIRST HINTS that Gregory Nelson might be having cognitive troubles were subtle. So subtle, in fact, that his doctor assured him nothing was wrong. "Everyone who hits a certain age just misses words," Nelson remembers him saying. When Nelson got home, he regretted not pushing harder for a referral. His entire family had noticed changes. Nelson, who is 70, scheduled another appointment and convinced his physician to send him to a neuropsychologist.

Nelson's greatest fear was that he was in the beginning stages of Alzheimer's—his father, though never formally assessed, had probably died of the disease. But Nelson didn't get a diagnosis, at least not right away. He waited months to be seen by the neuropsychologist his physician referred him to. That specialist performed a cognitive assessment, which indicated mild cognitive impairment, but the doctors couldn't yet tell him the cause. The neurologists in his area were completely booked, so there was another lengthy wait between his primary-care visit and his diagnosis of Alzheimer's.

The delay gave Nelson ample time to prepare for the worst, but it also created a lot of uncertainty and anxiety. "The bummer about waiting is that your brain just goes all over the place," he says. It would have been nice to have a definitive answer sooner, he says—to be able to say, "This is it, man. This is the diagnosis."

A quicker, simpler path to early diagnosis may be in sight with recently developed blood tests that can indicate whether someone has known markers for Alzheimer's. Unlike other diagnostic tools, such as spinal taps and positron-emission tomography (PET) scans, blood tests are relatively cheap and simple to perform. And they can help differentiate Alzheimer's from other neurodegenerative conditions and medical problems, potentially hastening access to specialists and therapies. The sooner someone knows they have Alzheimer's, the sooner they can plan for the future and assess possible interventions: Anti-amyloid treatments have been shown to modestly slow disease progression when given early in the course of the disease. Lifestyle changes also seem most effective when adopted before symptoms get too advanced.

But some experts worry that because these blood tests are so simple to perform, physicians might order them right away to provide a quick diagnosis without carrying out a fuller workup on the patient. The current tests come with several caveats that make interpretation tricky, especially for people who have no symptoms of dementia. A positive result doesn't guarantee that the person will develop cognitive problems. And there aren't any approved therapies for people without symptoms. "This is not a mass screening test," says Nathaniel Chin, a geriatrician and medical director for the Wisconsin Alzheimer's Disease Research Center in Madison. "This isn't something we just give willy-nilly."

Alzheimer's is named for German psychologist Alois Alzheimer, who first identified the hallmarks of the illness in the brain of someone with dementia: abnormal clumps of a peptide called amyloid beta wedged between brain cells, along with tangled filaments of a protein called tau. Although some tests can detect amyloid and tau while patients are alive, examining the brain postmortem is the only definitive way to diagnose the disease.

In living patients, neurologists typically rely on a battery of tests. Chin starts with a clinical history and cognitive testing. "You have to diagnose mild cognitive impairment or dementia first," he says. Cognitive testing can reveal patterns suggestive of Alzheimer's, but it's not proof positive. Vitamin deficiencies, certain medications, and multiple other factors can cause dementialike symptoms. "So truly, to know if it's Alzheimer's, you'd want to confirm it biologically," Chin says.

That kind of confirmation typically involves detecting amyloid beta and tau either in the brain with a PET scan or in cerebrospinal fluid with a lumbar puncture. Compared with the general population, people with Alzheimer's tend to have lower levels of amyloid beta and higher levels of a dysfunctional form of tau called phosphorylated tau, or p-tau, in their spinal fluid. Such tests are expensive and can't be done in a regular doctor's office. As a result, more than half of Alzheimer's cases—between 50 and 70 percent—are missed or misdiagnosed.

That's why there is so much excitement around blood tests. They're cheap, simple to perform and scalable. Researchers have spent more than a decade trying to figure out which blood biomarkers work best for disease detection, and to date p-tau217 seems to align most closely with results from PET imaging and spinal fluid analysis. When researchers compared several blood tests, they found that the level of p-tau217 had the strongest correlation to PET scan measurements of amyloid and tau levels in the brain.

Roughly a dozen blood tests are currently available to physicians, but only one, Lumipulse, has been approved by the U.S. Food and Drug Administration. The rest are available as lab-developed tests, which can be marketed without FDA approval. Accuracy varies, but some appear to perform as well as spinal fluid tests.

Amyloid beta can begin accumulating in the brain a decade or more before symptoms of Alzheimer's emerge, so many people hope that blood tests could eventually be used to spot the disease far earlier than is possible today. "There's this whole 10 or 20 years where interventions could potentially be done," says Zaldy Tan, a memory and aging specialist at the Cedars-Sinai Medical Center in Los Angeles. There is some evidence that

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higher-quality education and improved diet, exercise and social stimulation could help ward off symptoms [see "Cultivating Resilience" on page S3]. And preventive therapies are in the works. Anti-amyloid medications that have reached the market over the past couple of years are currently approved only for those with an Alzheimer's diagnosis, but ongoing trials aim to test whether they might also help stave off symptoms in people who have biological but not behavioral signs of disease [see "A Dangerous Silver Bullet" on page S10]. "What we're really trying to do is extend someone's functional independence and their ability to maintain a high standard of living," says Joel Braunstein, president and CEO of C2N Diagnostics, a St. Louis–based company that sells blood tests for Alzheimer's. "If we can forestall people from developing what we call clinical Alzheimer's disease, we will have made a profound impact."

Today, however, most experts agree that only symptomatic people should get blood tests. Lumipulse, for example, is approved for patients 55 years or older who have signs of Alzheimer's. In that group, the test has proven accuracy: about 92 percent of people with positive results had evidence of amyloid plaques on a PET scan or in their spinal fluid, and more than 97 percent of those who tested negative had no evidence of plaques.

For those who are asymptomatic, however, it's not clear whether blood tests would be useful. In this group, "we know we will have more false positives because the prevalence of the disease is lower," says Alicia Algeciras-Schimnich, a clinical chemist at the Mayo Clinic in Rochester, Minn. In fact, the field doesn't have a standard way to interpret blood test results for people who don't show signs of cognitive decline.

Does a positive test equal Alzheimer's? It depends.

Last year the Alzheimer's Association published revised criteria that rely on positive biomarkers—in the blood or elsewhere—to diagnose disease, regardless of whether the patient is symptomatic. Heather Snyder, the Alzheimer's Association's senior vice president of medical and scientific relations, says this approach echoes the criteria used for other diseases with known mechanisms. "Defining a disease by its biology rather than symptoms has been the status quo for years in other areas of medicine such as cancer, heart disease and diabetes," she says. "The Alzheimer's field is now making similar progress."

Many neurologists find this change problematic. Although abnormal blood levels of tau and amyloid put someone at risk of developing symptoms, they say little about the level of risk or the timeline for progression, says Nicolas Villain, a neurologist at the Pitié-Salpêtrière Hospital's Institute of Memory and Alzheimer's Disease in Paris. "It's an increased risk, but we cannot be more specific," he says. "It's not very informative for the individual to have this information."

In one recent study, researchers analyzed results from more than 2,100 adults who did not have dementia but had taken blood tests to measure amyloid beta, p-tau, and a few other biomarkers. Over the course of 16 years, 212 of the subjects developed Alzheimer's. The researchers found that the tests did an excellent job of predicting who would not develop the disease but a poor job of predicting who would. Less than 25 percent of those who tested positive developed Alzheimer's during the follow-up period.

Second, no proven preventive treatment exists. And although exercising more, eating healthier and staying connected to others have been linked to reduced risk of cognitive decline, there's little evidence that such interventions work by acting on amyloid and tau in the brain. It's more likely, Villain says, that these factors boost the brain's ability to stay sharp even when amyloid and tau are present.

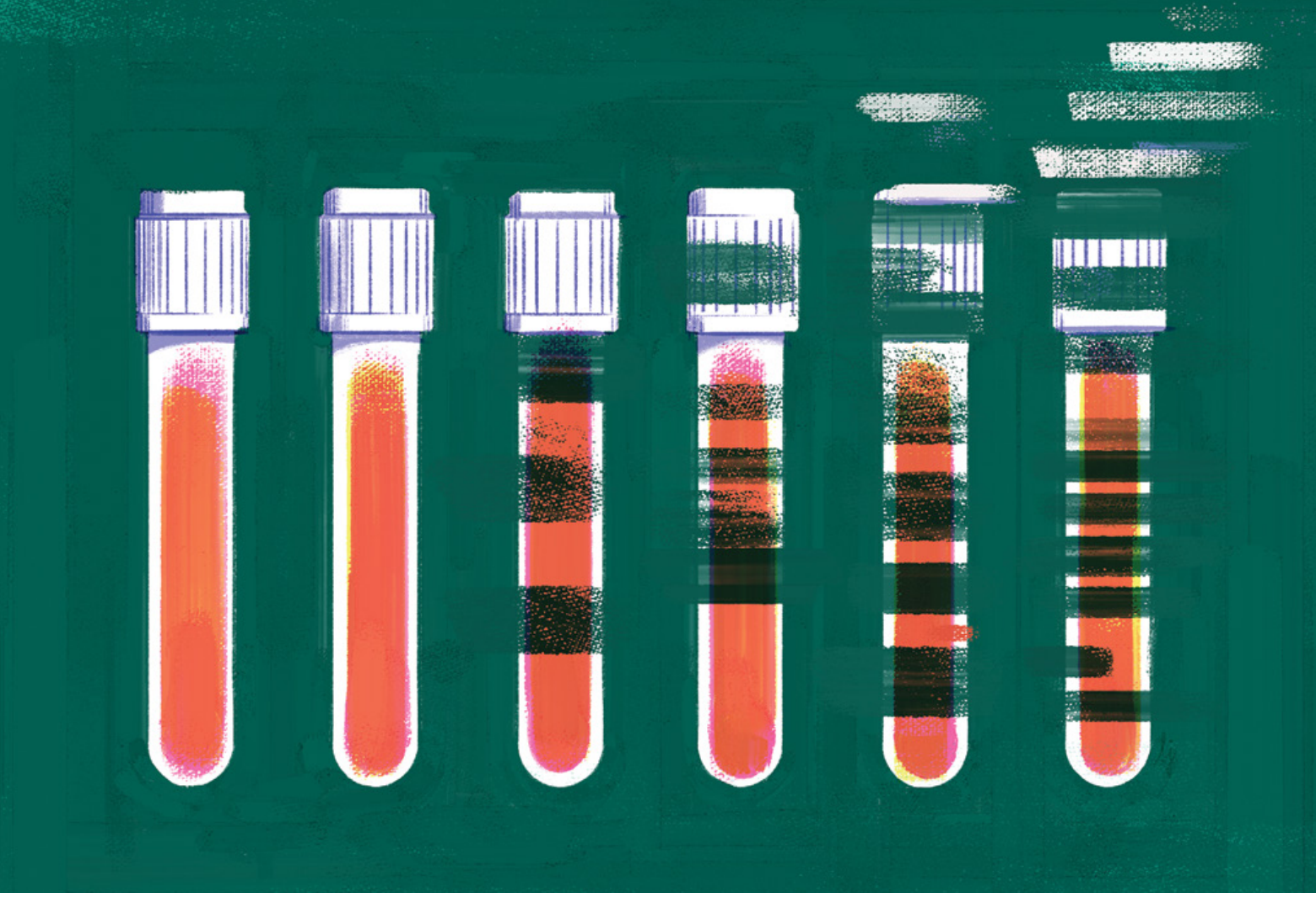
An international working group that included Villain and 45 other Alzheimer's experts from 17 countries developed a more nuanced assessment. The group split asymptomatic individuals with positive biomarker tests into two categories. Those with no symptoms and a low lifetime risk of developing them were classified as "asymptomatic at-risk" for the disease. Those with a genetic background that increased their risk or with imaging results suggesting their brain already had tau buildup were designated as having presymptomatic Alzheimer's. "It may seem to be only semantics," Villain says. "It's important semantics, however."

Even in symptomatic older adults, the tests aren't perfect. Measuring biomarkers in blood is trickier than doing so in spinal fluid. Because blood concentrations of amyloid and tau are far lower, the tests have to be more sensitive. And because there are so many other proteins in blood, the signal is harder to detect. What's more, p-tau can be elevated for reasons other than the presence of Alzheimer's. In patients with kidney disease, for example, p-tau levels might be high because the kidneys aren't able to properly clear the protein.

Some research also suggests that the tests might not be as accurate in certain racial groups. In one study, William Hu, director of the Center for Healthy Aging Research at Rutgers University, and his colleagues studied data from about 200 older Americans who had undergone detailed clinical evaluations and had given blood and spinal fluid samples. The researchers found that whereas a positive blood test was 87 percent accurate for white patients, it was only 58 percent accurate for Black patients. "So there's a high chance that they actually won't have Alzheimer's," Hu says. Other studies, however, failed to find racial disparities for blood biomarkers, and no one yet understands why such a discrepancy might exist.

Who Should Get Tested?

For those who are unsure about whether they or a family member should get a blood test, neurologists say the tests should be reserved for people experiencing memory loss or other cognitive problems. A blood screen can help differentiate Alzheimer's disease from other types of dementia or other health issues. If it comes back positive, a neurologist might still want to confirm the diagnosis with a lumbar puncture or a PET scan. "It's a serious diagnosis that I don't take lightly, so I want it to be as reliable as possible," says Zaldy Tan of the Cedars-Sinai Medical Center in Los Angeles.



For now, unless the patient has undergone cognitive testing and been diagnosed with cognitive impairment, most neurologists suggest forgoing the blood tests. "That's a conservative position, but that's what many think is appropriate right now," Chin says. There's good reason for the recommendation. Family doctors aren't always educated on all the caveats that go along with these blood tests, nor are they able to explain them in the little time they have with a patient. "I worry that this easy-to-click test is going to replace what clinicians are good at: talking to people and hearing their complaint," Chin says. If someone comes in because of memory problems and takes a blood test, the test could come back positive even if the person's lapses aren't the result of amyloid in the brain. Maybe "it's actually sleep apnea, depression or a medication side effect," he adds. "That elevated amyloid is just a red herring."

One of Chin's patients first visited his primary-care doctor with complaints of forgetfulness. The patient's lab work and magnetic resonance imaging came back normal, so, without performing any cognitive testing, the doctor did a blood test for Alzheimer's. The result was positive, and the patient assumed he had dementia. But additional testing at Chin's clinic revealed that the man had only very mild cognitive impairment. "He came to me thinking, 'I'm going to be talking about hospice and end-of-life stuff,'" Chin says. But he was probably years away from developing dementia.

In some cases, Hu says, multiple tests from different companies can also seed confusion. "One of my patients said, 'My tests tell me definitively I have Alzheimer's but also definitively I do not have it. So what does this all mean?'" he asks.

The Alzheimer's Association is working to develop clinical practice guidelines for blood-based biomarker tests. In July the group released the first two recommendations. "These are really focused on the specialty-care setting in patients who are living with cognitive impairment," says Rebecca Edelmayer, vice president of scientific engagement at the Alzheimer's Association. In those individuals, tests with 90 percent or greater accuracy can be used to diagnose the disease with no further confirmatory testing. That means patients wouldn't need to undergo a PET scan or lumbar puncture. Less accurate tests can be used to triage people for further testing as long as they have sensitivity of at least 90 percent for detecting disease and specificity of at least 75 percent for identifying people without disease.

Edelmayer says the group is still working on guidance for primary-care physicians and for people who are not cognitively impaired. "We really needed to evaluate all the evidence around individuals who are cognitively impaired first before moving into individuals who are cognitively unimpaired," she says.

Getting the diagnosis right is crucial, says neurologist Shauna Yuan of the University of Minnesota Medical School. "We have to remember that this is a terminal disease," she says. "It is not something we can take lightly."

The Care Abyss

As dementia cases rise, memory-care facilities are shuttering. A national collaboration wants to help solve the looming long-term-care challenge

By Tara Haelle

THE RATE OF ALZHEIMER'S diagnosis has declined steadily in recent decades, but as baby boomers age, the number of new cases continues to rise. The top risk factor for dementia is age, and by 2030 more than one in five Americans will be 65 or older. That means the prevalence of Alzheimer's in the U.S. could exceed 13.8 million people by 2060.

If current trends continue, many of them will have no place to go. Save Our Seniors, a collaboration of the American Health Care Association and the National Center for Assisted Living, estimates that more than 770 nursing homes have closed in the U.S. since 2020, and recent federal cuts to Medicare and Medicaid will almost certainly decrease access to long-term care. Older adults overwhelmingly prefer to age in place and receive care at home, but for that to be possible, there must be support for home caregivers, enough people willing to do those jobs, and coordination between local and state services.

A recently launched national resource funded by the National Institute on Aging, the State Alzheimer's Research Support Center (StARS), aims to help make all that a reality. By gathering data on the effectiveness, accessibility, and equity of state and regional programs for dementia care, then sharing those data, the researchers involved in the project hope to help states build partnerships that will aid policymakers at all levels in identifying the best solutions. Scientific American spoke with Regina Shih, an Emory University epidemiologist and co-principal investigator of StARS, about the problems our aging population is facing and how she and her colleagues are working to solve them.

The following interview has been edited for length and clarity.

As the U.S. population ages, how is the country meeting the needs of people with dementia?

Our long-term-care system is in a crisis. We have done a lot on the health-care side to improve the quality, delivery and accessibility of care. But when you think about assistance with activities of daily living at home—managing medications, transportation, toileting, bathing, getting dressed and making meals—that is the vast majority of dementia spending.

A recent study by a team at the University of Southern California determined that the national cost of dementia is $781 billion a year. Much of that is long-term care and unpaid caregiving provided primarily by family members. And it's lost earnings because family caregivers have to reduce their work hours or leave the workforce altogether.

What are the biggest challenges in dementia care?

The first is convincing those concerned about cognitive changes to seek dementia screening. Many don't believe it's worth getting a diagnosis, because they feel there's nothing to be done. But you can do lots of things in the early stages of dementia to prevent serious progression [see "Cultivating Resilience" on page S3].

There are also challenges in paying for care and in determining who provides it. How do you support family caregivers who are helping someone age in place, and what kinds of providers can help people manage medications and aid with transitions? If there is a hospitalization, how can you prevent long stays, dying in the hospital, or moving someone into a nursing home when they want to age at home? And at the end, it's about palliative care and a dignified death.

How does StARS aim to solve these challenges?

Our goal is to help states deliver dementia-care programs. We don't need to reinvent the wheel and create more integrated and coordinated dementia-care programs. There are wonderful models of care we don't get to hear about because they're in one institution or within one state. We want to study those models and learn how to increase access to those kinds of dementia-care programs, as well as how to pay for them and meet the needs of different caregivers. That's what StARS is about—helping state leaders and health-care providers increase the accessibility and affordability of dementia-care programs within their state.

Say your mother has dementia, or you suspect she needs a diagnosis because she's forgetting how to drive home or can't remember names of family members. You could talk to your primary-care doctor, but that's not the only avenue. States are creating innovative programs to help with dementia diagnoses.

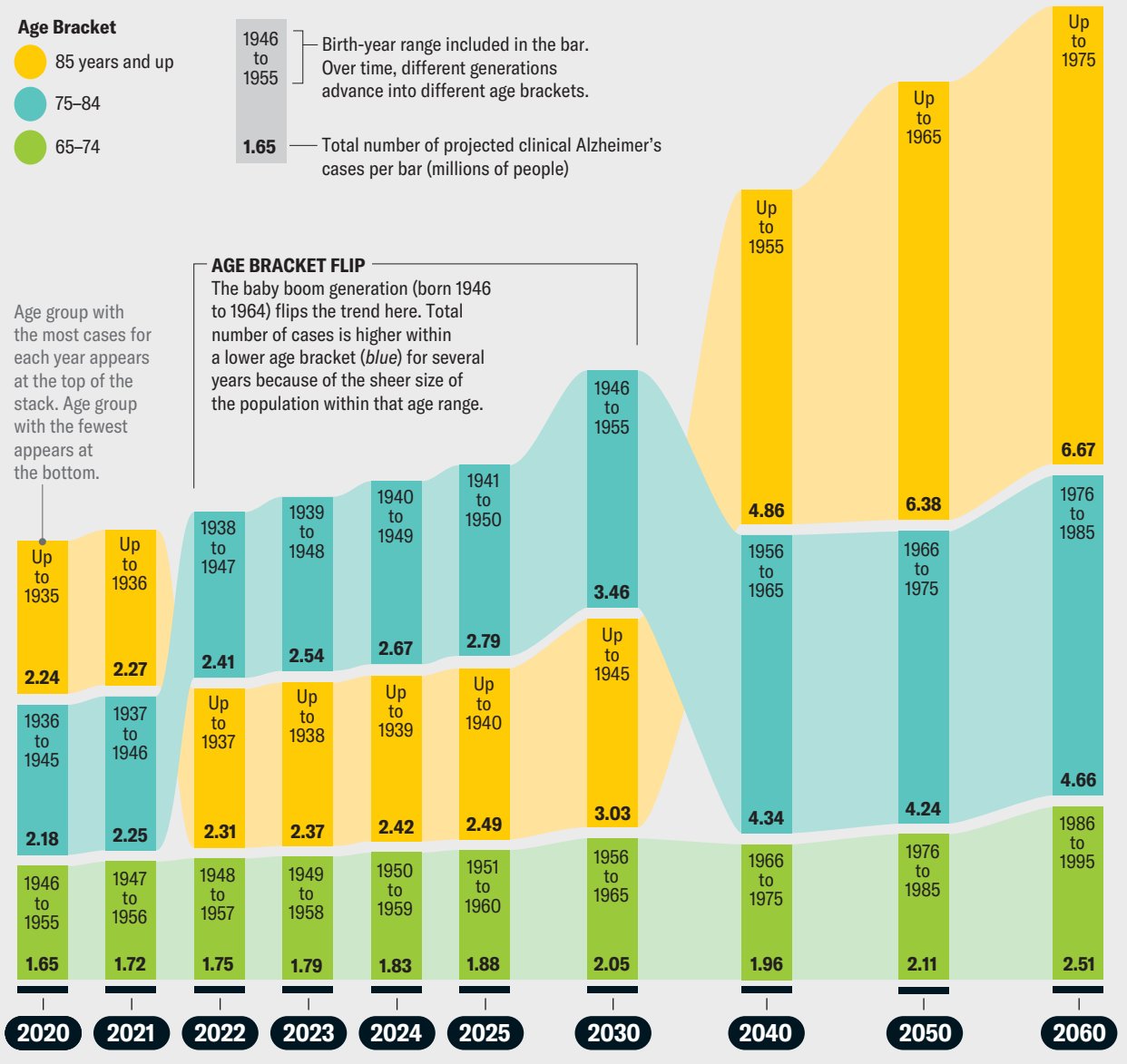
The next step is to help states deliver referral services. How does someone with dementia live in their home independently? How do they start preserving their memory? How do they drive independently when they don't remember the way home? If they can't prepare food on their own, how do they get meals? How do we get them physical therapy if they're starting to fall?

We want to help states learn from one another, to say, "State X, do you see how State Y is doing this? It is funding family-caregiver support programs in this way, or it funds meal deliveries in this way, preventing hospitalizations or emergency room visits."

One example is GUIDE, a coordinated care model being tested by the U.S. Centers for Medicare and Medicaid Services (CMS). The program assigns a "care navigator" to people with dementia and their caregivers, someone to help them access everything from clinical care to transportation. The goal is to enhance quality of life for people with dementia, to help them stay out of the hospital, receive better care and reduce caregiver burden. The GUIDE sites at the Univer-

Projected Rise in Alzheimer's Cases

Overall clinical Alzheimer's disease cases are projected to rise in the U.S. as the population grows older, according to a study published in 2021. Age is the greatest risk factor for the disease, and the nation's largest group—the "baby boomer" generation—is entering the riskiest age range. Given the resources currently devoted to dementia care in the U.S., access to that care will become increasingly strained unless states prioritize investment in support services.



Source: "Population Estimate of People with Clinical Alzheimer's Disease and Mild Cognitive Impairment in the United States (2020–2060)," by Kumar B. Rajan et al., in *Alzheimer's & Dementia*, Vol. 17; December 2021 (*data*)

sity of California, Los Angeles, and Emory University are among those being tested. There are potentially other innovative models of coordinating dementia care developed within states, and so we want to know whether aspects of those programs work for other states. Are the models serving all communities, rural and urban?

As caregiving needs grow, large numbers of nursing homes are closing. Why?

Costs for nursing home care are high, so CMS is helping states increase access to home- and community-based services, including bathing, physical therapy and end-of-life care. This CMS push has moved care away from nursing homes and means a lot of them have closed their doors. It's really a crisis to think about where these individuals are going and how much reliance there will be on family caregivers. I have had nursing homes reach out to me and ask, "Who is going to take the residents from our nursing home when we have to close?" The burden of care is going to shift to the public. If some-

Graphic by Jen Christiansen

one needs personal care or home care or someone to cook them meals, they have to be either wealthy enough to pay out of pocket or poor enough to be eligible for Medicaid. People who aren't eligible for Medicaid home- and community-based services often rely on family caregivers.

There are national support programs that can help people learn how to be family caregivers and navigate the care system. For example, I am a volunteer with the Area Agency on Aging (AAA) in Atlanta. Someone can sign up to come to a library or recreation center, and I'll train them to cope with their stress, to help prevent falls and to navigate behavioral symptoms that come with dementia.

How do family caregivers find out about these programs?

They can go to their local AAA. The name may vary by location; here in Georgia we have Georgia Memory Net. There are clinics across the state where anyone can walk in and say, "Can you help me determine whether my mom has dementia?" Once someone has a diagnosis, Georgia Memory Net provides referrals for services: food access, meal preparation, personal care, home care, physical therapy—all the things they need to stay in their home.

Georgia Memory Net is doing amazing work across the state to help both people with dementia and their caregivers, so other states want to replicate what it's doing. But do we know whether it results in better outcomes? Does it reduce the burden on caregivers or increase their quality of life? Does it reduce hospital admissions, improve affordability or help family caregivers stay in the workforce? We don't know, because there's no data infrastructure to track this information. That's what StARS is working to build.

One of StARS's goals is to establish partnerships between new programs and existing successful ones. What would that look like?

States fund things in very different ways, so that's one way they can learn from one another. There are states saying, "I would like to do something like Georgia Memory Net. How do I do that?" They would look to Georgia, and Georgia would provide them with support. Meanwhile Georgia could look at another state nearby, maybe Tennessee, to see how it has integrated its AAAs and its health-care system or to find out whether a certain type of service referral helps to decrease hospital visits and save money.

What kinds of dementia-care pilot projects are you looking to fund?

We have the health-care system—hospitals and clinics—and we have social service systems like what the AAAs provide, such as meal delivery or bus passes to go to the doctor. Those two systems don't talk to each other. StARS wants to help states link up their data systems so that an AAA can say, "When I refer people to Meals on Wheels, I think I'm helping them avoid homelessness and hospitalizations, but I'm not sure. When I give them services like this, does that avoid hospitalizations?" The data systems could then be linked together to show that, say, this person ended up in the hospital 60 or 90 days after she received a referral service, or in a year this is the number of hospital visits she had.

We also want to show that linking existing data across different settings of care could help states save money and share best practices with other states. Right now many AAAs have a wait list because the services are in such high demand. There are so many older adults with dementia that some states can't fully meet the demand for services right now. If StARS could show that referring and coordinating care helps save money by avoiding hospitalizations, for example, maybe states could make the case for more funding for those services. In many AAAs, we have education programs for family caregivers. If we could serve more of them, perhaps they could actually save money by avoiding downstream health-care costs. For example, if caregivers have information on how to reduce falls, the person they're caring for will be less likely to end up in the hospital. Or the AAA can send services to someone's home to install grab bars and to secure rugs. Those are things some caregivers can't afford or don't know they need.

What would be the ideal societal setup for the growing population of people living with dementia?

I don't think there is one particular set of services that could meet the needs of every single kind of person living with dementia. I would love to see integrated and coordinated dementia-care programs tailored to meet the needs of all kinds of people with dementia who have different family situations and different levels of access to care. I think there is a lot of promise in saying to states, "We are here to help you figure out what works for specific populations in your state within the ecosystem of care you've already built."

How can StARS help improve dementia care?

One way is to convince state policymakers to increase funding for family-caregiver supports, meal deliveries, home modifications to prevent falls, and other services. Another is to make sure states are aware of innovative ways to deliver those programs. Entities that deliver those services may not have the capacity to share what they do, so StARS wants to centralize those resources. And we could help answer questions such as, "I anticipate an increase in people with dementia in my state. Do I have enough geriatricians and direct-care workers?" Each state needs a financial case to build that pipeline of workers, to create programs and incentives for people to enter those programs.

We want to give states tools that help people with dementia and their caregivers across the full spectrum of care, from diagnosis to everything that follows, and ensure a high quality of life. That includes things such as knowing how to help people with dementia evacuate in a weather-related disaster. It's ensuring a dignified death and helping the family caregiver with bereavement. We have to help states deliver coordinated programs so that at every stage, no matter where you're at, the quality of life is the best we can hope for.

Tara Haelle is a science and health journalist based in Dallas. She is author of *Vaccination Investigation* (Twenty-First Century Books, 2018) and co-author of *The Informed Parent* (Tarcher, 2016).

Clifford Harper, seen here holding a photo of himself as a professor, was told by his physician that his cognitive decline might have begun 15 or more years before his memory loss became evident. The delay may be attributable to his education and physical fitness.

Prevention Intervention

The evidence is clear that racial discrimination, physical health and the environment contribute to Alzheimer's and other dementias. Now researchers are looking for ways to intervene **By Jyoti Madhusoodanan**

ABOUT FOUR YEARS AGO Clifford Harper, then 85, announced to his wife that he was quitting alcohol. Harper wasn't a heavy drinker but enjoyed a good Japanese whiskey. It was the first of a series of changes Linda Kostalik saw in her husband. After he'd cleared out the liquor cabinet, Harper, a prolific academic who has authored several books, announced he was tired of writing. Next the once daily runner quit going to the gym. Kostalik noticed he also was growing more forgetful.

The behaviors were unusual enough that, at an annual physical, the couple's physician recommended they consult a neurologist. A battery of medical tests and brain scans revealed that Harper's surprising actions and memory loss were the result of dementia.

Harper's neurologist at Oregon Health Sciences University (OHSU) asked whether he might like to enroll in a long-running study of dementia in African Americans. The study's focus on Black health piqued Harper's interest, and he decided to participate for as long as he could. "I hope it will help other men like me," Harper says.

As a Black American, Harper faces a risk of Alzheimer's disease and other dementias that is twice that of white Americans his age. The reasons for this disparity are still unclear, but researchers know Black Americans are particularly vulnerable to a number of confirmed risk fac-

Photographs by Gioncarlo Valentine

Harper spent years in the U.S. Coast Guard, where he experienced racism and recognized the protection his tight-knit community had offered him throughout his childhood.

tors, such as living in areas with higher rates of air pollution and encountering difficulties accessing healthy foods and high-quality education. Some studies suggest that experiencing racism and other forms of discrimination contributes to a higher risk of cognitive decline. Race or gender discrimination also raises a person's risk of heart disease and, as a result, some forms of dementia.

That's part of what prompted Harper to participate in OHSU's study, called the African American Dementia and Aging Project (AADAPt), which was established in part to capture the unique history and experiences of Black communities in Oregon. The state's first constitution banned nonwhite citizens from settling there. The ban was overturned by the early 1900s, and shipyard work during World War II brought an influx of Black workers to the region, but they still faced discrimination and racism in many forms. By the end of the war, racist lending practices—called redlining—led most of the Black community to live in segregated neighborhoods or those that were poor in resources needed for good health, such as parks and grocery stores.

Discrimination in the scientific world, along with other factors such as distrust of researchers, led to underrepresentation of Black communities in brain research. Even today clinical trials for new treatments of Alzheimer's include very few people of color. As a result, researchers and doctors are ill-equipped to understand the causes of dementia in these communities. "Not only are there health disparities around rates of Alzheimer's, but we've understudied the Black population in relation to the causes," says Andrea Rosso, an epidemiologist at the University of Pittsburgh.

Now that Alzheimer's and some other dementias can be diagnosed early and their progress potentially slowed, figuring out who's most vulnerable is even more critical. Diagnostic tests and interventions aren't yet reaching all those who need them. Researchers should include historically minoritized communities in studies of these new frontiers in dementia diagnosis and treatment, says epidemiologist Beth Shaaban of the University of Pittsburgh. If adequate attention isn't paid to diverse populations, communities that already experience disproportionate rates of dementia will be uninformed about their increased risk, how to lower it and how to access diagnoses and care. "We are very concerned that these disparities and the rapid evolution of the new technology could leave people behind," Shaaban says.

AADAPt and other studies aim to correct this inequity. The project seeks to understand the forces driving cognitive decline in Black Americans, identify protective factors that lead to healthy aging, and find practical solutions. The team hopes to eventually use the data to build predictive models that will catch cognitive decline early and potentially help

Harper had to fight for the right to earn his Ph.D. in English. He went on to become a playwright, author, theater producer and professor who wrote several books.

people such as Harper access new medicines and treatments via clinical trials.

At the turn of the century researchers projected that an aging baby boomer generation would drastically increase the incidence of Alzheimer's and other forms of dementia. No treatments or protective strategies were known at the time, and the search for solutions focused largely on the tangles of proteins that jammed up brain circuits.

In the past two decades, scientists have discovered that certain drivers of Alzheimer's may be controllable. In 2011 dementia researcher Deborah Barnes of the University of California, San Francisco, and her colleagues reported that poor education and smoking—things that could be addressed by behavioral changes and social reform—were among the greatest threats to aging brains. In a 2022 follow-up study, Barnes reported other modifiable risk factors for Alzheimer's, such as midlife obesity and sedentary lifestyle, which can raise a person's risk for heart disease.

"People had been so focused on genetics and medications. No one had really been thinking about the potential for prevention," Barnes says. "It was surprising to a lot of people to realize that these modifiable risk factors really could play a big role."

Decreased risk can come in many forms. Education is critical, as it nudges the brain to build more—and more resilient—connections between neurons and different parts of the brain. This so-called cognitive reserve can act as a buffer against degeneration as we age, Barnes says, and can preserve brain function even as plaques and protein tangles start to cause disease. Studies suggest that social engagement can also help build this cognitive reserve.

Heart health is crucial, too. High blood pressure, high cholesterol, and other kinds of heart disease can hinder blood circulation and starve the brain, which is a voracious consumer of oxygen and glucose. Although these problems don't themselves change protein buildup in the brain, they "kind of exacerbate what's happening there," Barnes says. "It's like a double whammy."

Over the years researchers have found many other ways to reduce dementia risk. Improving air quality is a big one. Although the mechanisms are unclear, studies in animals suggest that the ultrafine particles in polluted air infiltrate lung cells to eventually reach blood vessels in the brain or directly affect the brain's cortex, where Alzheimer's starts.

In addition to these modifiable threats, certain genetic variants are also linked to a higher risk of developing dementia. Partly because of this range of causes, "dementia" is a broad umbrella term, and how these varied threats converge to cause disease will dictate the form of dementia someone experiences. Alzheimer's is the most common form, and vascular dementia is a close second. Other conditions, such as Lewy body dementia and frontotemporal dementia, cause similar cognitive symptoms.

Addressing modifiable health risks,

Linda Kostalik (*right*) says that this experience has shown her aspects of Harper's personality she had not previously encountered. "One of the things that I've discovered is I'm probably married to the sweetest man in the world, and so it's not as scary," Kostalik says.

such as by improving education or encouraging heart-healthy behaviors, has slowed the rising toll of dementia, Barnes says. But not all communities have benefited equally.

Education quality, pollutant exposure and access to healthy foods are tied closely to where people live. "There's a number of ways our neighborhoods impact our cardiovascular and brain health," Rosso says. Historically, Black and Hispanic neighborhoods have been more likely to lack grocery stores. They also had fewer health-care facilities, and their schools had fewer educational resources available to students. Unsafe neighborhoods made it difficult for people to take walks or exercise safely outdoors. Highways and factories—major sources of air pollution—were often constructed in these already disadvantaged areas. And the residents were stuck where they were—discriminatory lending practices prevented them from moving to better-resourced locales.

Harper grew up in a historically redlined area of East St. Louis, Ill., and his health prospects were not initially promising. Yet it was a close community. Harper's brother-in-law encouraged him to stay in school. So did Charlie, the owner of a dry-cleaning business on the corner where Harper and his friends hung out. Charlie made the boys a promise: "If you go to college, I'll clean your clothes," Harper recalls. "He was shocked because most of us did."

The dry cleaner kept his word. "Charlie didn't realize that part of our success was because of him," Harper says.

Although Harper's career choices nourished his brain, leaving his childhood neighborhood exposed him to more discrimination. During his service in the U.S. Coast Guard, one of his superiors addressed him in a mocking drawl, insinuating that Black people were "slow and dumb," Kostalik says. Throughout graduate school Harper had to advocate repeatedly to pursue his English degree. "Back in those days, folks like me didn't find a welcome mat," he says.

That racism persisted throughout much of Harper's life. Kostalik says that when Harper was a professor at the University of Illinois, he would visit a nearby federal penitentiary to confer college degrees on inmates who had earned them. On one such occasion, she says, the father of one of the degree recipients approached Harper. "I don't care who you are or what you're wearing," he said. "You're still a [N-word]." Today Harper doesn't recall the interaction and doesn't mind forgetting it. "That's something I wouldn't want to remember," he says.

Studies show that a lifetime of such experiences takes a toll on heart and brain health. Last year researchers analyzed data gathered from nearly 900 families over a 17-year period to understand how discrimination can affect Alzheimer's risk. Based on interview records and blood samples from 255

Black Americans, they found that those who reported experiencing racism in their 40s and early 50s had higher levels of two blood proteins that serve as biomarkers of dementia.

Researchers are also learning how social interactions can cause biological change. In research presented earlier this year, Shaaban and her colleagues analyzed how blood vessel damage, connections between brain regions, and Alzheimer's biomarkers such as amyloid and tau proteins varied by race and sex. They found that white men had better connections across brain regions than Black men and both Black and white women in the U.S. White men also tended to have higher levels of amyloid accumulation, whereas the other groups tended to have more signs of vascular disease. "White men are the outliers," Shaaban says. "We think this has implications for how people think about what these biomarkers mean in different groups of people."

The results underscore the need for studies that are more representative of the populations that experience dementia, particularly because discrimination is not a risk factor that an individual can control. "You can tell people to exercise more," Rosso says, "but you can't tell them not to be discriminated against."

Harper has been diagnosed with vascular dementia, a form of dementia that is more common in Black men. In addition to memory loss, he started to struggle with balance recently, and he now uses a cane to walk. Harper says years of experiencing racism probably played a part in his diagnosis and symptoms. He had always made the effort to exercise and eat healthily, but he had little control over the discrimination he fought his entire life. "I am the result of being a Black man in this country," he says. "I have the highest degree you can get. But I'm a Black man."

The toll of discrimination has been difficult to quantify, in part because those who experience it are often overlooked by scientific research. As a result, understanding how different risk factors contribute to dementia in Black communities is challenging, Rosso says.

Data from AADAPt and other studies offer some clues. In a study published in May, researchers at the University of Wisconsin–Madison analyzed the links between adverse social experiences and vascular injuries in brain tissue.

The team studied 740 brain samples donated to Alzheimer's research centers. Regardless of race, the brains of people who had lived in disadvantaged neighborhoods or experienced other discrimination over their lifetime were more likely to bear signs of vascular damage, ranging from blocked vessels to hemorrhages.

Gathering such data can help clinicians improve how they measure Alzheimer's symptoms and track the disease's progress. Biomarkers do not differ by racial group, Shaaban says. But dementia can develop in different ways, which means two people with the same diagnosis could have different processes at work in their brains: whereas one may have a buildup of amyloid protein, another may experience more symptoms caused by blood vessel disorders. Studying diverse groups will help scientists understand how these biological mechanisms bring about different forms of cognitive decline, Shaaban says. It will also help them identify the best ways to treat, and prevent, Alzheimer's and related dementias.

At OHSU, the AADAPt investigators track the physical and mental health of participants at annual exams. If they spot signs of cognitive decline, they follow up to offer guidance or a referral to a specialist. They also conduct interviews with participants to understand how social experiences have shaped their health.

In a 2024 study, the AADAPt team reported that nearly three quarters of the subjects self-rated their health as good or excellent. Yet more than 80 percent had high blood pressure, 33 percent had diabetes, and more than 25 percent had a history of stroke. About two thirds of the participants rated their memory as good or excellent. The contrast between their strong sense of optimism and their medical history indicates a mindset that may be "a little bit protective" of brain health as they age, says gerontology researcher Allison Lindauer of OHSU, lead investigator on the study.

Capturing these nuances could help reduce dementia risk in innovative ways. "Identifying protective factors that are salient to these communities is important," Rosso says. "We don't want to write off the whole community and be like, well, you don't all have Ph.D.s, sorry."

In addition to working on the AADAPt study, OHSU neurologist Raina Croff began to explore whether neighborhood connections could guard against cognitive decline. She was born in the historically redlined Albina district of Portland and remembers it as tight-knit—much like where Harper grew up. "When your community is confined to a certain area, you're highly dependent on one another, and you can create quite strong social ties," she says. "You grow strong from within."

Croff and her colleagues designed several mile-long walks through the Albina district in an effort to encourage exercise and help build social connections. Each trail was marked with signposts sharing news clips, old advertisements and political campaign buttons. Participants in the study, known as SHARP (for "Sharing History through Active Reminiscence and Photo-Imagery"), walked in groups, discussing the signs and reminiscing as they exercised. The result was improved cognitive function in people with mild memory loss, Croff says.

Such projects can help solve many of the inequities created by systemic racism. They also provide a more complete portrait of brain health in minoritized communities: structural racism and a lack of resources can drain people's cognitive reserves, yet their social connections may act as a potent buffer.

That complete picture is precisely what the AADAPt researchers hope to glean about the brain health of aging Black Americans, Croff says. "Despite the many barriers, we can still feel empowered to change our health. I think that's important to anybody."

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Sweater holes can be fixed either by discreet darning or by creative "visible mending," popularized by designer Flora Collingwood-Norris.

Fast Fashion Needs a Green Makeover

A more circular economy in textiles will look good on everyone BY THE EDITORS

PEOPLE IN THE U.S. throw away at least 17 million tons of textiles every year—about 100 pounds of clothing per person. At the same time, unsold blouses, jackets, and other fashion-industry leftovers end up in dumps such as the one in Chile's Atacama Desert, so vast as to be visible from space. Many of these items are fast fashion—made quickly, sold cheaply, and in style for too short a time because the industry relies on novelty to keep consumers buying.

Fashion poses more than an aesthetic problem, however. Every year the global garment industry emits up to 10 percent of the world's greenhouse gas output and uses enough water to fill at least 37 million Olympic-size swimming pools, as an article in this magazine noted this past July. Cotton farming can involve massive quantities of pesticides, and yarn dyeing pollutes waterways with toxic chemicals. Synthetic polymers such as nylon are made with fossil fuels and shed microfibers with every wash.

It's time to embrace a circular economy in fashion—one that reuses clothes, fabrics and yarn; recycles to the extent possible; and encourages producers and retailers to choose textiles and processes that minimize the input of raw resources such as cotton or synthetic polymers. Our choices as consumers matter as well. How we select fashion and follow trends is one accessible way we can make a dent in climate change.

"We know the industry is overconsuming [resources] and overproducing in general," says Laila Petrie, director general of Future Earth Lab, a nonprofit sustainability organization. "Volumes have continued to increase, and that can't continue forever." Almost one third of the clothes produced every season are never sold and may go straight to landfills.

As awareness increases, many people are donating to or buying from thrift shops or, when they shop new, looking for "certified organic" labels. And many companies are trying to figure out how to remain profitable while producing less and ensuring that what they do make does less harm to people and the planet. Consumers and companies alone can't solve such a vast ecological and climate problem, however. The industry needs to be held responsible for scrutinizing entire supply chains and making modifications to reduce harm, Petrie says.

Last year California enacted an extended producer responsibility (EPR) law for textiles, which requires brands with more than $1 million in global sales to pay for reuse, repair or recycling of their products. Producers will begin collecting used clothes in 2030, but where those garments will end up is still unclear. "We're watching closely," says Rachel Van Metre Kibbe, founder and CEO of advisory firm Circular Services Group. "It will be interesting to see whether brands can lead their own transition." New York State and Washington State are currently considering similar bills.

EPR alone isn't enough, however. What's needed is "a fundamental shift in how we consume, make and sell products," Van Metre Kibbe says. What she has in mind is a circular textile economy, which begins with designing products with their entire life cycle in mind.

For instance, a shirt may need to be made with only one type of yarn or with an easily recyclable blend and labeled with its constituent fibers so it can be readily sorted, making it easier to recycle. Advanced recycling technologies, such as using enzymes to separate polycotton blends into cotton and polymer fiber, are emerging, but they are still expensive and are only now starting to be scaled up. Supporting the development of these technologies would help generate the kind of innovation economy many people claim the U.S. needs.

The Americas Act, a bipartisan federal bill proposed in March 2024, seeks to provide incentives for textile reuse and recycling. If enacted, it would provide a huge impetus toward establishing a circular textile industry in the U.S. As one of the largest consumers of textiles, the U.S. has the potential to also become one of the largest recycling economies in the world. "There's a real opportunity here—we just have to capture it," Van Metre Kibbe says.

An initiative called Fibershed shows how such a system might work. It started

Flora Collingwood-Norris

in California in 2011, connecting regional farmers, designers and producers in a sustainable clothes-making economy. The concept has since spread to 79 communities around the world.

Still, a significant portion of our clothing will continue to be made abroad, in places where farmers and factory workers toil in precarious conditions to grow cotton or sew apparel. Roughly 100 million people, especially women in the Global South, stitch garments, and only a tiny fraction of them are paid a living wage. Companies that source from developing countries need to devise strategies alongside their suppliers—collaborating with garment manufacturers and with farmers' groups—to improve conditions, Petrie suggests. Such a process can drive change in ways that are inclusive and therefore likely to be more effective.

As consumers, we can buy less, be more discerning in what we do acquire, buy or exchange used clothes, wear each garment longer, and find new uses for old pieces. Such practices were the norm decades ago, and some are returning.

In Germany, parents often buy kids' clothes from children's flea markets—particularly helpful because kids outgrow their clothes so fast. In India, old saris are overlaid and stitched together into a light quilt, a practice that has evolved into an art form. Moth holes in a beloved cardigan can be fixed either by discreet traditional darning or by the craft of "visible mending." And in the U.S., people routinely shop consignment, thrift and online marketplaces for used clothes in good condition, keeping those items out of landfills for a while longer.

Meanwhile we must remember that consumers are an influential voting bloc. We can prod regulators and brands to take action, and we can exercise our values by deciding which brands to support. What we wear every day is something over which we can and should exert a great deal of power. Deserts should not be full of unwanted T-shirts. Our waterways should not be full of fashion-related microplastics. ●

Science Makes the U.S. a Great Nation

History tells us what happens when great nations attack science BY PAUL M. SUTTER

ONE OF HISTORY'S dark jokes is that the Roman Empire, for all its vaunted accomplishments, made only a single great "contribution" to science: the killing of Archimedes. Today the U.S. risks suffering the same kind of shame.

In 212 B.C.E. the Romans sacked the Greek city of Syracuse after a prolonged siege, and a Roman soldier killed Archimedes, then the greatest living mathematician, physicist and engineer—and one of the greatest minds of all time. Exact accounts vary, but according to one, Archimedes was engrossed in sketching a problem in sand when his murderer arrived, sword drawn. Covering his work, the mathematician said, "I beg of you, do not disturb this." In response, the soldier struck down the 72-year-old man.

American science now faces its own sharpened edge. The Trump administration stands with its sword drawn. It's choking our universities. It's stamping out the free flow of ideas. It's cutting funding to basic science. It's ready to make the killing blow, all in the name of making America great again.

Despite declines in its favorability since the COVID pandemic, science remains one of the most trusted and best-regarded institutions in the U.S. And although modern science has many flaws, it is one of those few things we can point to as a society and say this, *this,* is what already makes us great.

Our technological and scientific prowess is the envy of the world, unmatched across the globe and indeed throughout human history. No other country, no other culture, no other civilization has matched what the U.S. has poured into fundamental research in the years since World War II.

Last year the U.S. government put about $90 billion toward funding of nondefense research. And for the relatively paltry sum of close to $100 billion—essentially a rounding error in total federal outlays—repeated year after year for decades, we have miracles made manifest: cures and treatments, consisting of a few milliliters of molecules, to balm the worst of our diseases; machines that breathe fire to take us to the stars; devices, held in our hands, that connect us to friends, family and strangers on the other side of the world. All those marvels, great and small, can trace their roots to publicly supported research.

It's easy enough to point to the monetary benefits of scientific research—and the immediate harms that will be done if the current administration's proposed cuts go through. One dollar of National Institutes of Health research funding produces $2.56 in economic activity. Cutting annual research funding in half would save the average American taxpayer $260 this year—and cost them $10,000 in future wealth. Federal funding of nondefense research has accounted for about 20 percent of our nation's business-productivity growth since World War II.

In addition, although the majority of trainees in science do not end up as career researchers, they go on to add value to a wide variety of organizations, including businesses and government agencies. Science takes our best and brightest and throws them into the crucible, pitting them against the toughest problems known to humanity, and then sets them loose to solve the everyday challenges of our modern economy.

Paul M. Sutter is a cosmologist at Johns Hopkins University, as well as an author, television host and U.S. cultural ambassador.

An engraving of the death of Archimedes

The true greatness of science, however, and of the society that supports it, is measured not in dollars but in the intangible—what we have learned and what we hope to accomplish. We have built telescopes to peer back through deep cosmic time and see the dim, faded light of the first galaxies that emerged in the heavens. We have developed electronic machines to mimic our own intelligent speech and, by doing so, allow us to wrestle with the nature of our humanity. We have set ourselves to a great mission of conquest—not of a people or a rival nation but of the scourge of cancer. We have had the courage to examine our history, our communities, our social connections to ask uncomfortable questions and reveal painful truths.

Is this not what great nations do? They don't just build bridges and roads and monuments of stone and steel. They erect edifices of the intellect. They place their stamp on history. They create gifts to be enjoyed by generations to come. They are beacons that future civilizations can emulate.

Americans have long held themselves as different than people in other nations. French historian Alexis de Tocqueville, an astute observer of early American life, wrote in his book *Democracy in America* that "the position of the Americans is therefore quite exceptional, and it may be believed that no democratic people will ever be placed in a similar one." Our modern institution of science is one of our country's truly exceptional achievements.

That is why fundamental science is worthy of public funding. No private enterprise would ever dare sacrifice profits to study the arcane corners of the universe. No single patron, no matter how wealthy, can provide the funding necessary to slake our thirst for answers. Only nations—great nations—can afford to take a slim measure of the public's treasury and devote it to science.

Science is part of what makes us noble. It demonstrates our abilities to the world and to history. It is a projection of our strength. Look at us, we say to the world, so wealthy and wise that we set our sights further, our minds deeper. It's here, in this nation, that we will produce works that will stand the test of time.

The minuscule savings achieved from the proposed cuts to science research won't be felt in the average taxpayer's wallet. But the cuts will hurt us. They will hurt us now and for decades into the future. That is the bitter reality that we are now facing: we are deliberately making our children impoverished—materially and intellectually—in the name of insignificant savings today.

The proposed budget cuts would kill all of this greatness—the learning, the advancement, the courage, the powerhouse of American ingenuity, and one of the pillars that we can stand on to rightly claim our place in history as a truly great nation.

How will our descendants remember us and this moment? Will they view us as a people that dared mighty things—or as so much blood in the sand? Go ahead, strike down science if you will. But remember this: The name of Archimedes echoes through the centuries. The name of the soldier who killed him does not.

I beg of you, do not disturb this. ●

mikroman6/Getty Images

A Little Light Launch

By Aimee Lucido

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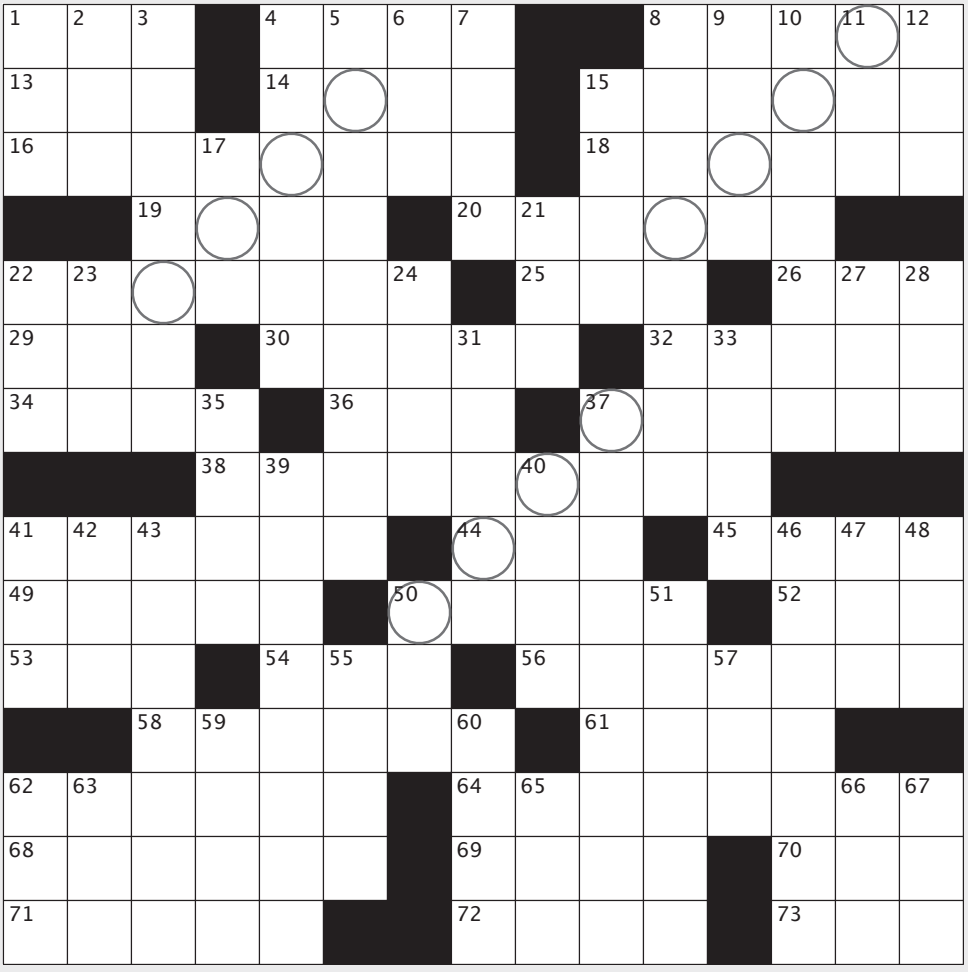
58 Weightlifter's injury
61 Is selfish with
62 Genre for Dwayne Johnson
64 "Breakthrough" mission that didn't happen in real life but does happen in another sense three times in this grid (*page 24*)
68 Himalayan highland inhabitant
69 Mario ___ (racing game)
70 Prefix with gram, graph or dermis
71 Big name in boxers
72 Takes to court
73 Have in view

Down

1 Indian lentil dish
2 Poke bowl fish
3 Just manages
4 Bellyached
5 City folk
6 Lass
7 Perform a low-friction activity
8 Gets to commit
9 Art Deco designer
10 At the start
11 Nutrition fig.
12 Oct. preceder
15 China-based online marketplace
17 "I could go on" (abbr.)
21 Early bird hrs.
22 Andean tuber
23 Word before a counterargument
24 ___ Grey tea
27 Boost
28 E.g., smooch on the subway
31 Southpaw
33 Large liquid holders
35 Some lit. degrees
37 Gig economy transport
39 Dilating doses
40 Electronic dance music DJ Steve
41 Animal that can add gestures to its lexicon
42 Possible subject of a decomposition experiment (*page 52*)
43 Take a load off
46 Some stuffed animals
47 Early web portal
48 "Don't worry abt it"
50 Acronym for the Reagan-era "Star Wars" program
51 Pixar movie starters
55 British psychoanalyst Freud
57 Respected pioneers, in hip-hop slang
59 The Emerald Isle
60 Takes the first step before starting a scientific experiment
62 Vesuvius fallout
63 Tea, in Mandarin
65 Phosphorylated protein that can show up in the brains of people with Alzheimer's (*page S13*)
66 Midwestern slang word expressing surprise
67 Formal wear that demonstrates a topological object (*page 22*)



Smoldering Alzheimer's disease

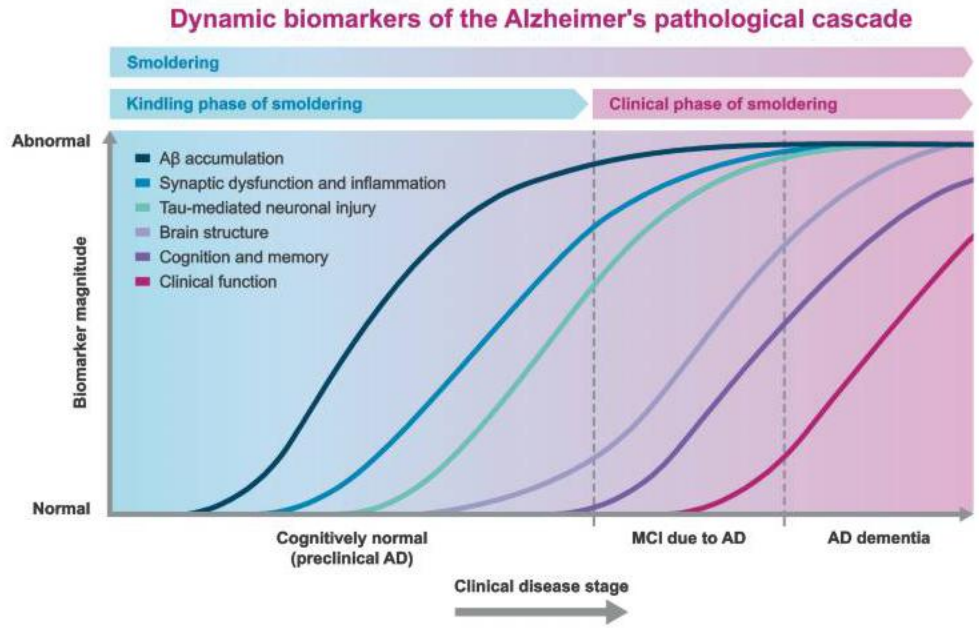


Figure 1. Smoldering across the Alzheimer's disease continuum. During the initial "kindling" phase of smoldering AD, there is a clinically silent buildup of pathological abnormalities decades before symptoms appear. The disease then progresses into a "clinical" phase, whereby ongoing smoldering processes continue to drive brain injury and clinical deterioration from mild cognitive impairment through to dementia of increasing severity. Aβ, amyloid beta; AD, Alzheimer's disease; MCI, mild cognitive impairment. Figure adapted from ref. 6 with permission.

"Smoldering" describes a group of processes that drive chronic disease progression, and introduces a fresh perspective to the complex interplay of Alzheimer's disease pathology.

Alzheimer's disease (AD) can be devastating for patients and their families and is a significant public health concern for society. Eisai's commitment to Alzheimer's drug discovery spans over four decades, culminating in the approval of multiple interventions, including a symptomatic treatment and an anti-amyloid monoclonal antibody. The development of novel disease-modifying therapies remains an active area of focus. Beyond drug discovery, Eisai is committed to advancing scientific understanding of AD, including characterization of AD biology to inform clinical practice.

MULTIPLE SMOLDERING PATHOLOGIES

AD is driven by a constellation of pathological changes that together result in progressive clinical symptoms first appearing as mild impairment, followed by worsening dementia[1]. When Alois Alzheimer discovered AD in 1906, he identified the two core pathological changes of amyloid-beta (Aβ) plaques and neurofibrillary tau tangles (NFTs) in the brain. Following more than one hundred years of research, we know that other changes, including neurodegeneration, inflammation, and vascular brain injury, also contribute to the onset of clinical symptomology characteristic of AD[1]. This network of interconnected pathologies spans multiple brain regions, collectively fueling the pathophysiological underpinnings of smoldering AD.

In a broad range of other disease states, the term "smoldering" has been used to describe a constellation of pathophysiological processes that drive gradual disease progression. For example, in multiple sclerosis (MS), much of the groundwork for this concept originated from studies on anti-inflammatory therapies, which showed that a significant reduction in lesions (also called plaques) was not sufficient to prevent disability progression[2]. These data, integrated among a synthesis of other studies, led to the viewpoint that numerous mechanisms beyond plaques, similarly called "smoldering MS", contribute to disease progression[3]. Indeed, smoldering is not restricted to inflammation in MS, but also encompasses other pathological changes including axon demyelination and mitochondria dysfunction, among others[3]. The smoldering model does not dismiss plaques as irrelevant, but instead, recontextualizes them as one component of the combined pathogenic dysfunctional process responsible for MS progression. Similarly, in AD, we recognize that the toxic processes that drive disease progression extend far beyond the focal, clinically measurable event of Aβ plaque deposition, and that a complex constellation of pathological changes is at play. This interplay of pathologies, including soluble Aβ (oligomers and protofibrils), insoluble Aβ (plaque), tau, inflammation, and neurodegeneration, all contribute to smoldering AD.

BEFORE THE SMOKE APPEARS

Decades before the onset of clinical symptoms in AD, a silent accumulation of neurotoxic material – referred to as "kindling" – sets the stage for smoldering disease activity. In the healthy brain, single units of Aβ, called monomers, are involved in several normal physiological functions, including processes required for learning and memory, among others[4]. In AD, normal Aβ production and clearance mechanisms are impaired, which starts the kindling process and causes these once-healthy Aβ monomers to misfold and aggregate into larger toxic Aβ structures called oligomers and protofibrils[4]. The oligomers and protofibrils diffuse throughout the brain and cause further pathological changes, including synaptic dysfunction, inflammation, and tau accumulation into toxic NFTs[4,5]. To counteract the kindling process, oligomers and protofibrils are sequestered into Aβ plaques, which also exert their own local toxicity[4]. This relentless cascade can occur unnoticed in a seemingly healthy person for up to 20–30 years[4,6] until the brain's neural reserve

is ultimately overwhelmed, igniting the onset of clinical symptoms (**Fig. 1**).

CLINICAL SYMPTOMS AND SMOLDERING ALZHEIMER'S DISEASE

While certain pathologies of smoldering AD (for example, tau) correlate more strongly with clinical decline, clinical symptoms are a consequence of collective brain injury driven by all components of smoldering AD, including toxic oligomers, protofibrils, plaque, inflammation, and neurodegeneration (**Fig. 2**). The complex interplay of smoldering pathology leads to the appearance of initially subtle clinical symptoms, which progress to mild cognitive impairment, and then mild, moderate, and eventually severe dementia. Recognizing and diagnosing AD early is crucial, since the currently available anti-amyloid therapies benefit patients in the early stages.

Researchers are actively working to understand what happens to smoldering disease processes and resulting clinical symptoms if one or more forms of Aβ are removed with an anti-amyloid therapy. In the revised criteria for diagnosis and staging of AD by the Alzheimer's Association (AA) workgroup, researchers noted that while clinical trials of anti-amyloid therapies support a positive relationship between the reduction of insoluble Aβ (plaque) on positron emission tomography (PET) imaging and clinical outcomes, a reduction of insoluble Aβ below detectable thresholds does not mean that the underlying pathogenic disease processes have all been stopped[1]. Indeed, after stopping treatment with an anti-amyloid therapy, patients have shown re-accumulation of pathological biomarkers and a reversion to placebo rates of clinical decline[1,7]. The AA workgroup introduced the term "treatment-related amyloid clearance" (TRAC) for patients treated with an anti-amyloid therapy who have changed from positive to negative on amyloid PET, yet in whom the pathogenic process is still active[1]. This term aligns with the concept of smoldering disease, in that even if Aβ plaque is reduced to negative levels on imaging, soluble forms of Aβ, including protofibrils, as well as other aspects of smoldering AD including tau and inflammation, continue to drive disease progression.

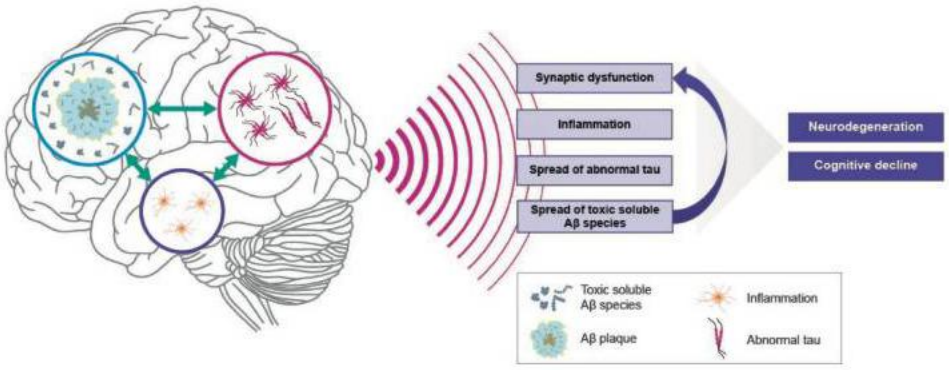


Figure 2. Proposed mechanisms of smoldering Alzheimer's disease. Abnormal Aβ, abnormal tau, and inflammation are key pathological processes that result in the progressive neurodegeneration and cognitive decline characteristic of AD. Soluble Aβ species are particularly important in fueling smoldering processes, in part due to their ability to diffuse readily throughout the brain, versus the immobile insoluble species. The extent and rate of clinical progression can vary considerably from person to person. Aβ, amyloid beta; AD, Alzheimer's disease.

IMPLICATIONS FOR CLINICAL PRACTICE

The concept of smoldering AD aligns with other chronic diseases, where progress has been made in understanding the multifaceted nature of smoldering disease to develop effective treatments targeting multiple pathologies. This refreshed model of AD pathophysiology arrives at a time when significant gains have been made in the field. Over the past several years there has been a cascade of global regulatory approvals for the two available anti-amyloid therapies for patients with early AD, one directed against aggregated soluble (oligomers and protofibrils) and insoluble Aβ, and the other directed against insoluble Aβ alone[8]. As we now understand, plaque removal alone does not address other aspects of smoldering pathology that continue to drive clinical deterioration in its absence. Smoldering AD helps to conceptualize the importance of intervention beyond Aβ plaque clearance, including the ongoing removal of toxic soluble Aβ species that fuel ongoing smoldering disease. Furthermore, targeting synaptic dysfunction, inflammation, and tau are important considerations to effectively address smoldering disease in AD.

Finally, the kindling phase of smoldering AD reinforces the need for early diagnosis and intervention before clinical symptoms appear. Currently, anti-amyloid therapies are approved for use in early AD patients experiencing clinical symptoms. However, we know that silent damage due to kindling processes occurs for decades prior to clinical decline. While implementation of a preclinical diagnostic approach requires ethical considerations as well as appropriate identification of individuals at high risk of progressing to symptomatic AD, the field is heading in this direction, with multiple trials investigating the use of anti-amyloid therapies in individuals with preclinical AD underway. Successful trials may someday lead to a world without symptomatic Alzheimer's disease.

Through the concept of smoldering AD, we hope to broaden disease state discussions beyond just plaque, to consider the chronic and truly multifaceted nature of this devastating disease. Our vision is to support the effort of a timely diagnosis and early treatment, to provide the best care for patients in need. ■

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How a Tiny Brain Region Guides Generosity

Whether and how much we help others may be determined by the brain's basolateral amygdala

BY TOBIAS KALENSCHER

IMAGINE IT'S SATURDAY MORNING. You're sipping coffee when your best friend texts, "Any chance you could help me move today?" You sigh—there go your weekend plans—but reply, "Of course." That afternoon you sweat as you carry boxes up a flight of stairs.

A week later a co-worker you barely know mentions that she's moving and could really use a hand. This time you hesitate. You are not as quick to offer help even though the request is nearly identical.

Why does generosity come so naturally for those we are close to but feel more like a burden when the recipient is a stranger or mere acquaintance? Psychologists call this tendency "social discounting": we are generally more willing to make sacrifices for people to whom we feel emotionally close, and our generosity declines as the social or emotional distance to the potential recipient of help increases.

But what happens in the brain when we make these decisions? And why are some people more generous to socially distant individuals than others are? In recent research, my colleagues and I gained new insight into these questions by examining a rare population of individuals with selective damage to a part of the brain called the basolateral amygdala. Our findings suggest that this small but important structure may be essential for calibrating our generosity based on how close or distant others feel to us.

Tobias Kalenscher is a professor of comparative psychology at Heinrich Heine University Düsseldorf in Germany.

The amygdala, a small, almond-shaped region nestled deep in the brain's temporal lobe, is traditionally known for its role in processing emotions, especially fear. But over the past few decades it has become clear that the amygdala, particularly its basolateral part, is a central hub in our social brain.

Across species, this region has been shown to participate in evaluating social rewards, empathic responses and decisions involving others. In rodents and monkeys, neurons in the basolateral amygdala encode the value of not just rewards for oneself but also the rewards received by others. And in humans, the structure has been linked to traits such as trust, empathy, moral decision-making and extraordinary altruism. Human amygdala volume also correlates with the size and complexity of a person's social network. And some evidence suggests that psychopathy and aggression are associated with a smaller, less functional amygdala.

So how, exactly, does the basolateral amygdala influence our decisions about whether to help others? One hypothesis is that this brain area allows us to balance competing helpful, social motives with self-interested goals. When you decide to help your best friend move, you are probably focused more on their benefit (making the move easier) than on the cost to you in time and effort. But when the person is a stranger, that mental calculation may shift. Some neuroscientists propose that the basolateral amygdala aids us as we navigate this trade-off by assigning value not just to our own well-being but also to the well-being of others.

To test this idea, my colleagues and I turned to a remarkable group of people in South Africa who have Urbach-Wiethe disease, a very rare genetic condition that causes selective bilateral damage to

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the basolateral amygdala while leaving the rest of the brain intact. In our study, we invited five women with this condition and 16 women without it to take part in a social discounting task. Each participant listed eight people from her own social network, ranging from her emotionally closest person (ranked as having a social distance of 1) to someone she barely knew (50) or a complete stranger (100). We then asked them to make decisions about how to split money. In each of several rounds, they received a fixed monetary amount and decided how much to share with each of their eight listed contacts. This task thus measured our participants' willingness to share resources depending on how emotionally close or distant they felt to the people in their social network.

As expected, the participants gave more to people they were close to than they gave to others who were more distant. That is, generosity declined as social distance increased. We found it interesting, however, that participants with damage to the basolateral amygdala were less generous overall than others, and their generosity decreased more sharply as social distance increased. They showed what we call steeper social discounting: they were still willing to help those they were emotionally closest to, but their willingness to give dropped off markedly for more distant individuals.

One participant with basolateral amygdala damage was an exception—she was ungenerous across the board, even toward her closest friend. But overall the pattern was clear: damage to the basolateral amygdala did not eliminate altruism, although it did distort the fine-tuned calibration of generosity based on social distance.

Notably, variations in personality, empathy or social network size did not explain the differences in generosity among our participants. Rather our participants with Urbach-Wiethe disease seemed unable to adjust their generosity flexibly to the social context.

At first glance our findings might seem to contradict earlier studies that found those with Urbach-Wiethe disease are actually more generous than others. For example, in past research people with this condition gave away more money in the trust game, a classic experiment in behavioral economics in which participants decide how much money to send to another player, the trustee. The amount sent is typically multiplied, and the trustee then decides how much to return. The initial amount sent is often seen as a measure of trust in the trustee. People with basolateral amygdala damage tend to send much more than others, even to untrustworthy trustees who fail to reciprocate.

Researchers have described this unusual pattern of trust as a form of "pathological altruism." In a similar vein, the authors of one study had people with Urbach-Wiethe disease respond to moral dilemmas involving hypothetical life-or-death decisions about others. They consistently refused to sacrifice one person to save many, revealing a marked reluctance to be responsible for causing harm to another individual in comparison with participants without the disease.

How, then, can we reconcile these earlier findings with our own results? We argue that the basolateral amygdala does not simply promote or hinder prosociality. Rather it is part of a neural network that helps people create a model of how the social world works, which they then can use to guide decision-making. With an intact basolateral amygdala, a person considers social context, social structure, social norms and learned expectations in social interactions when deciding whether to be generous or selfish.

When that system breaks down—as when someone suffers amygdala lesions—people may struggle to balance generous and selfish motives and consequently rely on simpler, default strategies that do not depend on networks that include this brain structure. In the trust game, the default assumption might be that others are trustworthy. In moral dilemmas, it could be to follow a rigid rule like "never harm anyone." Such ideas might have formed in childhood and, given damage to the basolateral amygdala, not been revised later in life, even in the face of contrary experiences with untrustworthy individuals. In our task, the default strategy is to maximize one's own payoff—unless the recipient is emotionally very close, in which case helping them comes automatically.

Although our study included only a small number of participants (which was unavoidable because of the extreme rarity of the condition), the distinctive pattern of brain damage in this group—symmetrical and precisely located in both hemispheres—is quite unique in neuroscience research. Other studies involving selective brain lesions have often relied on only one or two patients. We also feel confident in our conclusions, given how our work fits into a pattern of evidence drawing from more studies and participants that suggests amygdala functionality is crucial pillar of our social life.

The idea that the basolateral amygdala helps us weigh selfish and altruistic motives might sound abstract, but this interaction plays out in real life all the time. Think back to the moving-day dilemma. A generous impulse to help your friend move may come automatically because it is rooted in deeply encoded values and social bonds. Yet deciding whether to help an acquaintance requires something more: flexible, model-based decision-making that weighs social norms, reputational concerns and empathy against effort costs, self-care and the simple desire to have a pleasurable, lazy weekend. It's precisely in these gray areas that the basolateral amygdala seems to do its most important work.

Generosity is therefore not an all-or-nothing trait; it is a model-based social behavior shaped by the people we are interacting with and how close we feel to them. And deep in the brain, the basolateral amygdala is helping us do that calculus. ●

What I Wish Parents Knew about Social Media

I study social media for a living. Here's how parents can help their kids use it safely and productively

BY LAURA EDELSON

ON FRIDAY AFTERNOONS, shortly before the school bus arrives, my mom comes to my house. She ambles into my kitchen to make a cup of tea, and after a few minutes my preteen son runs through the back door. They will chat briefly, but inevitably my son will ask to watch YouTube videos on my mom's tablet. Then they will sit on the couch and watch videos of people playing Minecraft or Super Mario Odyssey or some combination of the two for an hour or so, until my husband and I finish work.

Occasionally I will find myself looking over their shoulders as formulaic videos with the same jump cuts and extreme close-ups, made by people I have never heard of (but who have millions of subscribers), play. I research social media for a living, and I still find myself wondering: Is it okay that my son is watching this? If junk food for the brain existed, these videos might be it.

Clearly, I don't ban all screens in my house, and I wouldn't tell you to ban them in yours, either. But in my work I've seen how little transparency social media companies offer parents and kids about how their systems operate—and how much harm that invisibility can do. In a recent look at the algorithms driving these platforms—what are called feed algorithms—my coauthors and I found that only one of the major platforms, X, makes details of how its system works publicly transparent. This is not okay.

There's some good news, though: through research not sponsored by social media platforms, we are learning more about where the most serious risks to kids and teens are and what things parents can do to manage those risks. Understanding the design of these systems, and talking with your child about them, is one of the most powerful tools you have to keep your family safe and supported online.

There are three things I wish every parent understood about how social media works and how to discuss it with their kids.

First, your teen isn't the customer—they're the product. I say teen because in general, kids under age 13 are not supposed to be on these platforms. Social media companies make money not just by selling ads but by collecting fine-grained data about what your child watches and reacts to and then monetizing those data by literally selling your child's attention to the highest bidder in the hopes of getting their money today or building a new lifelong customer for the future. Once kids are older than 18, social media companies can also sell their data directly to data brokers (just as they can do with yours). Just remember, on social media, advertisers are the customer, not you or your kid.

That doesn't mean people don't also get value out of social media, but for teens in particular, it can be hard to understand what they are exchanging for the entertainment they are getting.

What you can do:

- Talk to your kids about how the platform's business model works. Make sure they know that their at-

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tention and engagement are what's being sold.
- Talk to your teens about what they want to get out of social media. Do they want to keep up with their friends? Do they want to be entertained by influencers? Do they want to learn about trends or games or fitness? Decide together how they can be mindful about their goals and how you can support them in doing that. You should also figure out what they (and you) are and aren't comfortable giving away in exchange for whatever value social media delivers to them.
- Sit down together and go through the platform's ad preferences and privacy settings. Talk about what the settings do and decide together what's right for your child—and your family.

The second important point is that algorithmic feeds are designed to maximize usage, not well-being. Every major platform uses feed algorithms to keep users scrolling, watching or clicking as much as possible by offering them what they think the user will like next. I'm not aware of any platform that observes an "upper bound" on how much usage it will try to optimize for, meaning no matter how much social media users consume, their feed algorithms will keep trying to get them to use more. Platforms carefully calibrate aspects of what they show you in feeds, from the mix of topics and video lengths to other aspects of their system design, such as how often they notify users about reactions and comments to content those users have posted. Feed algorithms also adapt to each user's behavior and can quickly home in on whatever type of content users pay attention to or engage with the most. I think even the most social media–loving teen understands that more isn't necessarily healthy.

What you can do:
- Talk to your kids about how feed algorithms work. You can use the "feed cards" my co-authors and I developed to explain how these systems work on platforms such as TikTok, YouTube and Facebook.
- Ask your teen to show you their feed, then scroll through it together. Can you "hack" the algorithm by watching certain videos or reacting to certain types of content and seeing how quickly the algorithm adapts? Talk to your kids about how (and whether) they can stay in control of their experiences when they are using algorithmic feeds.
- Remember that you can set limits without banning social media. Social media algorithms will always try to get your kids to use them more. Some teens don't have the self-control to step away, even when they are having experiences that make them feel bad. Has something on X or Facebook ever made you incandescently angry? If so, that was probably the point—rage-bait works. And if you're over 25, your prefrontal cortex is fully developed; think about how that moment might have felt to your child. Talk to your kids and set sensible limits on how late in the evening they can use social media and how much time they can spend on it overall. Use in-app tools to set limits but remember that kids often know how to circumvent these, so pay attention to where your kid's device is, too.

Third, although content moderation exists, don't count on it. It's natural to hope that social media companies are catching and removing harmful content before your child sees it. But surveys show that even young teens report seeing content on social media that disturbs them. How is this possible? Platforms do take down a lot of content, but as my research shows, content removal often occurs after feed algorithms have already presented the harmful content to most people who will ever see it.

What you can do:
- Don't assume "the system" will catch everything; it won't.
- Ask open-ended questions about your kids' social media experiences: "What's the best thing you saw on TikTok this week?" "Have you seen anything that upset or confused you this week?"
- Remember that different platforms have different rules and different enforcement. If your teen is routinely having experiences that make them uncomfortable, it might be time for them to shift to another platform that feels safer for them or even take a break to reevaluate whether what they are getting from using a given social media platform is really worth it.

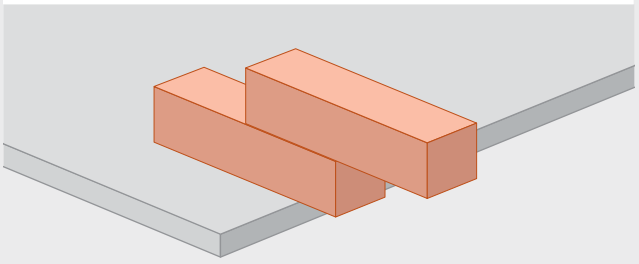
I'm not going to pretend any of this is easy. My research has shown that the transparency tools that platforms offer are difficult to use and understand. But we parents can give our kids their best chance to develop a healthy relationship with social media if we stay engaged, curious and consistent. Finally, hold the line on some nonnegotiables that have the most potential to cause harm:
- Make sure kids and teens don't have phones or other access to social media in their bedrooms overnight. Kids need sleep, not all-night Snapchat sessions.
- Don't let kids lie about their age to join platforms early or to get an "adult" account instead of a "teen" one if they're younger than 18. On many platforms there are meaningful differences in default settings, data collection and even feed algorithms between teen and adult accounts.

I'm still learning, as both a researcher and a parent. But what I've learned so far tells me this: there are ways to help teens have safer, better online experiences, but kids need involved parents and consistent rules to make sure that happens. ●

A Block-Stacking Problem with a Preposterous Solution

In principle, this impossible math allows for a glue-free bridge of stacked blocks that can stretch across the Grand Canyon—and into infinity **BY JACK MURTAGH**

HERE'S A MIND-BLOWING EXPERIMENT you can try at home: Gather some children's blocks and place them on a table. Take one block and slowly push it over the table's edge, inch by inch, until it's on the brink of falling. If you possess patience and a steady hand, you should be able to balance it so that exactly half of it hangs off the edge. Nudge it any farther, and gravity wins. Now take two blocks and start over. With one stacked on top of the other, how far can you get the end of the top block to poke over the table's edge?



Keep going. If you stack as many blocks as you can, what is the farthest overhang you can achieve before the entire structure topples? Is it possible for the tower to extend a full block length beyond the lip of the table? Does physics permit two block lengths? The stunning answer is that the stacked bridge can stretch forever. In principle, a free-standing stack of blocks could span the Grand Canyon, no glue required.

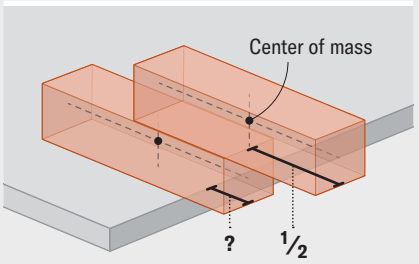
Don't click "checkout" on an infinite pack of Jenga blocks just yet. Real-world practicalities such as irregular block shapes, air currents and the crushing weight of an endless edifice may hamper your engineering aspirations. Still, understanding why the overhang has no limit in an ideal mathematical world is enlightening. The explanation hinges on math's harmonic series and the physics concept of center of mass, two seemingly simple ideas with outsize power.

Jack Murtagh is a freelance math writer and puzzle creator. He writes a column on mathematical curiosities for *Scientific American* and creates daily puzzles for the Morning Brew newsletter. He holds a Ph.D. in theoretical computer science from Harvard University. Follow him on X @JackPMurtagh

Your intuition might tell you that a single block can hang half of its mass beyond the table's edge before tipping. But why is that so? Every object has a center of mass—a single point at which we can imagine the entire object's weight to be concentrated when we're thinking about its balance. As long as the center of mass sits above the table, the object stays put. The moment that center of mass passes over an edge, however, gravity will pull the whole thing over.

In the case of a spoon, an item with irregular weight distribution, we can hang more than half of the utensil's handle over an edge before it tips because the center of mass lies closer to its head, where more of the weight resides. For our stacked bridge, we assume that our blocks all are identical and have uniform density (that is, they're not denser in some parts than others), so each one's center of mass sits at its middle point.

When we add more blocks, we must account for the center of mass of the entire tower. Consider the case of two blocks. We know the top block can extend half of its mass beyond the one below it. But after doing that, how far can we push out the bottom block?

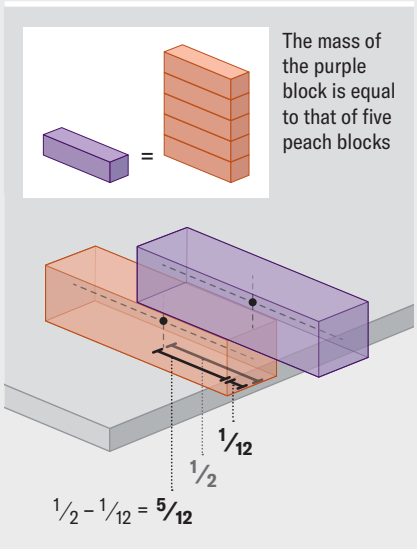


For simplicity, let's say each block has a length of 1 and a mass of 1. You'll find that the bottom block can poke out only a quarter of its length (compared with half when it was alone). At that point the center of mass of the top block and the center of mass of the bottom block are equidistant from the edge of the table (the bottom block's center of

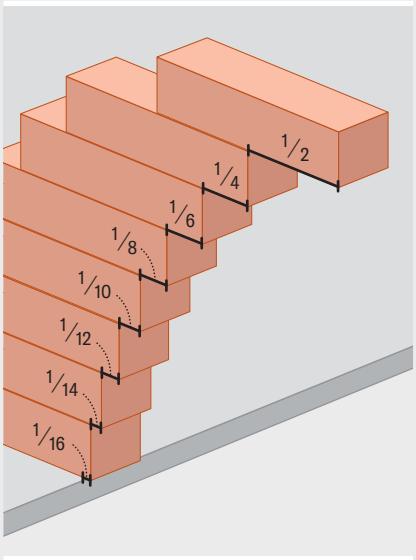
Graphics by Amanda Moñtanez

mass sits on the table ¼ in from the edge, and the top block's center of mass sits off the table ¼ beyond the edge). So the combined center of mass of the two-block system rests perfectly balanced above the edge of the table.

A pattern emerges as we continue to add blocks to the structure. The top block extends ½ beyond the one below it, the second block extends ¼ beyond the block below it, the third extends ⅙, the fourth extends ⅛, then subsequent blocks extend 1/10, 1/12, and so on. To see why, let's look at another example. Suppose we have a stable tower that contains five blocks, and we want to add a sixth block below it and then slide the whole structure out as far as we can. It's helpful to conceptualize this with only two blocks: one with a mass of 5 atop a single block with a mass of 1. We'll first scoot the heavy block as far as it will go so that its center of mass sits right above the bottom block's edge. We can then push the bottom block exactly 1/12 of a unit beyond the table's edge. How do we know that?



Again, the answer comes down to balancing out the centers of mass of the two blocks, but this time, because the top block is five times heavier, the bottom block's center of mass must end up five times farther in on the tabletop to counteract the weight. This relation is known as the law of the lever—think about how a book feels heavier in your hand the farther you move it away from your body, so that a paperback at the end of a fully extended arm might feel equivalent to a textbook held close to your torso. The distance between the top block's center of mass and the table's edge is 1/12, and the distance for the bottom block is ½ − 1/12 = 5/12, or five times more. A similar calculation reveals the correct overhang at every level of the tower.



Answering our opening question (how far out can the tower extend?) amounts to adding up all these successive overhangs. If you have 10 blocks, they can extend to ½ + ¼ + ⅙ + ⅛ + 1/10 + 1/12 + 1/14 + 1/16 + 1/18 + 1/20, which equals about 1.464 block lengths beyond the edge. But what is the limit to how far we can stack blocks? For that, we must add infinitely many of these shrinking terms. The resulting pattern bears a striking resemblance to one of the most famous infinite sums in math, the harmonic series, which takes the reciprocal of every counting number (that is, 1 divided by every positive integer) and sums the values: 1 + ½ + ⅓ + ¼ + ⅕ + …, and so on forever.

If you look closely, you might notice that the overhangs from the block-stacking problem are exactly half of each of these terms: ½ + ¼ + ⅙ + ⅛ + 1/10 + …

Calculus, the branch of math that digs into how things change, teaches us that even when we are adding up infinitely many shrinking terms, sometimes the sum converges on a finite value, and sometimes it diverges to infinity. The total of the harmonic series grows incredibly slowly. The first 100,000 terms add up to about 12.1, and the first million terms equal only around 14.4. Still, at a relentless snail's pace, the harmonic series grows forever.

Each individual overhang in the block-stacking problem equals half of a term in the harmonic series. Because half of infinity is still infinity, the tower's potential overhang also has no bound.

Of course, although translating pure math into practice always comes with hurdles, the block-stacking problem offers an amusing dexterity challenge. With only four blocks, you should be able to extend the top one a full block length past the edge (½ + ¼ + ⅙ + ⅛ ≈ 1.042). To fulfill my journalistic due diligence, I tried this at home with playing cards on my coffee table. After a few minutes of patient tinkering, I managed to balance the top card just beyond the edge, hanging it entirely off the table, and I felt like a magician.

Two full block lengths beyond any surface would require 31 pieces. Meanwhile 100 million pieces wouldn't even get you a full 10 block lengths of overhang, because the sum of the first 100 million terms in the harmonic series all divided by 2 equals about 9.5. So it will take some grit to span the Grand Canyon. At huge scales, physics kicks in to topple mathematicians' fun. But in idealized conditions where center of mass and the harmonic series alone rule the roost, the possibilities are literally endless. ●

Pets, Health and People

Only when human-pet relationships are strong, it seems, do owners get physical and mental benefits from their animals BY LYDIA DENWORTH

WE GOT OUR FIRST DOG when my oldest son was 10. A friend who was a teacher told me that was a perfect age for a kid to have a pet. "Jake can throw his arms around the dog when he doesn't feel comfortable hugging you anymore," he said.

It took a bit for me to get over his reminder that my child was growing up, but I immediately recognized my friend's insight. A beloved animal can make everything seem better. And most of us believe strongly that our pets make us healthier.

Yet the science of human-animal interaction has found mixed results when it comes to physical and psychological health benefits from pets. Depending on the study, for example, people with pets are either less or more likely to be depressed. Experts say this seesawing probably happens because, for some owners, pets serve as a calming influence and emotional support. But in other cases, the study may include more people who are already struggling mentally and get pets to try to feel better; then such participants are counted as depressed.

Owning a dog has consistently been associated with higher levels of physical activity, no doubt because of all that walking, which has social benefits, too. One of the very first studies in the field, published in 1980, found that people who had been hospitalized for a heart attack or coronary artery disease were more likely to survive the following year if they had a pet, and the researchers suspected that physical activity from walking dogs was partly responsible, although the results held for other kinds of pets, too. A 2019 analysis of several studies, published in *Circulation: Cardiovascular Quality and Outcomes,* showed a dramatic result: dog ownership was associated with a 24 percent lower risk of dying. But when other researchers reran the same numbers with more adjustments for confounding variables, that benefit nearly disappeared.

A history of physical activity is one potential confounder. "You're more likely to have a dog if you're already somebody who's active or wants to be active," says developmental psychologist Megan Mueller of the Cummings School of Veterinary Medicine at Tufts University. "And then once you have that dog, they probably help motivate you to be more active."

That's why much of the latest research aims to get beyond such problems by digging into the nuances of human-animal interactions. "Pets are not a medical intervention; they're a relationship," says Jessica Bibbo, a gerontologist at the Benjamin Rose Institute on Aging in Cleveland who studies human-animal interactions. And the quality of that relationship, such as the level of attachment and sense of social support, looks like a much better predictor of positive outcomes than just whether there's a pet in the home, Mueller says: "We are trying to isolate the factors that can help promote those positive relationships [with pets] so we can help people."

Carefully randomized controlled trials with therapy animals and laboratory experiments offer some clues. A 2025 study had 43 dog owners perform stressful tasks (such as public speaking) with or without their pets present. Those whose dogs accompanied them showed lower spikes of cortisol, a hormone that rises under stress. Another study of about 90 older adults attending a community center randomly assigned half the people to look after five crickets (yes, crickets!) in cages for eight weeks. All the people received the same advice about maintaining their own health. Those who cared for insects showed some improvement in mental and cognitive health compared with those who didn't.

Continued on page 77

Lydia Denworth is an award-winning science journalist and contributing editor for *Scientific American*. She is author of *Friendship* (W. W. Norton, 2020).

Illustration by Jay Bendt

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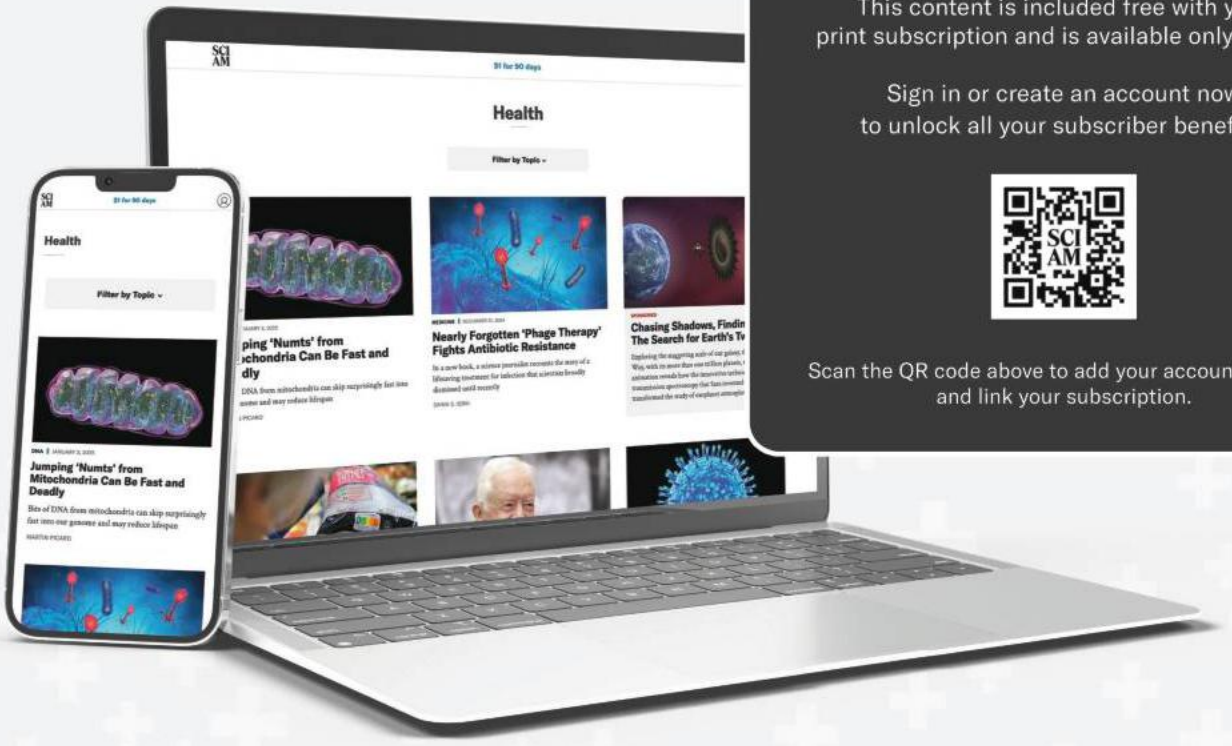

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Continued from page 74

For older adults, having a pet to care for adds a sense of purpose, Bibbo says, particularly when health is in decline. As part of their work, Bibbo is trying to build pet care into decisions about health care. People often take better care of themselves so they can also look after a loved animal, Bibbo says.

Some of the positive effects seen in controlled settings—such as reduced cortisol levels and heart rates—probably carry over to having a pet in real life, Mueller says, even though real life is messier. As with human relationships, strong, positive bonds with an animal seem to be some of the things that confer health benefits (although even here there are mixed results). Certainly pets provide social and emotional support for many people. There's a physical component as well from having a cat or dog sit in your lap. As a bonus, pets are viewed as nonjudgmental. "Pets aren't giving you any tough love," Mueller says.

For adolescents, that can be especially useful (my friend was right). Pets serve as "a bridge helping young people in their transition to autonomy," says Mueller, whose work focuses on that age group.

Still, we shouldn't ask too much. Even therapy animals are there to facilitate, not to fix, Bibbo says. And we can't expect pets to cure serious mental health issues, Mueller says. "But can having a dog or any pet help us build coping skills that are positive for managing anxiety?" she asks. Mueller thinks it's very possible.

People emphatically believe pets improve our quality of life, and that belief can affect health indirectly. In 2025 economists used a large British dataset with controlled variables to assess how much more money pet owners thought they would have to earn to get the same life satisfaction that pets gave them. The conclusion: up to $90,000 a year. That's enough to buy dozens of treadmills or go on many relaxing tropical vacations. Co-author Adelina Gschwandtner of the University of Kent in England says: "Are pets good for us? We were able to answer with a resounding yes." ●

Science History Images/Alamy Stock Photo

Love Letter from Photograph 51

"[c]learly Rosy had to go or be put in
her place.... The thought could not be
avoided that the best home for a feminist
was in another person's lab."
—James D. Watson, *The Double Helix* (1968)

I am the image, that final clue.
I know only this lab, where light can simmer for days,
coaxing shadows to slowly define
the tiny drop you tip so carefully
onto the end of a twisted paperclip.

The lab, and you, squinting into the lens
of a machine you developed—
hydrogen gas pumped through a salt solution—
on the fulcrum between question and discovery.

In the lab, the men call you names, mock your clothes,
your moods, your lips, unpainted.

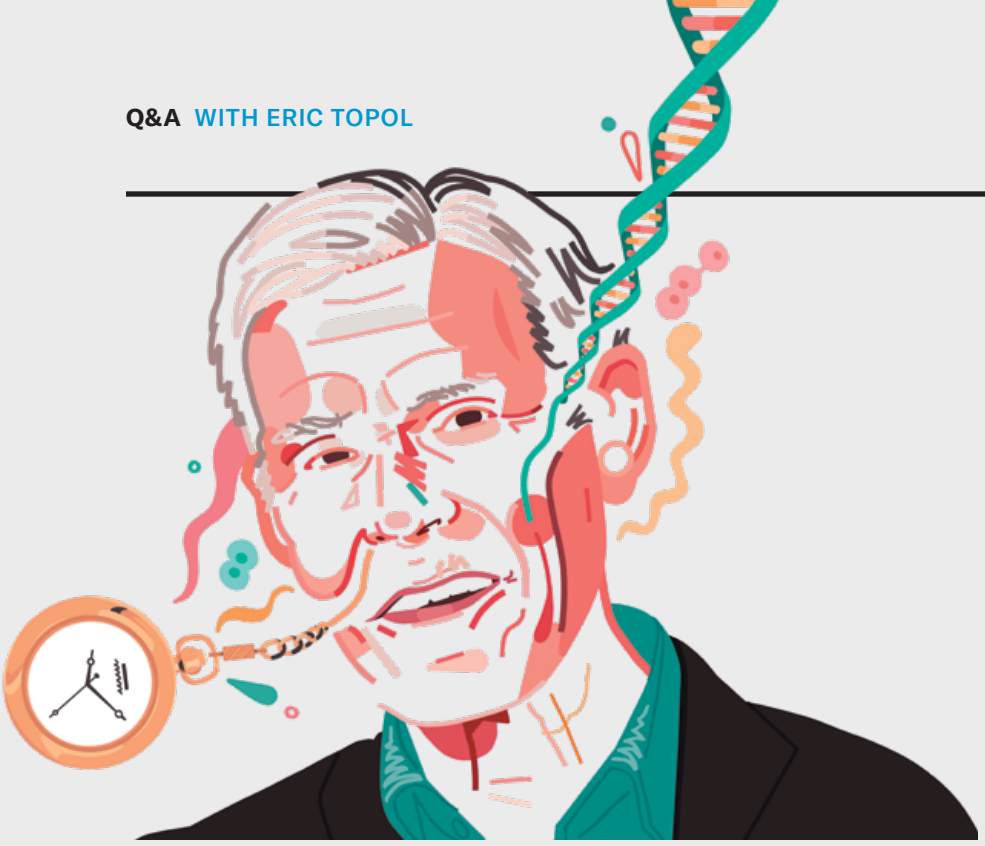
Your delight, your choice of gardenia
is science.

Not on the first but the fifty-first iteration,
I come to you
in the honey of crystallography
amid x-rays splattered off a fiber of wet DNA
like a tadpole on a sliver of glass.

I swim up, rapt, to visibility.
I whisper my secret only to you,
the clue, first word of the organic story.
The ancient code-script
pinned down at last—
The recipe for whale song
and peacock feathers
earlobe and pea plant, X and Y.

I fix my focus in your eyes, Rose Franklin.
I your discovery. You my laureate.

Faith Paulsen writes poetry from her desk at an insurance agency near Philadelphia. Her work appears in *Blue Heron, Mania, Poetica Review, Philadelphia Stories, Book of Matches, One Art, Panoply, Thimble,* and chapbooks *Cyanometer* (Finishing Line Press, 2021) and *We Marry We Bury We Sing or We Weep* (Moonstone Press, 2021).



Biological Age vs. Chronological Age

Investigating the science and hype of biological age tests

BY LAUREN J. YOUNG

HOW OLD ARE YOU REALLY? Birthdays may be a common tally, but your "age" isn't determined by time alone. New research increasingly shows the importance of considering chronological age as something very different from biological age—in which the body and its cells, tissues and organs all have separate "clocks" that can tick at different speeds.

"Calculating biological age, I think, is core to the advances we have made in the science of aging," says Eric Topol, a cardiologist and genomics professor at Scripps Research in La Jolla, Calif. "It's a way you can tell if a person, organ or any biological unit is at pace of aging—if it's normal, abnormal or supernormal."

In his newest book, *Super Agers: An Evidence-Based Approach to Longevity*, Topol delves into the recent surge in public interest in biological aging and the accelerating quest to refine ways of measuring it. Improved biological timekeeping can give a more precise picture of a person's longevity prospects and of potential ailments that can be prevented or treated early. SCIENTIFIC AMERICAN spoke with Topol about the latest research in biological aging, factors that might speed it up or slow it down, and what it can tell us about our current and future health.

An edited transcript of the interview follows.

Lauren J. Young is an associate editor for health and medicine at *Scientific American*.

How is biological age determined, and how has the research evolved?

This research was really started more than a decade ago by geneticist Steven Horvath with his "clock" test, with which, basically using saliva, you could look at specific genetic markers and predict a person's biological age. His clock is really known as an epigenetic clock or methylation clock. As people age, DNA changes and gets methylated—a methyl-group molecule attaches to specific nucleotides of DNA. I kind of liken it to the body rusting out. Essentially you're getting marks at specific parts of the genome that track with aging in humans and every other species of mammal.

In Horvath's initial test, there clearly was a detection of both alignment with the person's real age, or chronological age, and when it wasn't matching up. In other words, if a person's biological age was off by a few years from their real age, you'd wonder why.

Then what's proliferated in the more than 10 years since has been all these other clocks: protein clocks, RNA clocks, immune system clocks—you name it. Using plasma proteins from a blood sample, we can also clock organs—the heart, brain, liver or kidney. So we have seen just enormous advances in these clocks, and they keep getting refined with added features. There's a race to get the best clocks to predict survival.

What can biological age tests tell us clinically?

We can detect in an individual if something's not right at different levels. For example, if your biological age is five years older than your real age, is there an organ that might be linked with that? Then you can use these clocks to see whether lifestyle changes, prevention or treatment can slow down the pace of aging and get it into alignment with your actual age.

The question is: When will doctors actually start using them? The medical community is very hard to change. So it hasn't happened yet, but I believe it will eventually. Tests are also made available

Illustration by Shideh Ghandeharizadeh

by commercial companies, but they can be very expensive. You can run an epigenetic test in a very simple way for $10 or $20, but some of these companies are charging $200.

I haven't seen their publications to be able to say with confidence that they are doing things right, and the lack of standards from one company to the next is disconcerting. They don't want to shock customers by telling them that they're 10 years older than their chronological age. Eventually, I believe, we're going to have high-fidelity epigenetic clocks with no motivation for a provider to hold things back if a person's data are really bad.

Why might someone biologically age "faster" or "slower" than they do in actual years?

If you had to pick one mechanism behind why biological age and chronological age are misaligned, it would most likely be that some genes are either protective or linked with accelerated aging—but that's such a small part of the story. Another root cause appears to be that our immune system gets weaker and less functional as we get older. In the average person, this change starts around age 55 to 60. The immune system's level of protection drops, or it gets dysregulated—off track—and it can have an untoward, hyperactive response. When that happens, you start to see inflammation in the organs, such as in the arteries of the heart or the brain—it's what I call "inflammaging."

Obviously our lifestyle also has a big impact—eating a really healthy diet that's not proinflammatory and doesn't have a lot of ultraprocessed foods or red meat is beneficial. Good sleep health helps to reduce inflammation. There's only one thing that's been definitively shown to slow the epigenetic aging process, and that's exercise. I think these clocks ultimately are going to be very good incentives for people to adopt a healthy lifestyle. We can't get everybody to do all these things that we know help them, but if they get their own data and see something's off track, the hope is that they'd change their habits. That's, of course, just one of the ways to prevent diseases. There are also drugs and other treatments.

Improved biological timekeeping can give a more precise picture of a person's longevity prospects.

What environmental factors are also important to consider?

We have all kinds of food deserts in the U.S. We have air pollution and unmitigated accumulation in the air and water of microplastics and nanoplastics, which get into every part of our body and induce inflammation. And we have forever chemicals that are pervasive. These all play a role in health and aging.

Let's talk more about inflammaging. We know some inflammation can be good for the body—to fight infections, for instance—but a lot can be bad. How does chronic inflammation potentially accelerate aging?

Inflammation and aging are so tightly intertwined. The immune system is really the driver for good when it attacks pathogens and for bad when it promotes too much inflammation in walls of arteries or the brain. That's heart disease and neurodegenerative disease, respectively. But what's so exciting is we can dial up or down the immune system now. For example, there have been natural, amazing experiments with the shingles vaccines, which reduce dementia and Alzheimer's disease by 20 to 25 percent. So how does that work? Well, the vaccine amps up the immune system in people. That's going to be the critical thing in using these metrics: zooming in on the immune system and inflammation to keep people's immune system intact and stop it when it starts to go haywire. That's the future. In the last chapter of my book, I presented the first cut of my "immunome"—an assay of every virus and pathogen I've been exposed to, every antibody I have. But that's just scratching the surface.

The immune system clock could turn out to be the most useful of all; if I could pick one, that's the one I would want. But the immune system is very complex. Maybe we don't have to do a systematic, comprehensive assessment of our immunome that would include checking antibody titers and sequencing B cells, T cells and interferons. If we can use just a group of plasma proteins, that would be terrific. That remains to be seen. There's a human immunome project just getting started to try to compare things such as the proteins with the much more sophisticated and expensive ways to get at the health of an immune system.

What are the downsides of slowing down biological aging or of extending lifespan?

We feel really great if we get to age 85. "Super agers" who don't get one of the big four age-related diseases [type 2 diabetes, cancer, or heart or neurodegenerative disease] can say, "Well, I did it." Of course, if you get to age 98, you're really doing well. I think we're going to have a whole lot more super agers. But that's not going to get around the fact that eventually they're going to develop some problems—one of the big four or other conditions. It could be you get an infection because your immune system is just too weak. Or it could be you break your hip because your bone density is so low, and you wind up with a pulmonary embolus [a clot that blocks blood flow to the lungs].

Eventually you die, and you may have a chronic illness between that point of extended health span and when you die. I don't want to put a sense out there that super agers won't see problems in the latter stages of their lives. But the point is, let's extend the health span—high-quality life without these big age-related diseases—as much as we can before getting into the downturn of a health arc. ●

An artist's impression of our far-future sun is seen from a molten Earth landscape.

Can We Survive the Death of the Sun?

In a few billion years the sun will turn into a red giant star BY PHIL PLAIT

IT'S IMPOSSIBLE TO FORGET JUST HOW MUCH the sun affects life on our planet. It's overwhelmingly the source of our light and heat, providing just enough to maintain the delicate climatic balance we enjoy. That's not a coincidence; life on Earth evolved under the sun's influence and, given time, adjusts to any changes.

Adapting to the whims of a star is no small task, however. The sun may appear to be constant from day to day, but let time stretch out for millions or even billions of years, and things will change—a *lot*. And not always for the better.

Phil Plait is a professional astronomer and science communicator in Virginia. He writes the *Bad Astronomy Newsletter*. Follow him on Beehiiv.

For example, in its thermonuclear-driven core, the sun fuses about 700 million tons of hydrogen into 695 million tons of helium every second. The missing five million tons are converted into energy (via everyone's favorite equation, $E = mc^2$). This energy is enough, it turns out, to power a star. If you like mind-boggling numbers, the sun produces 4×10^{26} watts of power—400 trillion trillion watts. In other words, the energy our star emits in a single second is sufficient to satisfy humanity's total consumption for about 650,000 years.

It's also enough to warm our planet to its current comfy clime. In fact, by using some basic physics principles, it's possible to mathematically calculate how warm Earth should be given the

Chris Butler/Science Source

Astronomers argue about whether the expanding sun will reach Earth. As it stands, things don't look good.

sun's energy-emission rate. That solar energy flows into space in all directions around the sun, and a tiny fraction (about half of one billionth) of it is intercepted by Earth, heating our planet. Just how much heating takes place is a bit complicated and depends on the actual radiant flux from the sun, Earth's distance from it and reflectivity, and more. When we run the numbers, Earth's average calculated temperature today is approximately –15 degrees Celsius, colder than the freezing point of water.

Actual measurements of Earth's temperature, however, give an average that's much warmer: about 15 degrees C. The difference exists because greenhouse gases in the air essentially trap heat from the sun, warming Earth above the calculated temperature. This warming is mostly from natural greenhouse gases, mind you, but we're adding approximately 40 billion tons of carbon dioxide into the atmosphere every year, significantly increasing the warming effect. Note that this increase has occurred over the past century or so, a timescale far too short for there to have been any change in the sun; Earth's current climate change is all us.

But the sun's production of energy does change noticeably—over hundreds of millions of years. That helium created in the core is inert; think of it as ash from the nuclear fusion. It settles in the center of the sun, building up over time at the rate of 695 million tons per second! As it gains mass, it also gets squeezed by the tremendous weight of the sun's layers above it and becomes compressed. A basic law of physics is that compressing a gas heats it, so even though the fusion rate is mostly the same, the core of the sun is still slowly heating up over time—which means the sun itself is getting more luminous.

If we run the clock forward an eon or two, we find disaster. As the sun grows brighter, it will raise Earth's temperature so much that the planet will lose all the water vapor in its atmosphere and then, eventually, all its surface water. The oceans will evaporate. This global desiccation will pretty firmly plant a stop sign for all life on Earth. Still, if it's any comfort, that won't happen until three billion years from now.

The reactions going on in the sun's core get very complicated after this point, but the biggest effect is that our star's energy output will eventually increase prodigiously. All that energy will get dumped into the sun's outer layers. When you heat a gas, it expands, so the sun will swell up to huge proportions—100 to 150 times as wide as it is now. At the same time, its surface temperature will drop, making it ruddier, even as it radiates energy 2,400 times stronger than what it puts out now. This shift will transform the sun into a red giant star.

The sun will be so big, in fact, that it will consume Mercury and Venus. Earth may escape this fate; astronomers argue about whether the expanding sun will reach Earth. As it stands, things don't look good.

If Earth does survive, it won't be pretty. The temperature of our planet will be about 1,300 degrees C, hot enough to melt lead. During the day rocks on the surface will melt, and Earth will be a lava world. On top of that, our planet will lose its atmosphere to space when it gets this hot. Is there any way for Earth to avoid this destruction? How will the other planets fare?

To answer both these questions honestly and in the order they were asked: not really and not well. There will be a slight reprieve because as the sun expands, its solar wind will become much more powerful—so much so, in fact, that the sun will lose a substantial amount of mass. This means our star's gravity will weaken, and the planets will migrate outward, away from the solar system's central blast furnace.

But it's not enough. Jupiter, which is currently a chilly –110 degrees C, will heat up to more than 300 degrees C. Its icy moons will melt and start to boil away. We'll find no sanctuary there.

If you want to find even a marginally clement climate anywhere in that far-future solar system, you might have to look toward Pluto, which, by then, will be about 50 times more distant from the sun than Earth is now. Its surface temperature will be roughly –10 degrees C. That's still chilly, but remember the greenhouse effect: there is a lot of frozen methane and carbon dioxide on Pluto, so these ices could vaporize and possibly provide enough thermal retention to make the tiny world at least somewhat habitable, if not exactly comfortable.

What then? It gets worse, if you can imagine. The sun will blow off its outer layers, and the core will be exposed to space, transforming into what astronomers call a white dwarf. The incredibly hot core will be only about the size of Earth, so small that it will provide very little heat to the planets. They will cool once again, eventually dropping well below the freezing point of any biologically useful molecule.

If there's any modicum of good news here, it's that all of this won't happen for many billions of years. Who knows what humanity will look like by then or whether we'll still be around? If we are, well, more stars are born all the time, and they'll have planets, too. Packing up and moving is never fun, but if your house is on fire, there's not much choice. Perhaps we can find other Earths out there where we can settle down for an eon or three before this entire process starts up again. ●

Fusion Dreams

Scientists are refining three main models for fusion energy reactors

TEXT BY CLARA MOSKOWITZ
GRAPHICS BY MATTHEW TWOMBLY

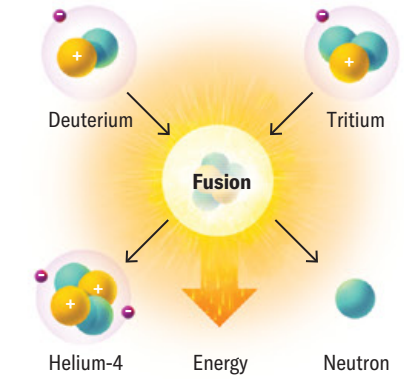
NUCLEAR FUSION promises a green and infinitely renewable supply of energy—if we can harness it. Fusion happens all the time inside the sun. But to re-create the process on Earth, we must control incredibly hot, chaotic matter in an exceedingly dense state.

Prototypes of several different fusion-reactor designs are being tested around the world. The National Ignition Facility (NIF) at Lawrence Livermore National Laboratory in California, for example, uses lasers to spark fusion in a small pellet of fuel. Tokamaks, such as the International Thermonuclear Experimental Reactor (ITER) in France, use electromagnetic fields to confine plasma and heat it to the temperatures and densities necessary to ignite fusion. And stellarators, such as the Wendelstein 7-X experiment in Germany, add a twist to the magnetic field concept of tokamaks.

It's too soon to say whether any of these technologies can overcome their challenges to become a reliable energy source. But the motivation to make that happen is clear. "Necessity is the mother of invention," says Laura Berzak Hopkins, associate laboratory director at the Department of Energy's Princeton Plasma Physics Laboratory (PPPL). "We have increasing energy demands and a changing climate, and fusion is the way we can address both those needs."

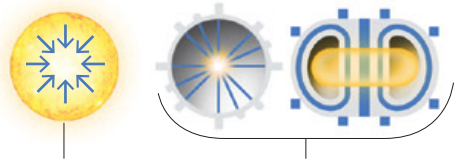
WHAT IS FUSION?

Nuclear fusion is the process by which two atoms combine to form a larger atom (minus a bit of mass) plus energy.



To achieve sustained fusion, the atoms must reach a certain temperature and density, and they must stay in these states for an extended period. There are three general ways to meet these conditions.

GRAVITATIONAL CONFINEMENT | INERTIAL CONFINEMENT | MAGNETIC CONFINEMENT



Within stars, gravity is intense enough to hold particles at the right heat and density for long enough to sustain fusion.

On Earth, inertial and magnetic confinement are two strategies to reproduce the conditions in stars. Both methods, however, still struggle to extract more energy from fusion than they use to produce it.

The goal is to get more sustained energy out of the system than goes in.

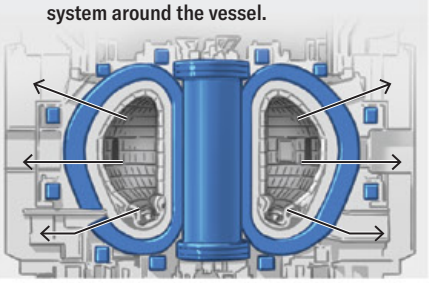
Experiments in 2022 at NIF —the most famous **inertial confinement** facility—provided proof of concept. The project did release more fusion energy than its lasers used to create the reaction, but charging those lasers incurred an energy cost.

Recent experiments using **magnetic confinement** have also demonstrated progress. Two different concepts—a stellarator and a tokamak—have each held superheated plasma at the right temperatures and densities for nearly one minute, achieving new records. Why is this significant? Containing the fuel for sustained times is a huge challenge. To understand why, let's dive into an example.

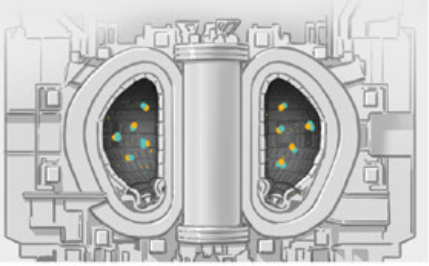
MAGNETIC CONFINEMENT

Tokamak reactors—such as the massive ITER project, which is still under construction—use a doughnut-shaped container. Here's how they work:

1 Remove all gas from the vacuum chamber, then charge the magnetic system around the vessel.



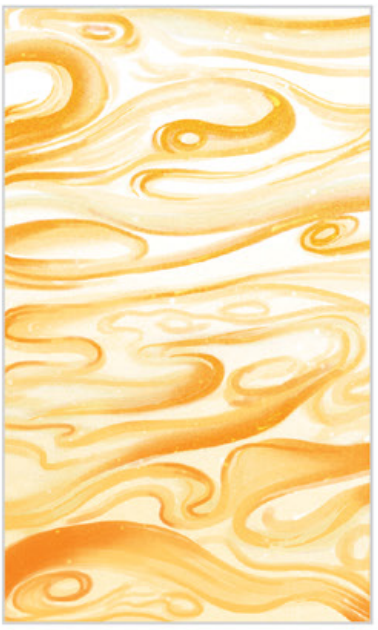
2 Inject a small amount of deuterium and tritium gas into the vacuum.



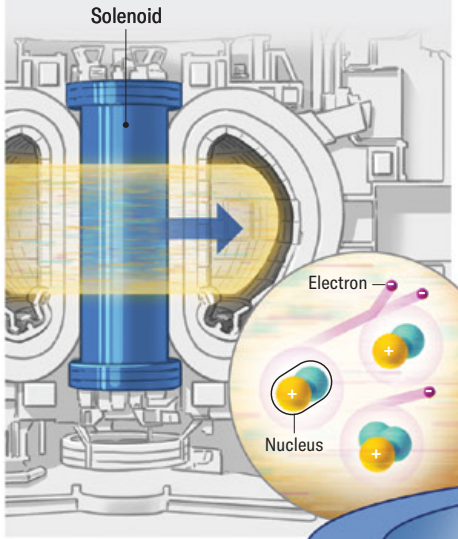
WHAT'S THE PROBLEM?

The process seems straightforward. So why is it so difficult?

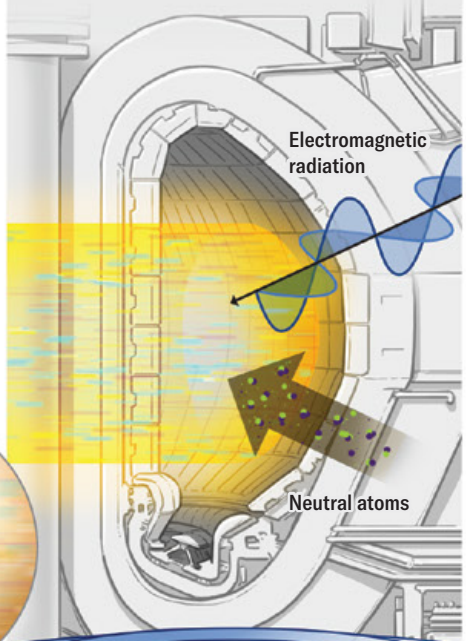
When left to its own devices, plasma is turbulent, with pockets of temperature variations that create convection currents. This turbulence also moves heat from the plasma core to the edge, dampening the fusion reactions.



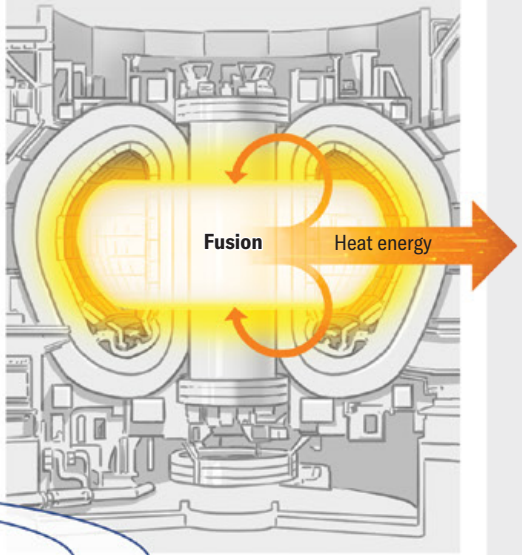
3 Switch on the coil of wire called a solenoid at the center of the tokamak to start up the magnetic field that will keep the gas contained. Run a powerful electric current through the vessel. This current strips electrons off the gas particles, which collide with other particles to kick off more electrons. The atoms become an ionized gas called a plasma, in which charged particles follow magnetic field lines.



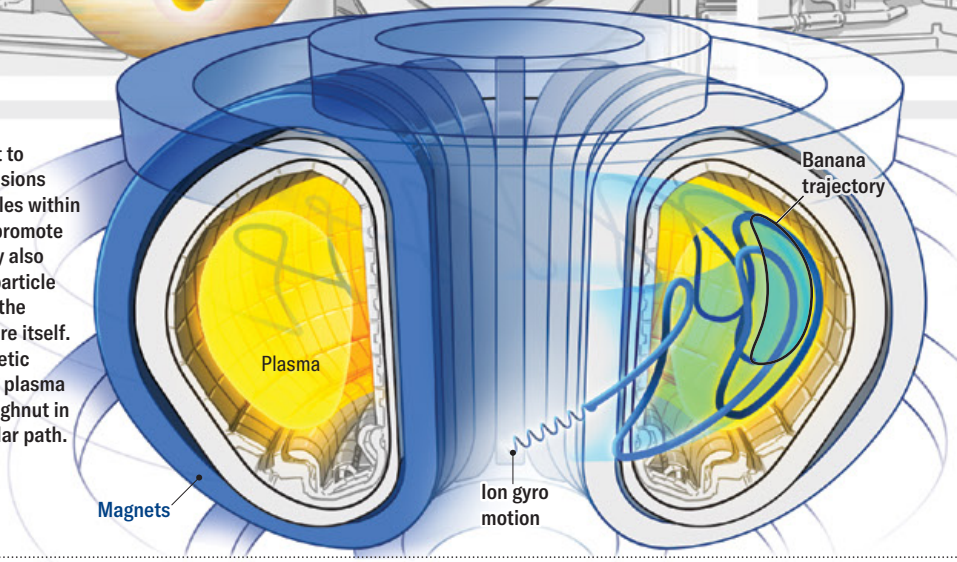
4 Heat the plasma to thermonuclear temperatures (150 million degrees Celsius) by injecting electromagnetic radiation and beams of high-energy neutral atoms.



5 As the temperature rises, the density and energy within the plasma increase, causing particles to collide and initiate fusion. Some of the energy released from each reaction is used to heat additional incoming fuel, perpetuating fusion. The goal is to then transfer most of the heat out of the reactor and use it to generate electricity via, for example, steam turbines.



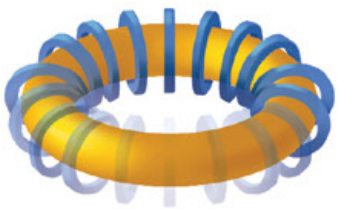
Scientists want to encourage collisions between particles within the plasma to promote fusion, but they also need to avoid particle collisions with the reactor hardware itself. Powerful magnetic fields steer the plasma around the doughnut in a roughly circular path.



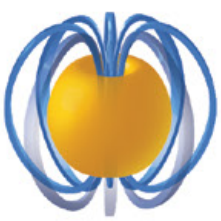
But a closer look reveals that the particle trajectories aren't that simple. Different plasma shapes each have benefits and drawbacks in maximizing temperature and density. Within the suspended plasma inside of a tokamak, particles move in two general patterns: helical motion (called ion gyro motion) and a banana-shaped path.

Different reactor shapes and sizes result in different plasma trajectories and have different pros and cons.

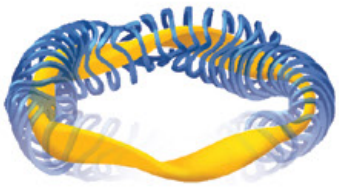
All tokamaks confine the plasma using a central electric current that can make fusion reactions difficult to maintain. **Traditional doughnut-shaped tokamaks** have more space in the middle. This space makes room to shield a central electromagnet from the heat of the plasma.



Spherical tokamaks—such as PPPL's National Spherical Torus Experiment-Upgrade—have narrower central areas than traditional tokamaks. They are more compact, can more efficiently confine plasma particles, and can be more economical to build. But the smaller central area requires a skinnier central electromagnet that can make the generation of the plasma current more difficult.



Stellarators, which take a twisted shape, don't require a central current to keep plasma trajectories in check. Magnets along the winding tunnel wall do the trick. But getting up to temperature can be tricky.



Because our energy demands are high and getting higher, it's likely that there is room for multiple models to succeed. "I'm confident that we need fusion," PPPL's Berzak Hopkins says, "so that makes me very confident that we will solve fusion."

50, 100 & 150 Years

1975 **CHARMING QUARKS**

"When the quark hypothesis was first proposed more than 10 years ago, there were supposed to be three kinds of quark. The revised version of the theory requires 12 kinds. In the whimsical terminology that has evolved, quarks are said to come in four flavors, and each flavor is said to come in three colors. ('Flavor' and 'color' are, of course, arbitrary labels.) One of the quark flavors is distinguished by the property called charm (another arbitrary term). The concept of charm was suggested in 1964, but until last year it had remained an untested conjecture. Several recent experimental findings, including the discovery last fall of the particles called J or psi, can be interpreted as supporting the charm hypothesis."

CLEANER GRAFFITI

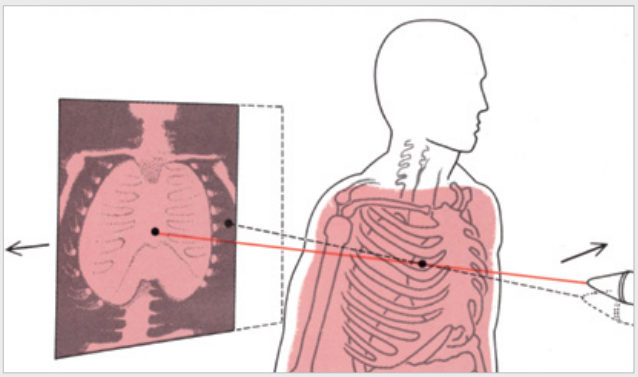
"Graffiti can be regarded as an intolerable nuisance or untrammeled self-expression. From either point of view the need is for clean surfaces; otherwise the public official loses all hope of restoring walls and conveyances to their original condition and the graffito artist runs out of canvas. The National Bureau of Standards puts forward a reasonable solution: Coat the surface in the first place with a special substance from which markings can be removed easily. The investigation turned up three preventive coatings that resist permanent bonding of most of the common types of marking. The three products are generically classified as a urethane, a dimethyl silicone and a styrene acrylonitrile terpolymer. McClure Godette, a chemist who worked on the project, said of them: 'These coatings cost just slightly more than a coat of paint, and they can be useful in making any future graffiti defacement easier to clean up.'"

1925 **FINGERPRINT EVERYONE**

"Two years ago two bank messengers with a bag containing $43,000 in currency were shot dead. Five bandits dashed into a car and disappeared. Twenty-four hours later the police found a car. On the windshield were the faint prints of a man's fingers, which coincided with those of a criminal whose record was in police headquarters. Four days later this man and two confederates were arrested in Cleveland. Three of the five men have since been electrocuted. Fingerprint records are of great value in other kinds of problems the police are called upon to solve, including missing persons and persons suffering from aphasia or amnesia. If all infants at birth were fingerprinted, the problem of foundlings would be solved. Fingerprints would be a distinct advantage to innocent citizens. Suppose a person was unjustly accused of a crime. They could prove their innocence by means of their fingerprints. This very thing has happened."

1875 **PATRIOTIC GALLIUM**

"At a recent session of the French Academy of Sciences, M. Wurtz presented a communication from M. Lecoq, announcing the discovery of a new metal analogous and allied to zinc and cadmium, and found in blende or sulphide of zinc in Spain. The existence of the substance was revealed by spectral analysis, two lines appearing which could not be traced to any other element. The new metal has not been reduced from its combinations, so its physical characteristics remain undetermined. It has been obtained, however, in the state of hydrochlorate and sulphate. The discoverer patriotically names the new element gallium." *Historical accounts say Paul-Émile Lecoq de Boisbaudran derived the term "gallium" from the Latin* Gallia, *which means "Gaul"—a region once ruled by Julius Caesar that encompassed present-day France.*



1975, Advent of Tomography: "Medicine is making the internal structures of the body far more accessible by noninvasive procedures. One, called reconstruction from projections, is coming into service. A tomogram is made by having an X-ray source move around a person in one direction, and film in the other direction, and mathematically combining X-ray images made from numerous angles into an image in three dimensions of organs within the body."

THE FIRST BAT SIGNAL?

"The roof of the Siemens-Halske factory at Berlin was recently the scene of [nighttime] experiments with the electric light, with a crowd in the streets staring with astonishment at a supposed wonderful natural phenomenon up in the clouds. The apparatus was arranged with an enclosed mirror, so that the rays were projected against the clouds, which served as a screen. In front of the mirror the signals were made, and these were repeated, of course on a gigantic scale, in the clouds. The light is to be adopted by the German army for night signaling." *Perhaps this is where the leaders of Gotham City, in DC Comics, learned to project an emblem of a bat on cloudy night skies to summon superhero Batman to a scene of distress.*

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"PEOPLE WATCHING," by Clarissa Brincat [Advances], should have quoted Laura Lewis as saying that humans' and chimpanzees' shared primate ancestor lived somewhere between eight million and five million years ago.